Correspondence

Role of ultrasound in bile duct obstruction

SIR,—The statements made by Dr Summerfield in his leading article (Gut 1988; 29: 741–5) are not supported by published work. First, ultrasound (US) has a sensitivity approaching 100% in the detection of bile duct dilatation. Second, US accurately defines the level of obstruction in almost all patients and in many will also show the cause of obstruction. The sensitivity of US in the detection of choledocholithiasis has improved significantly in recent years. An experienced radiologist will now detect between 56% and 79% of cases with bile duct stones, and false positive diagnoses are rare. It is true that stones within non-dilated ducts remain a diagnostic problem. Most pancreatic masses are well visualised with US and fine needle aspiration biopsy using US guidance permits a definitive diagnosis to be made. Gall bladder carcinoma and porta hepatis nodes are also well seen with US. A stricturing lesion in the absence of a pancreatic mass suggests a cholangiocarcinoma or much less commonly a benign stricture. The specific diagnosis of ampullary carcinoma is difficult. An intraluminal mass may be seen in the distal end of the bile duct or the bile duct may be dilated throughout its length without any obvious cause.

Ultrasound is cheap, non-invasive, and in experienced hands an accurate method of assessing bile duct obstruction. To advocate computed tomography (CT) as the initial diagnostic modality is to advocate the use of an expensive, invasive (it requires administration of intravenous contrast media), time consuming technique which is not readily available in every hospital. Most busy CT units would be unable to cope with the additional workload if asked to image all patients with suspected bile duct obstruction. The fact that clinicians find US difficult to interpret is an irrelevance. What is important is close cooperation between clinicians and radiologist and confidence by the clinician in the individual carrying out the scan. Clinicians can certainly orientate themselves better with CT images but the diagnosis of the exact cause of bile duct obstruction by CT is not readily made by anyone other than an experienced CT radiologist.

Bile duct obstruction should initially be investigated by US followed by endoscopic retrograde cholangio pancreatography (ERCP). Computed tomography should be reserved for equivocal cases and for those in whom surgery is contemplated.

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Reflux oesophagitis and oesophageal transit

SIR,—We read with interest the paper by Eriksen and coworkers (Gut 1988; 29: 448–52). The authors correctly convey the widespread belief that motility changes in the oesophageal body in gastro-oesophageal reflux are assumed to be secondary to reflux oesophagitis. It is misleading, however, not to acknowledge that many workers do not support this belief, and have postulated previously that impaired oesophageal clearance may well be a primary phenomenon.3,4 Furthermore, we are not convinced that the lack of correlation between transit time (which represents a complex of many factors) and endoscopic oesophagitis confirms the authors’ conclusions. Documentation of the presence of hiatus hernia, which in itself can influence oesophageal transit,1,4 and the effect of correction of gastro-oesophageal reflux on these changes would have been helpful.

We have previously conducted a manometric study on 48 patients with reflux oesophagitis which identified two separate manometric defects, one of which was proportional to, and one independent of, the degree of oesophageal mucosal damage. Furthermore, in 48 patients we studied by manometry and 24 hour pH monitoring before and after successful correction of gastrooesophageal reflux, 29 had significant manometric abnormalities, which were improved after surgery in 20 and unchanged in nine. These results suggest that some patients with gastro-oesophageal reflux have a pre-existing motor disorder of the body of the oesophagus, which may be
an important predisposing factor, but that progressive oesophageal mucosal damage may exacerbate the problem, thus establishing a vicious cycle. Fortunately, this latter component appears to be reversible by effective reflux control. In these circumstances, the association of reflux oesophagitis, not only with abnormalities of oesophageal body motility, but also with impaired gastric emptying and duodenogastric reflux, suggests that gastrooesophageal reflux may well be part of a diffuse upper gastrointestinal motility disorder.

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References

Spontaneous haemoperitoneum from rupture of a primary hepatic adenoma in an adult man — an update Sir,—You recently published a case report from the Clatterbridge Surgical Unit involving rupture of a primary hepatic adenoma in a male adult (Gut 1987; 28: 1170–2). In view of the interest shown in the case I feel it is appropriate to provide an update on the patient’s subsequent clinical course.

After initial surgery he remained well for almost two years, but then gradually deteriorated, became jaundiced and cachexic and died from a pulmonary embolus almost two years to the day after his original laparotomy. Necropsy confirmed a pulmonary infarct incriminating an embolic cause of death, but showed the source to be tumour embolus in the hepatic vein. The liver had areas of hepatic adenoma as before, but in addition areas of hepatocellular carcinoma. Stains for copper, hepatitis B antigen and alpha, antitrypsin deficiency were negative. There were pulmonary metastases from the hepatocellular carcinoma.

Re-examination of the original histopathology specimens from 1985 reconfirmed a benign hepatic adenoma. It is impossible to say whether, or not carcinoma existed at the time of the original presentation. Hepatocellular carcinoma is usually characterised by a short clinical course of three to four months, however, and often arises from pre-existing liver disease, such as cirrhosis. The absence of cirrhosis, Wilson’s disease, hepatitis and alpha antitrypsin deficiency, the presence of the benign adenoma, the long symptom free interval and consequent protracted clinical course, lead me to suspect that the patient eventually developed carcinomatous change in a pre-existing primary hepatic adenoma.

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Books


It was bound to happen; gastroenterologists can no more be shielded from the advance of the computer than anyone else. Like molecular biology, computer technology was not something that many of us met in our undergraduate education, but unlike molecular biology, many of us have had the opportunity to learn it from our children (‘Dad, have you been messing about with my computer again?’). The great thing about computers is that they are simple NON-SYSTEM DISK OR DISK ERROR to use and virtually ENTER NEW DATE (MM-DD-YY) foolproof. Above all, they save INVALID DATE a lot of time. Aside from their amusement value, why INVALID DRIVE SPECIFICATION do gastroenterologists need them? According to this symposium of enthusiasts, they can BAD COMMAND OR FILE SPECIFICATION simplify the construction of clinical data bases, the keeping of endoscopy records, and 54 FILE(S) 93184 BYTES FREE the