Discussion

Chairman: PROFESSOR SHEILA SHERLOCK

DR P. VESIN (Paris): I was impressed by the finding of Dr Davidson that the reinfusion technique improved renal function in the cases of diuretic-induced uraemia but not in the case of spontaneous functional renal failure. This point is central to our understanding of renal failure in cirrhosis, namely that plasma expansion will not improve renal perfusion in the spontaneous type of renal failure, but when it is diuretically induced such expansion is nearly always successful in reversing it.

DR H. RING-LARSEN (Copenhagen): How many of the Royal Free cases were really diuretic-resistant?

CHAIRMAN: It's impossible to say, but the use of the pump can be very worthwhile on economic considerations alone—that is to speed up the discharge of patients from hospital. The overall prognosis becomes worse the higher the dose of diuretics needed to control ascites, and neither diuretics nor an ascites pump will give you a new liver!

DR N. MACINTYRE (London): Does the patient's plasma volume determine whether or not he will have a diuresis when having a reinfusion?

DR S. P. WILKINSON (London): If the patient has good renal function then he will have a good diuresis. If he has poor renal function because of plasma volume depletion induced by diuretics then the reinfusion will correct this, after which he should also have a good diuresis.

CHAIRMAN: I'm not so sure that you can make such a clear distinction between diuretic-induced and spontaneous functional renal failure.

DR V. ARROYO (Barcelona): The distinction is clear, I think. Diuretic-induced uraemia may occur following low or high doses of diuretics and the features are of a high urine volume, a relatively high urine sodium concentration (usually greater than 30 mEq/l) and the patient is losing weight. If the diuretics are stopped renal function usually improves quickly. If you infuse saline or plasma is infused then the improvement is always very rapid. This condition carries a good prognosis. The mechanism is clear, Shear having shown that the maximal rate of transfer of fluid from ascitic compartment to plasma is about 700 ml/day and so a negative fluid balance of greater than this will induce plasma volume depletion and uraemia. In spontaneous functional renal failure, the plasma volume is already high, and in our experience, further expansion does not improve GFR or RPF. These patients have a low urinary sodium concentration and normal renal histology. The mechanism is a renal vasoconstriction.

DR WILKINSON: Reynolds and colleagues, when they analysed the frequency of functional renal failure in patients who had and had not received diuretics found that the frequency was the same in both groups.

CHAIRMAN: I still think that the spontaneous type of

renal failure can be initiated by diuretics. Our studies showed that there is a poor cortical flow in most cirrhotic patients, so the background is there on which an additional factor can so easily precipitate renal failure.

DR H. RING-LARSEN (Copenhagen): The xenon method that you used for measuring blood flow distribution, in my view, has largely been discredited. Whatever it means we have not found that frusemide administration alters the washout curve.

DR I. LAMEIRE (Tielt): We have compared the xenon method with a much more direct means of determining blood flow distribution, namely the microsphere method. There was no correlation between the two methods, although the xenon technique does appear to be a valid means of measuring total renal blood flow. As a method of determining distribution of blood flow with the kidney it is not reliable.

PROFESSOR H. DE WARDENER (London): I have never been impressed by the theory of hypoperfusion of cortical nephrons with increased perfusion of the juxtamedullary nephron as a mechanism of renal failure. What is often forgotten is that the GFR of the juxtamedullary nephron is twice that of cortical nephron so why should blood urea rise if there was such a redistribution?

Has anyone found that patients have become more amenable to diuretic therapy after reinfusion in spite of the absence of any change in glomerular filtration rate?

DR WILKINSON: Two of our patients whose GFR was between 40 and 55 did respond, after reinfusion, to the same dose of diuretics that they were previously resistant to, but in neither case was there a measurable rise in GFR.

PROFESSOR DE WARDENER: What is the natural history of functional renal failure—you see how you have bullied me into using the term—is it invariably progressive?

CHAIRMAN: We see much less of it than is, for example, experienced in the USA. If a precipitating cause, such as haemorrhage, is corrected, then it may reverse. If it occurs spontaneously then it usually carries a poor prognosis.

CHAIRMAN'S CONCLUDING REMARKS: My final comments must relate to the place, both currently and in the future, for the reinfusion machine. The main indication, I think, for it will be in the patient with very gross ascites in order to shorten his stay in hospital. We will have to be particularly careful about using it in the patient with severe liver failure as evidenced by bleeding or non-diuretic induced azotaemia. In the latter group of patients the prognosis is likely to be poor whether or not the machine is used and with or without diuretics. Dr Lévy to-day showed us a slide of a patient with 60 kilos of ascites to remove and I cannot think of a better or quicker way of doing this than by the Rhodiasc machine.