

tumours OR fast growing, metastasising tumours of different phenotype – that is, different morphological appearance. That is the problem, which, we think needs to be addressed with some urgency.

J PENSTON AND K G WORMSLEY

*Ninewells Hospital,  
Ninewells,  
Dundee, DD1 9SY*

#### **<sup>14</sup>C triolein breath test**

SIR,—It was interesting to read the manuscript by Mylvaganam *et al.* (*Gut* 1986; **27**: 1347–52). Our laboratory has been performing <sup>14</sup>C breath tests of various types for over a decade both as a research tool and as an aid to patient management. I noted that they used the eight hour cumulative data as their index. This makes the test rather long and contributes to an increase in reagent use and counting time. In 1981 we reported (*Clin Chem Acta* 1981; **112**: 371–4) a simplified method based on assessing the two to four hour rate. This correlates well with peak <sup>14</sup>CO<sub>2</sub> excretion and eight hour cumulative data. More recently (unpublished observations) we have shown that a single estimation at four hours post label ingestion is as discriminant as any of the other collection times. This greatly reduces the cost and complexity of the test.

It is surprising that this breath test and several others have not received more widespread acceptance in gastroenterology. The future I am sure will see a burgeoning of these simple (usually screening) tests in other fields as well as the gut. Perhaps the major roadblock has been the radioactivity (and the long  $t_{1/2}$  of <sup>14</sup>C) involved with these tests. The risk is minimal at the dosage levels used and even multiple tests result in little exposure, calculated in some cases to be less than the radiation dose from one abdominal radiograph. <sup>14</sup>C labelled substrates have been much vaunted as a solution to this perceived problem. As a research tool this is fine but for a practical screening test the label is very expensive and the detector (mass spectrometer) less readily accessible than a liquid scintillation counter.

ROSS BUTLER

*Gastroenterology Unit,  
Queen Elizabeth Hospital,  
28 Woodville Road,  
Woodville South,  
South Australia 5011.*

#### **Assessment of biliary tract pathology in familial adenomatous coli**

SIR,—Sarre *et al.* (*Gut* 1987; **28**: 303–14) have again confirmed the common occurrence of upper gastro-

intestinal polyps in familial adenomatous polyposis although there may be a geographical variation.<sup>1</sup> There is also an increased incidence of periampullary carcinoma in these patients<sup>2</sup> and the occurrence of biliary and pancreatic lesions has also been documented.<sup>3,4</sup>

The necessity to assess the biliary system in these patients has not yet been addressed but if the combined incidence of periampullary, pancreatic and bile ducts lesions is approximately 5%<sup>5</sup> endoscopic retrograde cholangiopancreatography is not only reasonable but necessary in those patients in whom adenomas of the stomach or duodenum have been demonstrated. At present, the incidence of biliary and pancreatic pathology in these patients remains anecdotal but their presence may be of some importance.

R J AITKEN

*University Department of Clinical Surgery,  
Royal Infirmary,  
Edinburgh.*

#### **References**

- 1 Aitken RJ, Torrington M, Elliot MS. Familial Polyposis coli in Cape Town with special reference to upper gastrointestinal polyposis. *S Afr Med J* 1985; **68**: 525.
- 2 Jones TR, Nance FC. Periampullary malignancy in Gardner's syndrome. *Ann Surg* 1977; **185**: 565–73.
- 3 Ushio K, Sasagawa M, Doi H, *et al.* Lesions associated with Familial Polyposis Coli; studies of lesions of the stomach, duodenum, bones and teeth. *Gastrointest Radiol* 1976; **1**: 67–80.
- 4 Lees CD, Herman RE. Periampullary malignancy in Gardner's syndrome. *Am J Surg* 1981; **141**: 378–80.
- 5 Jarvinen H, Nyberg M, Peltokallio P. Upper gastrointestinal tract polyps in familial adenomatous coli. *Gut* 1983; **14**: 333–9.

#### **Reply**

SIR,—In response to Doctor Aitken's letter regarding our article, Sarre *et al.* (*Gut* 1987; **28**: 303–14), I would like to make the following comments: In our communication we presented the prevalence of upper gastrointestinal polyps in a consecutive series of 100 patients with familial adenomatous polyposis undergoing upper endoscopy as a routine screening test. It was obvious in reviewing these results that the patients with adenomas of the duodenum had varying numbers of adenomas present, and some of them had adenomatous change in and around the ampulla. The dilemma that has been created by these observations relates to whether therapy of these lesions should be undertaken and if so by what means. We still do not truly know the incidence of duodenal, bile duct, or periampullary cancer in general in these individuals. We are not aware of any particular subsets that might be more likely to undergo malignant degeneration