Correspondence

Jaundice after open heart surgery: a prospective study

Sir,—We would like to comment on the Paper of Dr C-M Chu and others (January 1984;39:52–6).

In a similar study including 155 consecutive patients we obtained results which were different from those reported in this paper. In particular, most of the serum bilirubin was unconjugated, implying a haemolytic origin of the jaundice. Furthermore, hyperbilirubinaemia (serum bilirubin >3 mg/100 ml (51 μmol/l)) was found in 40% of patients on the first postoperative day and in 17% of patients on the second postoperative day. Usually the serum bilirubin reached its peak value on the first postoperative day, followed by a steady decrease; in only 13 patients (8%) did peak values appear later than on the first postoperative day.

The incidence of hyperbilirubinaemia was distinctly associated with valve replacement (60% on postoperative day 1) rather than with aortic coronary bypass surgery (30% on postoperative day 1: p < 0·01 with χ² test). This may be due to the higher number of aortic coronary bypass patients (53%) in our study than in that of Dr Chu and others (7%). Comparison of perioperative recorded data with the serum bilirubin levels on postoperative day 1 revealed a significant correlation with the amount of suction during operation, the duration of aortic cross-clamping, and the amount of blood administered (p < 0·05, Spearman correlation coefficient), as well as with the duration of artificial ventilation (p < 0·01).

We suggest therefore that hyperbilirubinaemia after extracorporeal circulation is a multifatorial process. As previously reported,¹ the main source of serum bilirubin is damaged red blood cells, as shown by the specific pattern of conjugated and unconjugated serum bilirubin, as well as the free plasma haemoglobin and haptoglobin. The higher incidence after valve replacement seems to be caused mainly by the greater amount of pericardial suction. In addition, the hepatic function of glucuronisation is impeded² according to the severity of the operation, as is shown by the correlation between serum bilirubin concentration and the duration of artificial ventilation or aortic cross clamping. In the individual case the importance of each of these factors needs to be considered.

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Asthma and irreversible airflow obstruction

Sir,—We read with interest the paper by Dr P Brown and colleagues (February 1984;39:131–6). Their results are consistent with our own data.

We assessed the influence exerted by the age of the patients and the duration of asthma on the recovery from acute exacerbations in 52 women (mean age 46·4, SD 15·1 years) with chronic asthma, all non-smokers and without chronic bronchitis.

A negative correlation was found between the age of the patients and the highest FEV₁ reached expressed as a percentage of the predicted normal value (FEV₁% P) (r = −0·34, p<0·05). There was significant correlation between age and duration of asthma. We therefore studied two subgroups of patients with similar ages (mean 50·8 (SD 10·6) and 48·5 (10·5) years respectively) and different duration of asthma (mean 30·7 (9·1) and 8·8 (5·2) years). The first subgroup was composed of 25 asthmatic women and the second of 20. The mean (SD) FEV₁% P of the first subgroup was 91·30 (17·5) and of the second 103·0 (17·6); this difference was significant (p<0·05).

Our findings suggest that asthma can cause irreversible airflow obstruction and that the obstruction is a function of the duration of asthma and of the age of the patients.

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

Sir,—We thank Dr Alvarez Sanchez and colleagues for drawing our attention to their work and note with interest the similarity of our results. Their patients were selected because they presented with acute asthma, in contrast to our group of patients with chronic stable asthma, in whom a systematic attempt was made to minimise any reversible element of airflow obstruction. The significant reduction in FEV₁% P with longer duration of asthma accords with our findings and it would be of interest to know the correlation of FEV₁% P and duration of asthma in their patients.

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Notice

The second International Postgraduate Course in Pulmonary Vascular Pathology will be held in Amsterdam on 22–24 May 1985. The course is designed to provide detailed information by an international faculty on the pathology of the pulmonary circulation in heart and lung diseases as well as of vascular malformations in the lung. Special emphasis will be placed on evaluation of lung biopsy specimens taken in such conditions and on problem solving aspects. Further information may be obtained from Professor CA Wagenvoort, Department of Pathology, University of Amsterdam, Academic Medical Centre, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands.

Correction

The pathology of asbestos related disease

In the editorial by Dr JMG Davis (November 1984;39:801–8) we regret that there is an error on p 806, line 22 of column 2, which should read "caused by asbestos only if asbestos is also present..."
Jaundice after open heart surgery: a prospective study.

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Thorax 1985 40: 80
doi: 10.1136/thx.40.1.80

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