Discussion

Chairman: PROFESSOR H. E. DE WARDENER

DR J. LIBMAN (London): In what percentage of cirrhotic patients can the ascites be controlled by spironolactone alone? If spironolactone is not sufficiently effective is there any point in adding another distal diuretic such as amiloride?

DR P. VESIN (Paris): Except in those patients with marked renal hypoperfusion, aldosterone is the major factor in sodium retention. With sufficient spironolactone a good sodium diuresis can be obtained in 80–90% of cases. With reference to your second question, we have found that where amiloride or triamterene alone have been ineffective, the addition of spironolactone has produced a good diuresis. However, if spironolactone has been ineffective, then addition of amiloride has had little effect.

DR A. EDDLESTONE (London): Could I ask why you treat ascites if it doesn't produce any local symptoms?

DR VESIN: To say that the patient doesn't develop symptoms is to say that they are not accumulating

sodium. Eventually the patient will have symptoms and they tell me that these are unpleasant—but I have no personal experience!

DR T. DYMCK (Manchester): Could I ask Dr Vesin to speculate on the mechanism of the rebound urinary loss of potassium that occurs on stopping spironolactone?

DR J. LEDINGHAM (Oxford): Surely the answer to that question is that there is no better stimulus to the secretion of renin and aldosterone than diuretics, particularly those acting on the distal tubule, so that when you discontinue them you see the effects of the elevated aldosterone on the renal tubule.

DR VESIN: I have proposed that explanation in papers but do not think that it is the complete answer. I would agree that these diuretics increase plasma renin and aldosterone levels in these patients, but to go from a daily urinary loss of, say, 25–400 mEq seems to me to be more than could be anticipated even with very marked increases in aldosterone.