pulmonary shunt effect and decrease right-to-left shunting through the ductus arteriosus, thus increasing arterial oxygenation.

The mechanisms influencing pulmonary vascular resistance in the face of hypoxia, hypercarbia, and acidosis are complex and we suggest that an agent such as sodium nitroprusside, which acts directly on blood vessels,⁴ is more likely to achieve effective pulmonary vasodilatation than one which acts by blocking autonomic receptor sites.

This view is supported by two facts. Firstly, cases of idiopathic respiratory distress syndrome treated with phenoxybenzamine,⁵ while showing a decrease in acidosis, have a much delayed rise in arterial oxygenation compared with the instantaneous effect of sodium nitroprusside seen in our case. Secondly, the systemic blood pressure rose in response to sodium nitroprusside. We suggest that sodium nitroprusside has a place in the management of patients with idiopathic respiratory distress syndrome who continue to deteriorate despite orthodox intensive care.

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(Accepted 20 January 1978)

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Reversible leg ischaemia due to diuretics

We wish to draw attention to a possible complication of over-enthusiastic diuretic treatment in the elderly. Five patients were referred by their practitioners over the past two years with leg ischaemia or superficial gangrene of recent onset. Three patients had good foot pulses and none had pain. All showed clinical evidence of appreciable dehydration, and had been on varying doses of diuretics for lengthy periods. Withdrawal of diuretics and rehydration alone resulted in dramatic improvement of their leg circulation and healing of gangrene within 48 hours. We believe that the diuretic treatment was responsible for precipitating the peripheral vascular changes in these patients. Vascular complications due to diuretics have been reported,^{1 2} but we can find no reference to leg ischaemia as a side effect.

Details of patients

Case No:	1	2	3	4	5	Normal range
Sex and age	F 84	M 79	F 71	F 85	M 78	
Daily diuretic(s)	Frusamide 80 mg	Frusamide 80 mg, amiloride 5 mg	Frusamide 40 mg, amiloride 5 mg	Frusamide 40 mg, amiloride 5 mg	Frusamide 120 mg	
Clinical presentation	Confused with swollen mottled right foot	Cold cyanosed feet and gangrenous patch of skin on right hallux	Confused and lethargic with mottled cold feet and superficial gangrene of left 1st and 2nd toes	Confused with pale cold left foot and an area of gangrene on 5th toe	Painless cold mottled ischaemic feet	
Pulses	To knees	In feet	To knees	In feet	In feet	
Hb (g/dl)	Not done	18.1	10.8	11.6	13.0	M 14-18, F 12-16
PCV (%)	Not done	52.5	38.1	45.4	40.2	M 42-52, F 37-47
Platelets ($\times 10^9/l$)	Not done	110	525	600	200	200-400
Urea (mmol/l)	Not done	12.0	25.0	13.5	7.5	2.5-7.5
Na (mmol(mEq)/l)	Not done	138	118	126	147	133-143
K (mmol(mEq)/l)	Not done	4.8	3.9	4.8	3.8	3.5-5.0

Conversion: SI to traditional units—Blood urea : 1 mmol/l $\approx$ 6 mg/100 ml.

Patients

Clinical details and the results of investigations are given in the table. All the patients were taking digoxin with their diuretics, but there was no other relevant drug treatment. The results of coagulation studies and serum protein estimations were normal in all. Evidence of haemoconcentration was shown by raised haemoglobin concentrations and packed cell volume in case 2, and by a raised platelet count in cases 3 and 4. In the latter two cases the presence of anaemia probably explained a normal packed cell volume. Cases 2, 3, and 4 had raised blood urea concentrations, and cases three and 4 were hyponatraemic. All these changes reverted to normal after rehydration.

Comment

Diuretics may cause ischaemia with leg gangrene because they can produce dehydration and hypovolaemia.³ The resultant poor tissue perfusion and increased blood viscosity with hypercoagulability might lead to occlusion of the distal small blood vessels.⁴ Another postulated mechanism for diuretic-induced ischaemia is intracellular swelling due to electrolyte changes and vasospasm,¹ and tissue necrosis without organic vascular occlusion has been reported in acute illnesses with dehydration and shock.⁵ The commonly recognised causes of gangrene include numerous vascular diseases, embolism, diabetes, and changes in blood constituents, but in our patients the major contribution appeared to be inappropriate diuretic intake and subsequent dehydration.

Overuse of powerful diuretics is common and often the initial dose for the acute condition is continued unnecessarily. There are numerous side effects of diuretic treatment,³ but less well known complications are mesenteric infarction,¹ and transient cerebral ischaemic attacks.²

Because of underlying disease elderly patients may be more at risk of ischaemic complications. In them similarly induced ischaemic changes may be more widespread than in younger patients, affecting more vital organs (such as the brain, heart, kidneys, or lungs), and initiating or increasing degeneration in these tissues.

Clinicians should consider restricting the long-term use of diuretics, particularly in the elderly who, while being those more likely to need these drugs, are also more at risk of developing complications.

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(Accepted 5 January 1978)

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