Thorax 1984;**39**:638–640

Correspondence

Atopy, immunological changes, and respiratory function in bronchiectasis

SIR,—Dr MB Murphy and others (March 1984;**39**:179) found no serum IgG subclass deficiencies in a study of 23 patients with bronchiectasis and commented that they knew of no other studies of IgG subclasses in this condition.

At the 1983 summer meeting of the British Thoracic Society we presented the results of IgG subclass estimations in 40 patients with bronchographically proved bronchiectasis and 40 patients with recurrent acute respiratory infections.[1] All patients had normal or raised total IgG. We found seven patients with serum IgG_2 concentrations of 0·04–0·57 g/l, below our normal range (1·1–10·7 g/l), of whom two had bronchiectasis. Five of the seven had concomitant IgA deficiency (one with bronchiectasis) and two had undetectable IgG_4 (one with bronchiectasis). There was no clinical difference between the patients with IgG subclass deficiencies and those with normal or raised levels.

Hence IgG subclass deficiencies can be found in patients with bronchiectasis. Patients with IgG_2 deficiency have increased susceptibility to recurrent pyogenic infections,[2] and have been reported to benefit from immunoglobulin replacement therapy,[2 3] and therefore we consider the detection of such deficiencies to be important.

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**This letter was sent to the authors, and Professor Fitz-Gerald replies below

SIR,—We were very interested in the findings of Drs Stanley and Cole, which appeared in abstract form after submission of our paper. The combined data from the two studies give a broad perspective of the importance of immunoglobulin and immunoglobulin subclass deficiency in bronchiectasis. Clearly, our study groups were somewhat different given the very high prevalence of IgA deficiency in the Brompton group, perhaps reflecting specialised tertiary referral to that unit. The data based on a combined total of 63 patients with bronchiectasis show that immunoglobulin subclass deficiency may occasionally contribute to the pathogenesis of bronchiectasis in that two patients had IgG_2 deficiency, one of whom had concomitant IgA deficiency. We are certainly in agreement with the therapeutic implications outlined by Drs Stanley and Cole and, indeed, made these very points in our paper.

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Role of lung scanning in assessing the resectability of bronchial carcinoma

SIR,—We are concerned with the practical, surgical implications for patients with bronchial carcinoma in the report by Dr DA Ellis and his colleagues (April 1983;**38**:261–6). Their conclusions state that for centrally placed tumours perfusion of 25% or less in the affected lung is likely to be associated with unresectability and that "The addition of lung scanning to the preoperative evaluation of selected patients with bronchial carcinoma should help to reduce the number of patients undergoing unrewarding thoracotomy." The authors base this major assumption on the results from only six patients, one of whom had a successful resection. To add any credibility to their statement it should be expected that this one patient would have the highest perfusion in the group, but in fact the patient had only 2% perfusion in the diseased lung.

Since 1980 in this regional thoracic surgical unit we have regularly undertaken quantitated ventilation and perfusion lung scans in patients with bronchial carcinoma considered to be of poor operative risk. During this period we have had seven patients with centrally placed tumours and reduced perfusion which were technically resectable on clinical, radiographic, and bronchoscopic assessment. The percentage perfusion for the lung containing the tumour, surgical outcome, tumour size, and reason for inoperability are shown in the accompanying table. Four out of seven patients with considerably reduced perfusion had a successful lung resection—these findings are in contradiction to those of Dr Ellis and his colleagues. There was no relationship between the surgical outcome and either the degree of impairment of lung perfusion or tumour size in our patients.

We believe that quantitated lung imaging in carcinoma of the bronchus is best reserved for estimating postoperative lung function[1] and not in determining tumour resectability, no matter what the level of perfusion. No patient with lung carcinoma should be denied potentially curative surgery on the basis of an unfounded statistical probability.

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**This letter was sent to the authors, who reply below.

Details of seven patients with technically resectable bronchial carcinomas

Patient No	*% perfusion*	*Tumour size (cm)*	*Surgical outcome*	*Reason for inoperability*
1	14	3	Resected	
2	26	9	Resected	
3	14	4·5	Resected	
4	4	5	Resected	
5	6	8	Not resected	Pulmonary artery and pericardial disease
6	24	Not assessed	Not resected	Invasion of aorta; subaortic lymph node disease
7	8	7	Not resected	Cardiac invasion and pleural effusion

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 function[2] and exercise performance.[3]

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[2] Ellis DA, Hawkins T, Gibson GJ, Nariman S. Prediction of lung function after pneumonectomy for bronchial carcinoma. *Thorax* 1982;**37**:786–7.
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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 premortem 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,