**Femoral Arterial Thrombosis in Nephrotic Syndrome**

The loss of a limb is always a tragedy, above all in a child. Primary arterial thrombosis is rare in childhood, but in this patient appears to be explicable on the ground of the tendency to coagulation found in the nephrotic syndrome, corticosteroid therapy, and femoral vein puncture.

### Case Report

A girl presented at the age of 1 year 9 months with a typical pure nephrotic syndrome, and renal biopsy revealed a ‘minimal change’ lesion. She was treated with prednisolone, but relapsed and remained on prednisolone for 7 months. She again relapsed following an upper respiratory tract infection and mild gastroenteritis. The dose of prednisolone was increased to 30 mg/day and a week later she was admitted to the West Kent General Hospital with cold extremities, impalpable peripheral pulses, and a blood pressure of 80/60 mmHg (12.7 cm cuff). Bladder catheterization revealed oliguria, and with some difficulty blood was obtained by femoral puncture; both groins were needled at this time. The blood urea was 146 mg/100 ml, and the patient was transferred to Guy’s in case she should require dialysis. On arrival her blood pressure had improved to 135/85 mmHg but within six hours it was clear that the arterial supply of the right leg was obstructed, with no pulse palpable below the femoral. Infusion of low molecular weight dextran produced no improvement. Arteriography revealed an obstruction at the popliteal artery which was then explored; an embolus with distal clot was found and removed. A femoral arterial catheter was left in situ, and through this heparin, urokinase, bretylium tosylate and tolazoline hydrochloride were variously infused without effect. Tolazoline hydrochloride was also given orally, and the child was maintained intermittently in hyperbaric oxygen. Despite desobilateration of the posterior tibial artery as far as the ankle, the foot remained grossly ischaemic with a line of demarcation at the hind foot, and four weeks later Mr. J. Batchelor performed a through-knee amputation.

During this part of her course the nephrotic syndrome was controlled with prednisolone 30 mg/day, leading to hypertension (diastolic 120–140 mmHg) treated with methyldopa. When healing of the stump was satisfactory, cyclophosphamide therapy was begun (Moncrieff et al., 1969). Assessment at this time showed a grossly Cushingoid child with a height of 86 cm (10th centile) and a blood pressure up to 160/120 mmHg.

Cyclophosphamide was continued for 16 weeks while prednisolone and methyldopa were gradually withdrawn. Seven months later she was walking well with her artificial limb and was on no drugs. Her blood pressure and appearance were normal and her urine remains protein-free in spite of occasional upper respiratory infections and routine immunizations. She had grown 7 cm and was on the 25th centile for height.

### Discussion

A number of factors may have contributed to what seems certain to have been a femoral artery thrombosis, with subsequent embolism of clot into the popliteal artery.

*Increased coagulability* of the blood of patients with the nephrotic syndrome has been discussed by a number of authors, but the exact mechanism is not clear. An increase in a number of coagulation

---

**References**


D. N. Challacombe,* M. Sandler, and Jennifer Southgate

*Present address: Institute of Child Health, Francis Road, Birmingham 15.*

From the Bristol Royal Hospital for Sick Children, and the Bernhard Baron Memorial Research Laboratories and Institute of Obstetrics and Gynaecology, Queen Charlotte’s Maternity Hospital, London W.6.

Correspondence to Dr. M. Sandler, Queen Charlotte’s Maternity Hospital, Goldhawk Road, London W.6.
is increasing from corticosteroid to and the nephrotic lesion in phosphamide it seems nephrotic cannot embolism However, some probably year femoral nephrotic reported after femoral Femoral It is and receiving larly likely femoral vein and trauma to femoral vein tion of coagulation and fibrinolysis Kendall, 1966) in plasma and X, 216 A view of children there with the nephrotic syndrome, in case, severe oedema result in this case, severe oedema result in this trauma to the femoral artery during attempts at femoral vein puncture. This would be particularly likely to occur in a shocked, nephrotic patient where venoconstriction and oedema would make it difficult to obtain a sample. Goldbloom, Hillman, and Santulli (1967) reported three nephrotic children receiving corticosteroids in whom femoral vein puncture was followed by thrombosis of the femoral artery and amputation, with death in one case. Femoral artery thrombosis has also been reported after femoral vein puncture in non-nephrotic infants (Nabseth and Jones, 1963). It is difficult to assess the amount of risk; thousands of femoral vein punctures are performed in children every year without incident (McKay, 1966), and probably some dozens of these are on nephrotic children. However, spontaneous arterial thrombotic episodes in children who do not suffer from embolism are extremely rare. It seems reasonable to us that femoral vein puncture should be avoided in nephrotic children unless the sample is vital and cannot be obtained elsewhere.

In view of the good results obtained with cyclophosphamide in children with the 'minimal change' lesion and the nephrotic syndrome (Moncrieff et al., 1969), it seems reasonable to add thrombotic episodes to the indications for transferring patients from corticosteroid therapy to this drug. Our patient had, in any case, severe steroid toxicity and increasing resistance to treatment.

Summary
A 2½-year-old girl with the nephrotic syndrome is described. During a relapse she developed femoral artery thrombosis leading to a through knee amputation. At the time of the incident the patient was hypotensive with a raised blood urea, was receiving corticosteroids, and had bilateral femoral vein punctures to obtain blood for electrolytes. All these factors could have contributed. We conclude that femoral vein puncture should be avoided where possible in patients with the nephrotic syndrome, and that thromboembolic incidents are an indication for the use of cyclophosphamide in these patients.

References
McKay, R. J., Jr. (1966). Diagnosis and treatment; risks of obtaining samples of venous blood in infants. Pediatrics, 38, 906.
J. S. CAMERON,* C. S. OGG, F. G. ELLIS, and M. A. SALMON
From Guy's Hospital, London, and the Park Hospital, Oxford.

Malignant Sacrococcygeal Teratoma—A Problem in Diagnosis

Sacrococcygeal teratoma occurs in about 1 in 40,000 births (Gelb et al., 1964), and presents at birth as a sacrococcygeal mass which may vary considerably in size. It is predominantly a female condition. Failure to recognize and treat adequately surgically at this time will result in a fatal outcome in what should be primarily a benign condition.

Berry, Keeling, and Hilton (1969) reported 58 sacrococcygeal teratomas seen at The Hospital for Sick Children, Great Ormond Street, London, over a 35-year period up to 1969. 10 of these were or