Discussion

Chairman: SIR DOUGLAS BLACK

CHAIRMAN: Dr Wilkinson showed in his patients with fulminant hepatic failure that when the effective renal plasma flow was less than approximately 150 ml/min, the Limulus test was positive. Do you have any information about the responsiveness of these patients to any form of plasma expansion?

DR S. P. WILKINSON (London): All of these patients received plasma expansion in the form of fresh frozen plasma, given for the treatment of their coagulation disorder. Although up to 2 l was infused daily, we saw no improvement in renal function.

DR P. VESIN (Paris): We have also investigated the effect of end-to-side portacaval anastomosis on renal function and in the three patients studied neither glomerular filtration rate nor renal plasma flow were improved. I would also like to comment on Dr Smith's findings. We have studied ten patients with diuretic-induced uraemia and in each case the plasma renin level had increased following the diuretics. The rise in plasma renin is more likely to be the result rather than a cause of the renal hypoperfusion.

DR I. K. SMITH (London): I agree. From our own studies, and those of Schroeder *et al.* that I quoted, we had the impression that although the plasma levels of renin were high, the turnover of the various components of the system were reduced.

DR P. OPOLON (Paris): I would like to make three points to Dr Wilkinson. Firstly, I was surprised at the

high incidence of renal failure in the patients with fulminant hepatic failure. We often see it as a terminal event, but in not more than 30% of our cases is it an important feature of the illness. Could the fact that many of your patients had taken an overdose of paracetamol account for the greater frequency? Secondly, your patients had high plasma volumes, whereas we have found low blood volumes in fulminant hepatic failure, even when the patients had not bled. Failure to correct this could be important in the development of renal failure. Thirdly, do you not think necrotic liver is a good source of endotoxin?

DR WILKINSON: In only a third of our patients with renal failure did this develop as a terminal event. In the others it was either present on admission or developed shortly after. The incidence and types of renal failure in the paracetamol group was similar to that found in those with viral hepatitis. We also have some data based on urinary lysozyme concentrations which suggest that paracetamol is not directly nephrotoxic. The high values for plasma volume that we observed may, at least in part, have been due to the use of fresh frozen plasma already mentioned. Finally, Fine's group in America analysed livers for endotoxin and showed that the normal liver does not contain endotoxin, and that if this is found at post-mortem then this is indicative of a pre-existing circulating endotoxaemia.