Mean ($\pm$ SD) basal skin flow changes (% plus or minus) during local and contralateral hand cooling tests before and during naftidrofuryl treatment

Test	Before treatment	During treatment	P value
Local cooling	-21.5 $\pm$ 26.9	-9.0 $\pm$ 18.5	P = 0.001
Normal range	+ 7.5 $\pm$ 19.6		
Contralateral hand cooling	-49.2 $\pm$ 19.5	-25.4 $\pm$ 12.1	P < 0.001
Normal range	-23.4 $\pm$ 10.4		

test. Individual cooling test results that were within the normal range before the treatment were not much changed by naftidrofuryl. Five patients had epigastric discomfort while taking naftidrofuryl. This was the only side effect.

Comment

The mechanisms causing RP can be divided into systemic microvascular reflexes and local mechanisms (local hyperreactivity of the blood vessel walls and increased local blood viscosity)—the relative importance of each differing in individual patients—and an already decreased basal flow when such mechanisms are normal.² Earlier studies^{3,4} suggest that naftidrofuryl helps in various peripheral vascular disorders without causing hypotension and flushing. It enhances the tissues' oxidative potentials after conditions of raised anaerobic tissue metabolism.⁵ This study shows that naftidrofuryl has a beneficial effect on the basal skin flow and both the reflex and the local mechanisms underlying RP. Its effect on the microvascular reflexes was most pronounced, indicating that a better clinical effect might be expected in patients in whom these are impaired. A controlled clinical trial is therefore warranted in patients selected on these grounds. They will be found mostly among patients with RD, RA, and possibly SLE.²

I thank Lipha Pharmaceuticals Ltd for placebo capsules.

¹ Haavik Nilsen, K, *Microvascular Research*, 1978, **15**, 251.

² Haavik Nilsen, K, *Scandinavian Journal of Rheumatology*, 1978, in press.

³ Forrest, H, *Scottish Medical Journal*, 1978, **23**, 45.

⁴ Judge, T G, *Scottish Medical Journal*, 1978, **23**, 43.

⁵ Shaw, S W J, and Johnson, R H, *Acta Neurologica Scandinavica*, 1975, **52**, 231.

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Plasma lipoproteins in renal transplant patients

Changes in plasma lipoprotein patterns in chronic renal failure and after renal transplantation were reported by us,¹ and our observations have been confirmed by others.² We have re-examined the plasma lipids in 17 of our original patients four years after our initial investigation.

Patients, methods, and results

Of our 32 original patients in 1971, 17 were available for study in 1975, exactly four years later. Six of the remainder had died, five had rejected their allograft and been returned to haemodialysis, and four were unavailable for study. Out of the six who died, two had myocardial infarcts, two had renal failure due to rejection, one had haemorrhage from a duodenal ulcer, and one had a subarachnoid haemorrhage. The group consisted of seven men and 10 women with an average age of 41 years (21-56). Each patient was seen and examined after a 12-hour fast in a renal transplant follow-up clinic. Venous blood samples were analysed by semiautomated techniques

for cholesterol and triglycerides and lipoprotein patterns electrophoretically according to techniques previously described.¹

The comparative body weights, prednisolone dose per day, and plasma cholesterol and triglyceride concentrations of the 17 patients studied in 1971 and 1975 are shown in the table. During this follow-up period the dose of corticosteroids had been gradually reduced so that in the second study period the average ($\pm$ SD) dose (11 $\pm$ 1.1 mg/day) was virtually half that used in the initial period (21 $\pm$ 2.3 mg/day). Concomitantly the plasma cholesterol and triglyceride concentrations had significantly fallen and were within the normal range. Furthermore, the lipoprotein patterns had returned to normal in 11 cases, only two remaining in class IIa and four in class IV. There had been no significant change in glomerular filtration rate over this period (63.5 $\pm$ 6.1 ml/min and 73.9 $\pm$ 7.6 ml/min).

Comparative mean ($\pm$ SD) plasma triglyceride and cholesterol concentrations, glomerular filtration rates, body weights, and prednisolone dose/day in 1971 and 1975 in renal transplant patients

	1971 (n = 32)	1975 (n = 17)
Triglycerides (mmol/l)	2.4 $\pm$ 0.2	1.4 $\pm$ 0.1*
Cholesterol (mmol/l)	6.5 $\pm$ 0.3	5.2 $\pm$ 0.2*
GFR (ml/min)	63.5 $\pm$ 6.1	73.9 $\pm$ 7.6
Weight (kg)	61.4 $\pm$ 2.6	60.2 $\pm$ 2.7
Prednisolone (mg/day)	21 $\pm$ 2.3	11 $\pm$ 1.1*

*P < 0.001 (paired t test).

Conversion: SI to traditional units—Triglyceride: 1 mmol/l $\approx$ 86 mg/100 ml. Cholesterol: 1 mmol/l $\approx$ 39 mg/100 ml.

Comment

Vascular disease is the commonest cause of death in patients with chronic renal failure on haemodialysis. Cardiac and vascular accidents account for over 30% of all deaths in patients with renal transplants, taking second place only to infections.³ Our initial study in 1971¹ was prompted by a similar clinical observation. Like other authors, we found substantial hypertriglyceridaemia in the non-nephrotic renal failure group, whereas in the transplanted individuals taking an average prednisolone dose of 21 mg/day cholesterol, triglyceride, and non-esterified fatty acid levels were significantly raised. Insulin concentrations remained normal throughout. Postulating that these raised values could well be related to the corticosteroid treatment, this further study was undertaken four years later when the corticosteroid dose in the original patients had been reduced from a mean of 21 mg to 11 mg/day. A pronounced improvement was observed. Most patients had normal concentrations of cholesterol and triglycerides, and lipoprotein patterns had returned to normal.

¹ Ghosh, P, *et al*, *Transplantation*, 1973, **15**, 521.

² Casaretto, A, *et al*, *Lancet*, 1974, **1**, 481.

³ Gurland, H J, *et al*, *Proceedings of the 13th European Dialysis and Transplantation Association*, ed B H B Robinson, vol 13, p 38. London, Pitman Medical, 1976.

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Prenatal diagnosis of a galactosaemia heterozygote by fetal blood enzyme assay

Although dietary exclusion of galactose greatly improves the prognosis of galactosaemia, in all six pregnancies referred to us where the mother had given birth to an affected child the parents requested diagnostic amniocentesis so that they could choose to have only unaffected children. We describe such a mother who refused to accept the risk of having another affected child but who presented at 20