Oles’s disease is not a telangiectasia but a tumour

Sr.,—I have read with great interest the instructive article by Zentler-Munro et al. about an association of vascular abnormalities and hepatic fibrosis in Oles’s disease. Above all, I was impressed by their photographs. In Figure 3, there are dilated vascular spaces practically devoid of erythrocytes and still other ones full of red blood cells. I covered the latter by white stain and looked with astonishment how few hepatocytes remained from original hepatic tissue. To all appearances, a tissue of hepatic lobules was almost destroyed by vessels. 

It is of particular interest, the number of these vessels was not decreased by hepatic fibrosis, but it was increased as the consequence of a pathological process, which would result in the untimely death of a patient. The cause of this process could be an abnormal increase of growth factor in the portal venous blood. This hypothesis is in agreement with the clinical data that Oles’s disease is not a telangiectasia but a tumour. The only other way to explain this phenomenon is that the hepatocytes in Oles’s disease are replaced by other cells which are not normal hepatocytes. This theory is supported by the observation that the hepatocytes remaining in the liver of a patient with Oles’s disease are of different size and shape from normal hepatocytes. If this hypothesis is correct, it could be possible to treat Oles’s disease by using anti-tumour agents. This is a promising field for further research.

Sr.,—I have read with great interest the instructive article by Zentler-Munro et al. about an association of vascular abnormalities and hepatic fibrosis in Oles’s disease. Above all, I was impressed by their photographs. In Figure 3, there are dilated vascular spaces practically devoid of erythrocytes and still other ones full of red blood cells. I covered the latter by white stain and looked with astonishment how few hepatocytes remained from original hepatic tissue. To all appearances, a tissue of hepatic lobules was almost destroyed by vessels. 

It is of particular interest, the number of these vessels was not decreased by hepatic fibrosis, but it was increased as the consequence of a pathological process, which would result in the untimely death of a patient. The cause of this process could be an abnormal increase of growth factor in the portal venous blood. This hypothesis is in agreement with the clinical data that Oles’s disease is not a telangiectasia but a tumour. The only other way to explain this phenomenon is that the hepatocytes in Oles’s disease are replaced by other cells which are not normal hepatocytes. This theory is supported by the observation that the hepatocytes remaining in the liver of a patient with Oles’s disease are of different size and shape from normal hepatocytes. If this hypothesis is correct, it could be possible to treat Oles’s disease by using anti-tumour agents. This is a promising field for further research.

Oesophageal carcinoma in Sri Lanka

Sr.,—With reference to the article by Sagar (Gut 1989; 30: 561–4), I would like to add that Sri Lanka was a transit point in the old silk route between China and Rome. Marco Polo also stopped over in Sri Lanka (then identified by him as Ceylon) on his return from China.1 On the diet of the natives of the northern region of Ceylon, seven centuries ago, Marco Polo wrote, ‘They have no grain other than rice. They have sesame, from which they make oil. They live on milk, flesh and rice and have wine made from trees’.

The wine he referred to, is known as ‘kailu’ in Tamil language, and is produced from the palmyrah palm Borsassae flabellifer.

Stephen and Uragoda2 have reported that cancer of the oesophagus is the commonest among the patients admitted to thoracic units in Ceylon, and Ceylon is one of the few countries with a high incidence of both oral and oesophageal carcinoma. In China,3 the male-female ratio of patients with oesophageal cancer is reported as 2:1. But in Sri Lanka, the incidence of oesophageal carcinoma is higher among women than men.4

S S KANTHA
Dept of Physiology and Biochemistry, Medical College of Pennsylvania, Philadelphia, PA 19129, USA


Oesophageal carcinoma in Sri Lanka

Sr.,—With reference to the article by Sagar (Gut 1989; 30: 561–4), I would like to add that Sri Lanka was a transit point in the old silk route between China and Rome. Marco Polo also stopped over in Sri Lanka (then identified by him as Ceylon) on his return from China.1 On the diet of the natives of the northern region of Ceylon, seven centuries ago, Marco Polo wrote, ‘They have no grain other than rice. They have sesame, from which they make oil. They live on milk, flesh and rice and have wine made from trees’.

The wine he referred to, is known as ‘kailu’ in Tamil language, and is produced from the palmyrah palm Borsassae flabellifer.

Stephen and Uragoda2 have reported that cancer of the oesophagus is the commonest among the patients admitted to thoracic units in Ceylon, and Ceylon is one of the few countries with a high incidence of both oral and oesophageal carcinoma. In China,3 the male-female ratio of patients with oesophageal cancer is reported as 2:1. But in Sri Lanka, the incidence of oesophageal carcinoma is higher among women than men.4

S S KANTHA
Dept of Physiology and Biochemistry, Medical College of Pennsylvania, Philadelphia, PA 19129, USA


Ranitidine noce (Rhs) vs ranitidine mane and noce (Rbid)

Sr.—We wish to comment on the abstract by Dobrilla et al. (Gut 1989; 30: A726) stating that there is no significant difference between Rhs and Rbid healing rate at two weeks but the authors did not mention which statistical test and significance level they used.

We found, however, using the χ2 test, without Yates correction, a significant difference between the two treatments at two weeks (χ2= 5.2 resulting in a p value<0.05).

It would be interesting to know why according to the authors the difference in healing rate was not significant at two weeks.

PLAS
PH VAN WILDER
Stationstraat 6, 8420, De Haan, Belgium

Reply

Sr.—With regard to the letter of VanWilder and Jan Plas, the comment of these authors is correct but there is an error in our abstract.

In fact, while the reported ranitidine bid healing percentage by week two is correct (64–2%), the absolute figures are 102/159 and not 107/159, as indicated. Using the χ2 test on the correct number of patients, the difference between the two treatment groups proves to be statistically not significant.

Further details can be found in the full paper which now has been published. (Clin Trial J) 1989; 26: 153–62.

G DOBIRLLA
Department of Gastroenterology, General Regional Hospital, Bolzano, Italy

H pylori and duodenal ulcer

Sr.—Dr Carrick and colleagues are to be congratulated on quantifying in their 137 subjects the strength of the risk factor for duodenal ulceration (relative risk = 51) of duodenal infec-