Correspondence

Sulindac hepatotoxicity

SIR,—We were interested in the well documented report by Dr Whittaker *et al* concerning two patients with hepatotoxicity due to sulindac (*Gut* 1982; 23: 875). We report here a patient whom we believe suffered from this, but in whom a liver biopsy showed histological features differing from those reported by those authors.

A 45 year old Asian woman presented with a six day history of jaundice, abdominal pain, and fever. Her drug history was of six years therapy with dihydrocodeine for back pain, and three months' treatment with sulindac (Clinoral). Four days after the onset of jaundice her family practitioner treated her with co-trimoxazole. On admission she had tender hepatomegaly and her spleen was just palpable. Serum bilirubin was 200 $\mu\text{mol/l}$, aspartate amino transferase (AST) 246 IU/l ($N < 40$), gamma-glutamyl transpeptidase 1140 IU/l ($N < 35$), and serum alkaline phosphatase 595 IU/l ($N < 133$). Viral hepatitis markers were negative. After exclusion of extrahepatic obstruction by ultrasound examination, in view of the persisting symptoms, liver biopsy was performed 10 days after admission, which was also 10 days after withdrawal of drug therapy. At this time bilirubin was 78 $\mu\text{mol/l}$, AST 440 IU/l, and alkaline phosphatase 1040.

The histological features (Figure) were as follows: the vascular pattern was retained but there was mild

hyperplasia of liver plates. There was focal hepatocyte necrosis, with focal inflammatory infiltrates throughout the parenchyma, and eosinophils present among the infiltrating cells. The Kupffer cells were not notably reactive. Portal tracts were unremarkable but with a predominantly mononuclear cell infiltrate. There was no evidence of bile duct proliferation or obstruction, and ceroid pigment was not apparent.

Twelve weeks after presentation, however, the function test returned to normal and the patient was well and has remained so subsequently.

The clinical features of our patient are similar to those of Whittaker *et al*, and of other cases of sulindac hepatitis, but are obviously not specific. The histological features, with eosinophils present in the inflammatory infiltrate, and little reaction among the Kupffer cells, are suggestive of a drug reaction. The ceroid pigment noted in both Whittaker's cases was not found. There may therefore be a variety of histological abnormalities associated with a reaction to sulindac.

E A FAGAN, N WALFORD,
AND H J F HODGSON

Royal Postgraduate Medical School,
Hammersmith Hospital,
London.

Histological appearance of liver biopsy, taken 16 days after onset of jaundice attributed to sulindac. (Haematoxylin and eosin. $\times 150$)

Books

Contemporary patient management series. Peptic ulcer By Herman J Kaplan. (Pp. 96; illustrated; \$19.50.) New York: Medical Examination Publishing Co. 1983.

This short review has conventional chapters on duodenal ulcer and its complications, gastric ulcer, special aspects, surgical treatment, Zollinger Ellison syndrome, and acute stress ulceration. In this book duodenal ulcer is said to have pain occurring at 11 am, 4 pm, 10 pm, and 1 am, which is usually relieved within a matter of minutes by food or antacids; assessment of the gastric secretory capacity is said to be a useful procedure even though the appendix, *Protocol for gastric analysis*, alleges there is no pathognomonic range of acid production for any disease state with the possible exception of the Zollinger Ellison syndrome. Radiography is said to be the most practical and useful tool in diagnosing duodenal ulcer.