Discussion

Chairman: PROFESSOR SHEILA SHERLOCK

CHAIRMAN: I am most surprised at your results on the changes in portal pressure you describe, Dr Arroyo. How did you measure the pressure?

DR V. ARROYO (Barcelona): By cannulation of the umbilical vein—we were also surprised by the fall in pressure observed.

DR P. VESIN (Paris): I would like more information from Dr. Lévy about his patients with severe renal failure. Were there any patients in whom plasma volume increased without a change in GFR?

DR V. G. LÉVY (Paris): We don't have sufficient simultaneous measurements to answer that question.

DR O. JAMES (Newcastle): In about a third of Dr. Lévy's cases the reinfusion had to be stopped because of blockage of the membrane. Is this related to the protein content of the ascitic fluid?

DR LÉVY: Not necessarily, but it is more frequent when this is higher than 3 g/100 ml or when the patient has neoplastic disease.

MR S. PARBHOO (Bristol): Have you found any correlation between blockage of the membrane and either plasma or ascitic fluid fibrinogen levels? Have you looked at the membrane for fibrin? Have you tried to 'unblock' a membrane with fibrinolytic agents?

DR LÉVY: It's a deposit of protein and not fibrin so there is no indication for fibrinolytic agents.

DR A. DAVIDSON (London): We have found fibrin on the membrane and I will speak about that later.

DR C. GIPS (Gröningen): Dr Van Waes, what dose of frusemide do you use? We give 20 mg every 2 hr during reinfusion.

DR L. VAN WAES (Wondelgem): 40 mg.

DR LÉVY: In my view frusemide is the best way of inducing hypokalaemia in these patients! Without frusemide the loss of sodium compared to potassium is very high. When frusemide is given the ratio is much smaller. This is why we are against its use.

DR I. LAMEIRE (Tiel): Surely one 40 mg bolus of frusemide will not induce severe hypokalaemia?

DR VESIN: I agree with Dr Lévy. Even a small dose of frusemide can induce severe hypokalaemia in a cirrhotic and there is no way of predicting this. The loss of sodium in excess of potassium when frusemide is not given is a theoretical advantage to the cirrhotic patient.

DR P. OPOLON (Paris): Dr Van Waes, do you give your patients a sodium load during reinfusion as we often do?

DR VAN WAES: Never. We give frusemide to prevent pulmonary oedema.

CHAIRMAN: It seems ridiculous to me to give a sodium load when you are trying to get rid of sodium.

DR OPOLON: A point of disagreement with Dr Arroyo—we do not think that variceal haemorrhage is a contra-indication to reinfusion, since it can be controlled with a Sengstaken-Blakemore tube.