SIR,—We are grateful for the opportunity of replying to the points made by Dr Williams and his colleagues. As we pointed out in our paper, the interpretation of any data used to predict resectability of bronchial carcinoma depends critically on the criteria used by individual surgeons to decide whether or not potentially curative surgery is technically possible. For this reason it is difficult to compare our results with theirs. The best index of successful resection would be information on survival, which they do not give. We pointed out that in our series only one of 10 patients in whom the preoperative perfusion of the affected lung was less than 25% of the total survived two years and we suggest that this supports the use of scanning in the assessment of operability. As with most investigations, the additional information given by scanning has to be interpreted in the light of other clinical, radiological, pathological, and functional data and whether or not a thoracotomy is performed will not be determined by the scan in isolation from other information. Our suggestions on the quantitative use of scanning in assessment of operability were in fact more conservative than the recommendations in some earlier studies.1

We would agree entirely with Dr Williams and his colleagues on the value of scans in prediction of postoperative lung function2 and exercise performance.3

JAUNDICE AFTER OPEN HEART SURGERY: A PROSPECTIVE STUDY

Sir—The article by Dr C-M Chu and others (January 1984;39:52–6) has prompted us to draw attention to the histological changes in the liver, and the medical counterpart, of the condition they describe following cardiac surgery. Undoubtedly their patients had "ischaemic hepatitis," a recognised cause of jaundice,1 but one which we feel is underdiagnosed in the United Kingdom. We have seen four medical patients who have developed this entity. Three were associated with cardiogenic shock due to myocardial infarction and one with shock lung due to renal failure following repair of a ruptured abdominal aortic aneurysm. Invasive manometry in these cases revealed the following pathophysiological mechanisms to be involved: (1) a high right atrial pressure, which causes hepatic congestion due to impaired venous drainage; (2) prolonged hypotension leading to underperfusion of the liver; and (3) hypoxaemia despite supplementary oxygen. These findings are similar to those of Dr Chu and his colleagues.

Our patients survived from three to nine days after hospital admission and developed preterminal bilirubin levels ranging from 135 to 475 μmol/l (7-9 to 27-8 mg/100 ml) and AST levels ranging from 350 to 2360 IU/l. All the patients succumbed to their myocardial or pulmonary disease. At necropsy the livers showed marked venous congestion. The capsules were smooth and stretched. Microscopically the specimens revealed changes caused by ischaemia (fig). Owing to their location hepatocytes at the periphery of the liver acinus receive blood at a lower oxygen tension than those cells close to the hepatic artery. They are thus more susceptible to damage when hypoxia and hypotension are present, and when in addition there is failure of venous drainage extensive cell necrosis at the periphery of the acinus may occur. As bile flow is dependent on arterial oxygen tension hypoxia may lead to slowing of bile secretion and even cholestasis.2 All of these changes were seen in our patients.

The elevated serum bilirubin and AST levels may lead the clinician to consider a diagnosis of viral hepatitis. Awareness of the possibility of ischaemic hepatitis should,
Severe congestion at the periphery of a hepatic acinus in case 1. The portal tract (PT) is intact. (Haematoxylin and eosin, × 33.)

however, lead to intensive efforts to reduce right atrial pressure, improve oxygenation, and maintain an adequate blood pressure. Reversal of these factors may protect the liver from further injury.3

If the diagnosis is still in doubt a liver biopsy should, if possible, be performed as the histological features of ischaemic hepatitis are characteristic and quite dissimilar to those of viral hepatitis.

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Notices

Can we have safer cigarettes?

A one day conference entitled “Can We Have Safer Cigarettes?” will be held under the auspices of the Chest, Heart and Stroke Association at the Bloomsbury Crest Hotel, London WC1, on Thursday 8 November 1984. The conference will be chaired by Professor Geoffrey Rose and should be of interest to those concerned with the formation of policy on the health effects of smoking, clinicians, community physicians, epidemiologists, health educators, and concerned lay groups. Subjects included: the market—its patterns and changes (Mr PN Lee); smoke constitution and respiratory disease (Dr TW Higgenbottam); smoke constitution and coronary heart disease (Professor NJ Wald); nicotine and smoking behaviour (Dr MAH Russell); advice to patients (Dr SG Spiro); national policy—a personal view (Mr R Peto). There will be periods for panel discussion and general discussion. Full details are available from the Chest, Heart and Stroke Association, Tavistock House North, Tavistock Square, London WC1H 9HE.

British Thoracic Society: future meetings

6–7 December 1984
Kensington Town Hall, London
NB Abstracts required by mid September

3–5 July 1985
University of York

5–6 December 1985
Metropole Hotel, Birmingham

18–20 June 1986
Cheltenham