Failure of heparin to alter the outcome of pneumococcal meningitis

SIR,—Dr J T MacFarlane and his colleagues (10 December, p 1522) interpret the finding of fibrin degradation products (FDP) in cerebrospinal fluid (CSF) as evidence for cerebral vascular thrombosis in pneumococcal meningitis. Treatment with heparin, however, failed to alter the outcome or hasten the rate of fall of FDP in CSF.

In a study of 252 patients admitted to a neurological/neurosurgical unit[1] we detected FDP in CSF in 23·4% of patients. The FDP were commonly present in association with other low-molecular-weight coagulation proteins and, while present in meningitis, were also detected in neoplastic and vascular neurological disorders (including subarachnoid haemorrhage). Leakage of FDP and other proteins across a damaged blood-CSF barrier can occur in a variety of neurological conditions and their presence in CSF does not necessarily reflect either vascular thrombosis or a secondary increase in fibrinolytic activity within the subarachnoid space.

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[1] Anderson, M, *et al*, *Journal of Clinical Pathology*. In press.

Reticulocytopenia in immune haemolytic anaemia

SIR,—We read with interest the report of Dr U M Hedge and others (3 December, p 1444) and note that two of their patients were children.

Of the last nine children (ages 1½-7 years) seen in this department with "immune" haemolytic anaemia, five have had low reticulocyte counts at presentation (see table). When their haemoglobin concentrations were at their lowest four had positive direct antiglobulin tests of complement-binding type and cold non-specific autoagglutinins were identified in their serum. The range of reticulocyte counts was 2·7-10·8% (median 6·6%) compared with 34-49% (median 39·5%) for those with "warm" antibody immune anaemia. One further patient had 2·7% reticulocytes at presentation and was of interest in that she had an antibody directed against the group A antigen. The consistent features for all those with reticulocytopenia was their association with infection, usually viral (for example, adenovirus 5 upper respiratory infection and myocarditis in patient 9) and their rapid recovery, usually following commencement of steroids. The latter rapidly caused a rise in reticulocytes up to the range seen in the "warm" types.

We presume that in these "Lederer-type" anaemias the antibodies induced are also directed against young red cells, but we could always detect them by the direct Coombs test, albeit in low concentration.

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Clinical and laboratory features in nine children with "immune" haemolytic anaemia

Age at onset (years)	Type of onset	Haemoglobin (g/dl)	Reticulocytes (%)	Serology	Subsequent course
5	Gradual	4	45	"Warm" antibody	Splenectomy. Slow recovery
1½	Gradual	9·3	34	"Warm" antibody	Rapid recovery
7	Gradual	5·6	34	"Warm" antibody	Very slow. Splenectomy
5	Gradual	10·2	10·4	Cold agglutinins	(Mycoplasma) Rapid recovery
1 11/12	Gradual	4	49	"Warm" antibody	Fairly rapid recovery
4	Acute	6	2·7	Cold agglutinins	Rapid recovery
5	Acute	3·8	10·8	Cold agglutinins	Rapid recovery
2	Gradual	4·1	2·7	IgM anti-A antibody	Rapid recovery
1 7/12	Acute	5·6	3·6	Cold agglutinins	Rapid recovery

Bronchiectasis in acute leukaemia

SIR,—In their recent paper (1 October, p 857) Dr P J Kearney and his colleagues ascribe great value to prophylactic antibiotics for bronchiectasis in acute leukaemia. They administer four antibiotics in a rotating scheme but give no details about the effect of this regimen.

In three of their patients the sputum cultures showed, among others, *Streptococcus viridans*, most probably because of contamination of the specimen with mouth flora. The *Neisseria* spp cultured from the sputum of three patients were possibly pathogenic, but here too contamination with flora of the oropharynx would have to be ruled out.[1] With respect to *Haemophilus influenzae*, permanent eradication is very difficult. However, the most important pathogen is the pneumococcus in the sputum (typing would be useful to assess virulence here). We prefer in such cases to give only one course of a narrow-spectrum penicillin (for example, phenethicillin), if possible in combination with isolated nursing of the patient during granulocytopenia.

As a rule we are very reluctant to use antibiotics prophylactically either in cases of bronchiectasis or in granulocytopenic patients because of the risk of colonisation and infection with multiresistant Gram-negative micro-organisms. Recently, we saw a severely neutropenic patient with bronchiectasis colonised with highly resistant Gram-negative micro-organisms. Because of its relevance we describe this case briefly.

A 38-year-old woman with bronchiectasis since childhood and still persisting in the remaining lung tissue despite three operations had been treated with numerous courses of antibiotic therapy by many different physicians. Since 1961 she had had a slight pancytopenia of unknown cause. In December 1976 the pancytopenia became more severe and the diagnosis of aplastic anaemia was made.

On admission to the isolation ward of our hospital in March 1977 there was bronchorrhoea and cultures of the washed sputum[1] repeatedly showed a large number of highly resistant *Alkaligenes faecalis* and *Pseudomonas aeruginosa*, probably as a consequence of previous antibiotic therapy. Because of the impossibility of eradicating these micro-organisms and the absence of pulmonary infiltrates treatment was restricted to very intensive physiotherapy and neither systemic antibiotic therapy nor antibiotic inhalation was used. However, partial antibiotic decontamination of the oronasopharynx and gut was carried out[2] and she was nursed in a laminar flow isolator. For more than 10 weeks she had less than $0{\cdot}5 \times 10^9$ granulocytes/l (500/mm³) and less than $0{\cdot}1 \times 10^9$ granulocytes/l (100/mm³) for 10 days. The aplastic anaemia was treated with 15 mg prednisone daily and she also received a course of rabbit antithymocyte globulin (700 mg in 4 days).[3] Probably as a result of the latter treatment the bone marrow recovered partially (the granulocyte count rose to about 1×10^9/l (1000/mm³)). During the entire course the large numbers of *A faecalis* and *P aeruginosa* persisted, but pulmonary infiltrates and infections at other sites have not developed so far.

We are of course aware that the success obtained in this case does not constitute proof that our policy is the right one.

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[1] Mulder, J, *Proceedings of the Royal Society of Medicine*, 1956, **49**, 773.
[2] Guiot, H F L, and van Furth, R, *British Medical Journal*, 1977, **1**, 800.
[3] Speck, B, *et al*, *Lancet*, 1977, **2**, 1145.

Hair in the theatre

SIR,—Human hair is often contaminated with *Staphylococcus aureus*[1,2]; ruffling it disperses bacteria-carrying particles, and it is generally accepted that the hair on the heads of people in operating theatres should be completely covered during operations. However, the modern fashion which dictates that men should have long hair has apparently caused them to forget the principle.

Surgeons from hospitals all over the country have appeared in their operating theatre clothes in a number of recent television programmes. Few of them have been wearing caps which covered all their hair. Instead they have sported caps which perched inadequately on hairy heads. In contrast, the nurses' hair has always been properly covered. Hoods which cover all the surgeon's hair are available, but in my experience the men complain that their heads get too hot if they wear them.

Surely the answer is that surgeons and anaesthetists must either put up with the heat, put up their hair in buns, or get it cut. Would it be too great a sacrifice, or like Samson would they lose their strength? Long hair is banned in factories; why should it be tolerated in operating theatres?

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[1] Summers, M M, Lynch, P F, and Black, T, *Journal of Clinical Pathology*, 1965, **18**, 13.
[2] Noble, W C, *Journal of Clinical Pathology*, 1966, **19**, 570.