Heparin therapy in meningococcal septicaemia

Sir,

The occurrence of disseminated intravascular coagulation in meningococcal septicaemia has now been well established from fatal cases (Fox, 1971) and from laboratory studies (McGehee, Rapaport, and Hjort, 1967). The importance of early recognition of this condition in the management of meningococcal septicaemia has recently been emphasized by Hunter (*Archives*, 1973, 48, 233). In the following case simple and quick laboratory confirmation of clinical suspicion led to an early application of anticoagulant therapy, with satisfactory recovery.

Case report

A 3-year-old boy who had previously been well became irritable after breakfast, developed a fever at midday, became anorexic, and then vomited. 3 hours later he was noted to be drowsy and to have developed a generalized rash. He was transferred immediately 60 miles to the regional hospital where he was seen 12 hours after the onset of symptoms. He was found to be irritable with poor peripheral circulation, pulse 170/min, systolic blood pressure 70 mmHg, and temperature 40.5 °C. Petechiae involved the entire body, including the palate and buccal mucosa. He had marked neck stiffness and an easily elicited Kernig's sign. Fundi were clear; spleen was palpable. CSF contained 12 white cells/mm³ and a total protein of 42.3 mg/100 ml, but was sterile on culture. Hb 12.4 g/100 ml, haematocrit 36%, white blood count 7000/mm³, platelets 76,000/mm³; prothrombin time 24 seconds (control 16.5 sec). Serum fibrin degradation products on the same blood by the rapid slide Latex agglutination test were >10 µg/100 ml and <40 µg/100 ml.* Intra-

venous penicillin G 500,000 units 6-hourly, sulphadiazine 500 mg 6-hourly, and hydrocortisone 50 mg 6-hourly were begun, together with intravenous heparin.

A heavy growth of meningococcus in the nasal swabs and in the blood cultures was reported.

Within 12 hours white blood count rose to 24,000/mm³ with 75% polymorphs and platelets to 128,000/mm³; thereafter recovery was rapid and complete.

After the initial 48 hours of treatment, the hydrocortisone and heparin were reduced in graduated doses over 3 days. He was discharged after 2 weeks and short-term follow-up showed no neurological deficit.

The clinical picture on admission was that of meningococcal septicaemia with an unfavourable prognosis, since (a) petechiae appeared 6 hours before admission, (b) CSF contained 12 white cells, and (c) white blood count was 7000/mm³ (Stiehm and Damrosch, 1966; Hunter, 1973). Disseminated intravascular coagulation was suspected on clinical grounds and was substantiated by the low platelet count and prolonged prothrombin time.

A. D. SMITH

Department of Paediatrics,
International Grenfell Association,
St. Anthony, Newfoundland.

REFERENCES

- Das, P. C., Allan, A. G. E., Woodfield, D. G., and Cash, J. D. (1967). Fibrin degradation products in sera of normal subjects. *British Medical Journal*, 4, 718.
- Fox, B. (1971). Disseminated intravascular coagulation and the Waterhouse-Friderichsen syndrome. *Archives of Disease in Childhood*, 46, 680.
- Hunter, J. (1973). Heparin therapy in meningococcal septicaemia. *Archives of Disease in Childhood*, 48, 233.
- McGehee, W. G., Rapaport, S. I., and Hjort, P. F. (1967). Intravascular coagulation in fulminant meningococccemia. *Annals of Internal Medicine*, 67, 250.
- Stiehm, E. R., and Damrosch, D. S. (1966). Factors in the prognosis of meningococcal infection. *Journal of Pediatrics*, 68, 457.

*Thrombo-Wellcotest mean normal level 4.9 ± 2.8 µg/ml (Das *et al.*, 1967).