Correspondence

Portal and mesenteric thrombosis revealing constitutional protein C deficiency

SIR,—We read with interest the article of Valla and colleagues in the June issue (*Gut* 1988; **29**: 856–9). We had to deal with a similar case when in May 1984, a 49 year old woman was admitted to our hospital for a small bowel obstruction. At the subsequent laparotomy, 1 m ileum was found necrotic and the histological examination of the resected bowel showed an ileomesenteric infarction. Peroperative liver biopsy showed no abnormality. The presence of splenic, mesenteric and portal veins thrombosis was assessed by means of ultrasonographies, CT scan and coelio-mesenteric arteriography. Three small oesophageal varices were noticed at endoscopy. An extensive bilateral phlebitis of the legs occurred in the postoperative period and was treated by heparin, and a Greenfield caval filter device was implanted. Haemostasis and coagulation investigations, including antithrombin III, were found normal, but measurement of protein C was not carried out. In April 1985, the patient was readmitted after three episodes of gastrointestinal bleeding. Varices were large. Liver function tests, platelet count, prothrombin and cofactors, antithrombin III and protein S antigen were all found in the normal range. Myelogram showed well balanced myeloid populations. Protein C antigen (immunoenzymatic) was found decreased at 39% (n=70–120). To prevent recurrence of variceal haemorrhage, a trans-section of the oesophagus was done, followed by a new phlebitis of the left leg, treated with heparin and then calciparin. In December 1985, a recurrence of variceal bleeding as a result of a rupture of residual varices of the lower oesophagus required treatment by sclerotherapy for six months. Follow up was satisfactory and the patient has not suffered from phlebitis or gastrointestinal bleeding since. The patient never received antivitamin K (AVK). Protein C, controlled in October 1987, was still very low in activity at 40% and slightly lowered in antigen at 59%. An inquiry in 1987 in the patient's family with measurements of protein C antigen showed that, among the four children and the three brothers and sisters of the proband tested, only one son had a slightly decreased protein C antigen rate (63%), while his protein C activity was normal. None of the proband's relatives had a clinical history of thrombosis.

Constitutional protein C deficiency is still generally considered to be responsible for venous thrombosis of the lower limbs, pulmonary embolism, and sometimes thrombosis of the mesenteric vein. Yet, in the

recent series of Green *et al.*,¹ two cases of splanchnic venous thrombosis with protein C deficiency were probably constitutional deficiencies. Valla and colleagues' case report and our own case confirm that constitutional protein C deficiency is probably not exceptionally engaged in the pathogenesis of chronic portal thrombosis. Other works by Valla *et al.*,² however, suggest that proteins C and S deficiencies should not explain the majority of 'idiopathic' portal thrombosis. Nevertheless, we agree with the authors that measurements of proteins C and S, now easily feasible in most centres,³ should be carried out in patients with portal thrombosis when no overt cause is present. An interesting point is the prevention of recurrent variceal haemorrhage in those patients. The authors suggest the use of β -blockers against portal hypertension, arguing with relevance that it is compatible with AVK. Our patient has been doing well for three years after sclerotherapy but she did not receive AVK because she was protected against pulmonary embolism by her Greenfield filter. The problem seems to put in the balance the risk of bleeding from postsclerotherapy ulcers if the patient is treated with AVK and a therapy, propranolol, whose efficiency might not be as satisfactory as sclerotherapy.

F PRAT, D OUZAN, N TRECZIAK, AND C TREPO

*From the Hepatology Unit,
Hotel Dieu Hospital,
69288 Lyon, France, and
Laboratory of Hemostasis and Coagulation,
E Herriot Hospital,
69003, Lyon, France*

References

- 1 Green D, Ganger DR, Blei AT. Protein C deficiency in splanchnic venous thrombosis. *Am J Med* 1987; **82**: 1171–4.
- 2 Valla D, Casadevall N, Huisse MG, *et al.* Etiology of portal vein thrombosis in adults. A prospective evaluation of primary myeloproliferative disorders. *Gastroenterology* 1988; **94**: 1063–9.
- 3 Rapaport SI, Zivelin A, Donnelly K. A plasma protein C activity assay suitable for a clinical laboratory. Its use to measure activity in hereditary and acquired deficiency states. *Am J Clin Pathol* 1987; **4**: 491–7.

Nicotine and ulcerative colitis

SIR,—Cigarette abstinence is sometimes a feature of ulcerative colitis, and perhaps of some cases of Crohn's disease. Behavioural regulation of physiological homeostasis is well known, and influences many needs such as alleviation of hunger and thirst. A proposal that cigarette smoking is symptomatic of one or more physiological deficiencies in bio-