The mean number of cigarettes smoked by smokers was 14 a day (range 4-30). The ex-smokers were smoking 16 cigarettes a day (range 4-40) before giving up.

A total of 16 (14 men, 2 women) of the 33 smokers had had fistula failures compared to only two (one man, one woman) of the 32 ex-smokers and five (four men, one woman) of the 36 non-smokers. The incidence of fistula failure was significantly higher ($\chi^2 = 16{\cdot}32$, $p < 0{\cdot}001$) in smokers than in ex-smokers and non-smokers combined. Of the seven fistula failures in non-smokers and ex-smokers three occurred immediately after operation. No similar association could be established with any fistula failure in smokers.

Comment

Brescia *et al* described the use of the arteriovenous fistula for chronic haemodialysis in 1966[3] and since then its use has become well established. Several long term complications have been described, however, including thrombosis, local infection, septicaemia, pseudoaneurysms, aneurysmal dilatation, neurological complications, steal syndrome, ischaemic peripheries, pain, and cardiac failure.[4] Thrombosis and infection are the most common.

Cigarette smoking is an aetiological factor in many diseases, particularly vasculo-occlusive disorders,[5] so it is not surprising that late occlusion and thrombosis in arteriovenous fistulas occur more often in cigarette smokers than in non-smokers.

We have looked at a number of other factors that might influence the long term patency of fistulas but we have found no association between failure and age, sex, hypertension, length of time on dialysis, or renal disease.

We thus feel that all patients undergoing haemodialysis using an arteriovenous fistula should be strongly advised to refrain from cigarette smoking.

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[2] Wolin L. Arterio-venous shunts for prolonged intermittent haemodialysis. *JAMA* 1967;**202**:99.

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(*Accepted 13 December 1982*)

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Transient autonomic and sensory neuropathy in newly diagnosed insulin dependent diabetes mellitus

Insulin dependent diabetics may develop a peripheral sensory or motor neuropathy when first diagnosed. This may deteriorate for a short time when insulin treatment is started and then clears completely, being unrelated to any subsequent chronic neuropathy. Similarly transient reversible changes in the somatic nervous system may be found during periods of impaired diabetic control. We report a case of transient autonomic neuropathy affecting the bladder in a newly diagnosed insulin dependent diabetic.

Case report

A 44 year old woman was admitted as an emergency case on 2 January 1981 with a 48 hour history of anorexia, vomiting, drowsiness, and confusion. On admission she complained of shortness of breath, though direct inquiry elicited no other definite symptoms. Examination showed tachypnoea and ketosis. She was drowsy, though there were no other abnormal neurological findings; optic fundi were normal. Investigations confirmed a diagnosis of diabetic ketoacidosis and she was treated with neutral soluble insulin, initially by continuous infusion. Further investigations showed evidence of septicaemia with *Escherichia coli* and vulvovaginitis due to *Candida albicans*; midstream urine culture also grew *C albicans*. She was treated with parenteral cephalosporins and local and oral nystatin; blood sugar values eventually stabilised on a 1000 kcal (4·2 MJ) diet in addition to Rapitard insulin 40 units in the morning and 24 units at night.

While in the ward she complained of urinary incontinence on standing and on walking. There was no incontinence on coughing, nor urgency or urge incontinence, and no symptoms of voiding difficulty. She denied urinary symptoms before admission.

When seen for urodynamic assessment on 28 January her symptoms had changed to those of impaired bladder sensation. She was having to strain to void in addition to having occasional postmicturition dribbling and incontinence on standing. She also complained of vague areas of impaired sensation over her back and face and of ageusia, which had been present since the start of insulin treatment. On examination the only notable findings were of impaired pinprick sensation affecting both legs to mid-calf. Facial and perineal sensation were normal and there were no retinal changes.

Dual channel subtracted cystometry performed by a rapid fill saline technique showed no evidence of either bladder sensation during filling or detrusor activity during voiding phases of the cystometrogram. Voiding was achieved to completion by abdominal straining (table). Despite this evidence of neuropathy there was no residual urine, the urine remained sterile, and there was nothing to suggest upper tract damage. No active intervention was therefore proposed.

Urodynamic findings at initial presentation and four months later

	28 January	15 May
Residual volume (ml):		
Before investigation	30	0
After investigation	0	0
First sensation of filling (ml)	None	450
Cystometric capacity (ml)	1250*	800
Pressure rise at:		
500 ml (cm H_2O)	15	6
Capacity (cm H_2O)	25	18
Maximum voiding pressure (cm H_2O)	(All strain)	30
Peak flow rate (ml/s)	12	19
Flow pattern	Strain	Normal

*Filling discontinued, though no sensation of filling or urge to void had occurred.

Four months later the diabetic control remained good and urinary symptoms were improved; urodynamic values showed a return towards normal (table). By nine months after presentation she was free from urinary symptoms, bladder sensation was normal, and she voided without straining.

Comment

While it is certainly possible that the transient autonomic neuropathy in our patient represented a plaque of demyelination, visual evoked responses were normal and we think that the weight of circumstantial evidence is very much in favour of a diabetic aetiology. Though it has been suggested that autonomic nerves may be less susceptible to metabolic damage than somatic fibres,[1] Hreidarsson[2] detected reversible changes in the pupillary responses of diabetics at times of poor control. Cystometry, like pupillography, offers a good opportunity to study the state of the autonomic nervous system, and our patient showed that reversible changes in the system may occur analogous to the previously recognised reversible changes in the somatic nervous system.

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(*Accepted 9 December 1982*)

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