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CORRESPONDENCE

Correspondents are asked to be brief

Hepatitis Hazard in Clinical Laboratories

SIR,—Further to your leading article on hepatitis hazards in regular haemodialysis (2 December, p. 501) it may be useful to report some preliminary findings from the survey of laboratory staff being carried out under the auspices of the Association of Clinical Pathologists. During the period 1964-70 biochemical technicians had the highest number of cases of hepatitis and the highest attack rate, contrasting sharply with 1971, when no case was reported in this group—conceivably because of their increased awareness and improved technique and facilities. The 31 cases among staff of all types reported in 1970 and 1971 were scattered through the country and showed an even sex distribution, with 58% below the age of 30 years. Encouragingly, none was fatal and only eight required hospital admission—five of 11 Australia-antigen-positive, one of 10 Australia-antigen-negative, and two of nine untested cases.

A more detailed summary was recently presented at the Royal Society of Medicine, and in due course a more complete report of this continuing survey will be published. Only with the co-operation of numerous members of the A.C.P. has this study been made possible, and I am also grateful for the assistance given by Mrs. M. E. Martin of the Medical Records Department of Glasgow Northern Hospitals and by my secretary, Miss E. H. Simpson.—I am, etc.,

NORMAN R. GRIST

University Department of Infectious Diseases,
Ruchill Hospital,
Glasgow

Changes in Lung Capillary Permeability in Renal Failure

SIR,—Dr. W. A. Crosbie and others (18 November, p. 388) have shown that there is increased pulmonary capillary permeability to sodium in the pulmonary oedema of renal failure. In their discussion, and in the title of their paper, they imply that this change in permeability is in some way a particular feature of uraemia, albeit a feature that is seen only when the uraemic lung becomes oedematous. In doing so they ignore the work of Bauman *et al.*,[1] who showed an increase in lung capillary sodium permeability in the pulmonary oedema of heart failure, and of Lilienfield *et al.*,[2] who demonstrated an increase in pulmonary capillary thiocyanate permeability in "pulmonary congestion" of cardiac origin. It would seem from these studies that pulmonary capillary permeability increases in pulmonary oedema associated with conditions other than renal failure. It is very surprising that the authors have not studied patients in pulmonary oedema with normal renal function before attributing permeability changes to uraemia.

Chinard[3] has discussed the pulmonary capillary barrier to diffusion of small molecules (such as urea or thiocyanate) and ions. He presents evidence that the permeability of the barrier (which he believes to be the basement membrane and interstitial space) depends upon its hydration. In pulmonary oedema the gel matrix of the basement membrane and interstitium would imbibe water, swell, and become more permeable. Although this would mainly affect the passage of small particles, an increased protein permeability might be expected if the change of hydration was extreme.

If raised pulmonary capillary sodium permeability were a feature of uraemia per se, then one would expect that the lung capillaries of some non-oedematous dialysis patients would show this excessive permeability. However, non-oedematous patients with normal permeability were not found in Dr. Crosbie's study. One patient in whom three separate estimations were made showed a fall of "permeability fraction" as his oedema cleared. If the increased sodium permeability was a consequence of renal failure, then one would not expect to see the permeability return to normal in this way.

In their discussion Dr. Crosbie and his colleagues state that the capillary pressure in the lung and the plasma protein osmotic pressure may be normal in pulmonary oedema of renal failure. They opine that "a change in the permeability of the capillary wall would provide an alternative explanation" for the oedema. The importance of the permeability changes must not be exaggerated, particularly when one remembers that the permeability change could not be demonstrated in the non-oedematous state. Moreover, an increased permeability to ions and small molecules cannot of itself cause oedema in the face of normal capillary hydrostatic and osmotic pressures. Increased capillary permeability is important in oedema formation when it allows plasma proteins to cross the capillary wall. This study has shown no increase in protein permeability and therefore has added little to our knowledge of the aetiology of the pulmonary oedema of renal failure.—I am, etc.,

P. D. SNASHALL

Faculty of Medicine, University of Southampton,
General Hospital,
Southampton

1 Bauman, A., Rothschild, M. A., Yalow, R. S., and Berson, S. A., *Journal of Applied Physiology*, 1957, **11**, 353-361.