Discussion

Chairman: SIR DOUGLAS BLACK

CHAIRMAN: Whereas Drs Weston and Vesin appear to have had little success with diuretics in the management of renal failure, and indeed consider them to be possibly harmful, Dr Rodés had a much more satisfactory experience when diuretics were combined with plasma expansion. Have other groups combined diuretics with plasma expanders?

DR P. VESIN (Paris): We have tried combining diuretics with plasma expanders, with success in some cases, but it is very difficult to predict what will happen and, as Dr Rodés stressed, a controlled trial is necessary to evaluate this form of therapy.

DR V. LÉVY (Paris): We have also had some experience. High doses of frusemide alone have been of no value, and the combination of frusemide plus plasma expansion is a difficult treatment to control. However, plasma expansion is essential since these patients already have a depleted effective extracellular volume because of splanchnic pooling and if the frusemide induces a diuresis the ECF will become even more depleted and this will cause a further decrease in renal perfusion. Our results with this combined therapy have not always been as good as Dr Rodés described.

DR ANDREWS (London): What is the mechanism of the renal failure that these patients have?

CHAIRMAN: Surely renal ischaemia is the most important factor?

DR VESIN: The most important factor in my view is a reduction in the 'effective' plasma volume or blood volume which leads to a renal hypoperfusion.

DR E. N. WARDLE (Newcastle): But what is 'effective' plasma volume and what is the level you would wish to see in this particular situation?

DR VESIN: We know that even at the pre-ascites stage of cirrhosis the total plasma volume may be very high. Subsequently with progression of the disease, and especially in those who develop renal failure, this value may fall, even though it is still higher than that considered normal for control subjects. The important thing is the change, not the absolute figure. By 'effective' we mean that present in the systemic circulation. In the patient with portal hypertension much of the plasma volume is pooled in the splanchnic bed and is thus 'ineffective'.

DR R. WILLIAMS (London): Dr Rodés described functional renal failure as being precipitated by such factors as haemorrhage or infection. In our experience the renal failure, when precipitated in this way, more often has the features of tubular necrosis.