Hepatocellular carcinoma in idiopathic haemochromatosis

Sir,—The recent reports in Gastroenterology1 and Gut2 of hepatoma developing in the non-cirrhotic liver of patients with treated haemochromatosis prompts us to record the following case:

A 42 year old man presented in 1973 with marked irregular hepatomegaly. A liver biopsy showed cirrhosis and haemochromatosis. Venesection over a four year period resulted in clinical improvement and the liver became normal in size. Tiredness and sexual ability were helped by occasional injections of Nandrolone decanoate.

In 1981 he developed erythromelalgia and a thrombocythaemia of over $10^9/l$. Both responded to intermittent courses of hydroxyurea. Alpha foeto-protein level, ultrasound and isotope liver scans were normal.

In 1987 at the age of 57 he presented with a two month history of fever, rigors and weight loss of 22 kg. Alpha foeto protein was over 1100 $\mu$g/l (normal <5). Computed tomography scan showed multiple filling defects within the liver. Two liver biopsies showed normal liver tissue without fibrosis or excess iron. Subsequent guided needle aspiration showed hepatoma cells. He did not respond to chemotherapy and died two weeks later. Autopsy was not carried out.

The apparent complete reversal of fibrosis and iron storage in the liver of this patient suggests that some other mechanism may be responsible for the tumour development. It would be interesting to know if anabolic steroids were given to the patients in the other reports.

Perhaps this sequence of events may be more common than hitherto realised because of a reluctance to report single cases.

F SHEEHAN, C E CONNOLLY, AND C F MCCARTHY
Department of Gastroenterology, Histopathology and Medicine, Regional Hospital, University College Galway, Ireland

References

Primary mucinous adenocarcinoma developing in an ileostomy stoma

Sir,—The case report by Smart and colleagues (Gut 1988; 29: 1607–12) highlights a rare but significant complication of a longstanding ileostomy. They have shown that colonic metaplasia has occurred on the ileostomy of their patient, who initially had the colectomy for ulcerative colitis. I have recently reported a case,1 in which colonic metaplasia occurred on the ileostomy stoma of a patient who had had a total colectomy for polyposis coli. The external aspect of the ileostomy was the site of several tubulovillous adenomas, which appeared to arise in areas of colonic metaplasia. This patient had no evidence of polyposis elsewhere in the gastrointestinal tract. Another case report, not mentioned by Smart and colleagues, records colonic type polyps on an ileostomy, with malignant transformation in one of them, in a patient who had a colectomy for familial polyposis.2

Colonic metaplasia and adenomatous transformation may also occur within ileostomy reservoirs, either with continent ileostomy or with sphincter saving procedures.3 The potential for colonic metaplasia, with subsequent neoplasia, is a cause for concern at a time when increasing numbers of patients are being offered ileal pouches. Those who create such pouches must be aware that colonic type tumours may arise in the ileal mucosa. This risk seems to be greatest in patients with familial adenomatous polyposis, but the report by Smart and colleagues shows that there is also a similar risk in patients with ulcerative colitis.

C D JOHNSON
Department of Surgery, University Surgical Unit, F Level, Centre Block, Southampton General Hospital, Southampton SO1 6HO

References