with adult respiratory distress syndrome (ARDS). Comparison was made with nine patients with interstitial lung disease (ILO) and 18 control patients (C). The total cell count was reduced in the BALF of ARDS patients ($0.19 \times 10^9/l$ *v* $0.58 \times 10^9/l$ (ILD) *v* $0.42 \times 10^9/l$ (C)) and the cell type ratios altered significantly in favour of neutrophils (43% (ARDS) *v* 1% (ILD) *v* 1% (C)). Protein content was significantly greater in the ARDS BALF. The ratio BALF: serum was significantly greater in ARDS patients for albumin, transferrin, ceruloplasmin, and C3 component of complement. The relationship of log BALF/serum to molecular weight was linear ($y = -0.0077, x + 1.945$; $r = -0.9751$; $p<0.05$), indicating preservation of molecular selectivity during increased permeability at the capillary alveolar barrier.

Neutrophil kinetics and oxidative injury associated with cardiopulmonary bypass

D ROYSTON, JS FLEMING, S WESTABY, J DESAI, K TAYLOR In this study we have investigated the time course of egress of neutrophils into the lung in seven patients having surgery for coronary artery bypass grafting. In addition we measured peroxidation products in the plasma using the Thiobarbituric acid (TBA) reaction. This assay was used to give an index of tissue injury induced by oxidant species (Fleming JS *Artif Organs* 1984;**8**:91-6). Samples of central venous (CV) and left atrial (LA) blood were taken after administration of heparin (300 U/kg) and at 30 minutes in bypass and then two minutes prior to and at 5 minute intervals up to 25 minutes after removal of the aortic cross clamp (AoXC) while still on bypass. Samples were analysed for white cell, neutrophil and lymphocyte count and plasma TBA reactive products. The results showed that there were no significant differences between CV and LA cell counts whilst the AoXC was in place. However, there was a highly significant ($p<0.001$) egress of neutrophils into the lung following AoXC removal. This was associated with a highly significant ($p<0.001$) rise in CV neutrophil count. There was also a significantly ($p<0.01$) higher LA concentration of TBA reactive products compared to CV at 10, 15 and 20 minutes following AoXC removal. These data suggest that any oxidant lung injury associated with cardiopulmonary bypass may occur during the period of partial bypass.

Complement activation and the change in lung function with intravenous radiological contrast material

N ASHFORD, N CRACKNELL, J MCCARTY, P DOYLE, T HIGENBOTTAM Reactions to intravenous radiological contrast material include dyspnoea and wheeze. The mechanism for contrast reactions remains unknown, although ***in vitro*** studies suggest activation of complement with the generation of C3a (Dawson P. *Br J Radiol* 1983;**56**:447-8). We have studied 10 patients including 5 smokers undergoing urography using diatrizoate (Urografin 325). Spirometry was recorded before and five minutes after the injection. Lung epithelial permeability was also measured using a small radiolabelled molecule ^{99m}Tc DTPA as an index of damage to the alveolar capillary barrier (Jones JG. *Br J Anaesth* 1982;**54**:705). Blood samples were taken before and five and 20 minutes after injection. Haemoglobin, white cell count and platelet count were determined together with complement, C3, C4, CH50, and C3a by radioimmunossay (Hugli JE. In: *Future prospectives in clinical laboratory immunoassays* 1980). No patient suffered any contrast reaction. There was a small but significant fall in FEV_1 after contrast injection from, FEV_1 3.8 to 3.5 l ($t = 3.42$; $df = 9$; $p<0.01$). There was also a fall in DTPA T½, from 28.9 to 21.5 ($t = 2.55$, $df = 9$; $p<0.025$), indicating increased clearance. However, there were no significant changes in white blood cell or platelet counts nor in complement level. Intravenous injection of diatrizoate can cause increased clearance of DTPA and a reduction in FEV_1. Complement activation does not appear to be the cause, but we suspect direct toxic damage by Urografin to the alveolar capillary barrier.

Notice

British Thoracic Society: future meetings

3–5 July 1985 University of York, York
11–12 December 1985 Kensington Town Hall, London
18–20 June 1986 Cheltenham Town Hall, Cheltenham

Members should note the change of date and venue for the 1985 winter meeting. Abstracts for the summer meeting, July 1985 should be received at the head office, 107 Sydney Street, Brompton, London SW3 6NP, by 6 April 1985.