The level of prolongation of the prothrombin time at which a contemplated percutaneous biopsy should be abandoned in favour of a transjugular or plugged procedure has likewise not been defined. It would be helpful if future audits or reviews aimed to clarify this point. A prospective study of the efficacy and duration of action of fresh frozen plasma is underway at the Queen Elizabeth Hospital.

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Reply

EDITOR,—As Dr Jolobe will be aware from the design of the study, we are reporting on current practice over a wide range of hospitals in England and Wales, and, as he points out, suspected malignancy was the indication in about half of those patients over 65 years of age who had a percutaneous liver biopsy. As the data were drawn from the last 10 biopsies done in each hospital, they do not tell us how many patients with suspected hepatic malignancy were treated without histological confirmation, but it is likely that there were many. The frequency with which clinicians found the result helpful, however, emphasises that they found the confirmation useful in discussions with the patient and relatives and in establishing a management plan in a condition that has such a poor prognosis. Clinicians will be only too aware of the harm that can be done by the occasional mistaken diagnosis of a terminal condition. The relative roles of biochemistry, imaging, and liver biopsy in the diagnosis of hepatic malignancy in this study is the subject of a separate communication,[1] but in this study the specificity of ultrasonography was only 86% and sensitivity 82%. Furthermore, in a small number of cases a non-malignant diagnosis was positively established at liver biopsy when malignancy had been suspected on ultrasound examination.

Like Dr Fisher, we believe the data supporting the relation between coagulopathy and haemorrhagic complications are important in view of previous negative reports. Bleeding can occur, however, with normal clotting. The precise indications for alternative techniques such as plugged or transjugular biopsy techniques (used very rarely in this study) remain empirical, but they are an attractive alternative to the expensive and precious resource of human fresh frozen plasma.

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Increased CA 125 in tuberculous peritonitis

EDITOR,—O'Riordan *et al* reported the case of a patient with tuberculous peritonitis, ascites, and pleural effusion who also had a remarkably increased concentration of serum CA 125 (*Gut* 1995; **36:** 303–5). Because of the increase in this tumour marker an ovarian cancer was suspected and a laparotomy was performed with negative results for cancer. The authors consider the increased values of CA 125 the most interesting aspect of the case, and review other published reports on increased CA 125 values in patients with tuberculous ascites.

I believe, however, that the only interesting and noticeable aspect of the case would have been if the patient had had a normal value of CA 125. This tumour marker, commonly used in the diagnosis and follow up of ovarian cancer, increases in a variety of processes involving pleura, pericardium, and peritoneum including endometriosis, peritonitis, pelvic inflammatory disease, and surgical trauma.[1–3] The increase in CA 125 is not unexpected because this antigen has been detected on mesothelial cells in pleura, pericardium, and peritoneum, particularly in areas of inflammation.[4]

Benign serous effusions, particularly ascites, are associated with increased serum concentrations of CA 125 with values up to 100 times the upper normal limit in some cases.[1 5 6] CA 125 has proved to be an excellent marker for ascites in patients with benign liver diseases (sensitivity 98·4%, specificity 95·9%, efficiency 96·9%).[6] This marker is also very sensitive to minimal amounts of ascites and correlates very well with the amount of ascitic fluid.[6] Thus, the specifity of CA 125 for ovarian cancer is very low in the presence of ascites of whatever origin.

In addition, the return of CA 125 to normal after anti-tuberculous treatment in the patient of O'Riordan *et al* is not unexpected. CA 125 decreases to normal values when the ascites is removed in cirrhotic patients and increases again when ascites recur.[6 7]

The authors conclude that 'tuberculosis needs to be considered in the differential diagnosis of ascites with increased tumour markers'. It is well known, however, that CA 125 is an unspecific marker of ascites of whatever aetiology, and tuberculosis, a disease that only uncommonly produces peritoneal effusions, represents only one of the many aetiologies of ascites. Finally, I believe that a laparotomy should not have been performed in this case because both computed tomography and cytological study of ascitic fluid were negative for ovarian cancer and the very high concentration of CA 125 could have been easily explained by the existence of both ascites and pleural fluid.

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Reply

EDITOR,—Dr Collazos' points concerning CA 125 are valid. He is, however, an expert in the area and has published widely on CA 125. We felt this case to be of interest to a general audience because they might not have been familiar with the wide range of differentials for increased serum CA 125. The concentrations of CA 125 are high in many conditions as outlined in Dr Collazos's letter. We mentioned all of these and more in our discussion. Dr Collazos does not mention how he would have made the diagnosis. He might have legitimately requested laparoscopy in place of laparotomy. He suggests neither. We believe he overstates the value of negative computed tomography and non-diagnostic ascitic fluid cytology in excluding intra-abdominal tumour, however. Such measures can only reduce the relative risk that a tumour is present.

One additional point of some interest is that despite our considerable experience with immunocytochemistry and CA 125 in cytological preparations, we were unable to identify positive staining in the mesothelial cell population in the ascitic fluid specimen in retrospect. Had the immuno-panel been performed during the case this too would have urged us towards more invasive investigations.

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Adenomas and a family history of colorectal cancer

EDITOR,—The data presented by Gaglia *et al* (*Gut* 1995; **36:** 385–90) imply that adenomas in hereditary non-polyposis colorectal cancer (HNPCC) occur with increased frequency, show a predilection for the proximal bowel but are not especially large, dysplastic or likely to show villous change. It is interesting to note that these data are at complete variance with our own.[1] We have suggested that the high risk of colorectal cancer in HNPCC is explained not by increased initiation of