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SIR,—Dr Rauws *et al* (*Gut* 1989; **30**: 798-803) describe an interesting and accurate test for *C pylori* but we dispute that it is 'inexpensive and simple' and their dismissal of endoscopy and biopsy, although invasive, represent a simpler option. In contrast with the ¹⁴C-urea breath test, it takes two minutes to take blood sample to measure serum *C pylori* antibody titres cheaply and easily by ELISA, a technique which is already widely available. Using the MICROELISA test it is possible to measure 200-300 samples within five hours.

The sensitivity and specificity of the ¹⁴C-urea breath test are quoted at 95 and 98% respectively by Rauws *et al*. Although there are occasional subjects with high serum titres to *C pylori* in the absence of active infection the sensitivity and specificity of the MICROELISA technique are both 94% (1-3). We do not think that these small differences between the two test are important.

In the time taken to carry out the ¹⁴C-urea breath on one subject, the serum samples from 10-20 subjects can be processed to measure *C pylori* titres. The material for MICROELISA cost 3.5 pence per sample and ¹⁴C-urea alone is quoted at £1.07 sterling per subject dose.

Thus while we agree that the ¹⁴C-urea breath test is sensitive and non-invasive (although requiring the administration of radiation), we do not think that it is either inexpensive or simple. Serum *C pylori* antibody titres measured by MICROELISA have several advantages and represent the optimum technique

for screening for *C pylori* especially in children, if endoscopy and biopsy are not to be carried out.

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Duodenal ulcer in sickle cell disease

SIR,—We read with interest the paper by Lee *et al*¹ describing their experience with duodenal ulceration in sickle cell disease (SCD) in Jamaica. They studied 51 patients with dyspeptic symptoms from among over 1000 patients with sickle cell disease, a prevalence of dyspepsia of about 5%. Dyspeptic symptoms are said to be more common in sickle cell disease patients,^{2,3} occurring in 33% of 60 patients aged over 30 with SS disease in Jamaica.² In the report from Accra, Ghana, no figures were given for the proportion of sufferers in their sickle cell disease clinic.³ We questioned a cohort of 49 consecutive patients (27 men, 22 women) presenting to us with sickle cell disease in Kumasi. Twenty seven (55%) had SS phenotype (mean age 22 years), 21 SC phenotype (mean age 26 years) and 1 S-beta-thalassaemia. Only three (6% - two SC, one SS) had a history of epigastric pain related to eating. In two, however, the symptoms had been minor and not recurrent and in the third case the symptoms had been absent for four years (unpublished observations).

Sergeant *et al*¹ reported a prevalence of duodenal ulcer of 30.5% in patients over 25 years with SS phenotype, although the overall prevalence in their sickle cell disease clinic was 7.7%, a figure little different from their estimate of 6% among age and sex matched controls attending a general medical clinic. In Britain, Brozovic and Anionwu⁴ reported an incidence of peptic ulcer of 4% in 139 patients with sickle cell disease (or 7% of those with SS phenotype). As pointed out by Lee *et al*, it is impossible to

be certain of a higher prevalence of dyspepsia or peptic ulcer in sickle cell disease patients without a knowledge of the general population prevalence.

Some guide may be given by a knowledge of the incidence of peptic ulcer in patients presenting with epigastric pain. Of 190 consecutive endoscopies carried out in Kumasi, Ghana for epigastric pain (unpublished observations), 41% were found to have peptic ulcer disease (78 duodenal ulcer, two gastric ulcer), a figure not very different from that of 31% among 51 dyspeptic patients with sickle cell disease in the series reported by Lee *et al*¹ and 45% of 20 dyspeptic SS disease patients in an earlier report from Jamaica.² We suspect that ethnic and social mix, together with climate and environment in Kumasi are not dissimilar to urban Jamaica, so our figures may well be comparable with those which might be obtained there.

In a further study, we carried out haemoglobin electrophoresis in 207 consecutive patients undergoing upper gastrointestinal endoscopy for upper abdominal pain or gastrointestinal haemorrhage, of which 70 had peptic ulcer disease. There was no significant difference in the prevalence of sickle cell disease between peptic ulcer patients (the only two with sickle cell disease had SC phenotype) and those with normal or other endoscopic diagnoses, nor with a group of 70 unselected university students.³ We did find, however, that smoking and male sex were significantly more highly represented among those with peptic ulcer and that the mean age of peptic ulcer patients was 43 years. Although this does not disprove a higher incidence of peptic ulcer in sickle cell disease, it does argue against this as a major factor in ulcer pathogenesis.

There are other possible reasons for a high incidence of duodenal ulcer in developing countries, which may include dietary factors, stress related to urbanisation⁴ and an exceptionally high prevalence of *Campylobacter pylori* infection, as we have reported from Ghana⁵ and others from Rwanda.⁶

The available evidence is insufficient to determine whether peptic ulcer is more common in sickle cell disease and therefore we support the statement of Lee *et al*¹ that further case control studies are required.

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News

New format of *Gut* – January 1990

This journal together with most of the associated journals of the *British Medical Journal* Publishing House have been redesigned after a series of discussion meetings during 1989.

The journal will emerge in its new A4 format in January 1990. This development will incorporate a larger typescript and the introduction of a flexible column width will greatly enhance the presentation and legibility of Tables, Figures, and illustrations. – Ed.