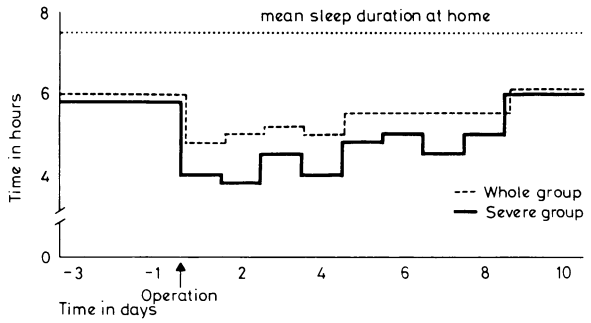
Routine hypnotics and pain relief were prescribed by ward staff not associated with the study.

Though five patients claimed to be bad sleepers at home and four more regularly took hypnotics, the striking finding was a reduction of the mean duration of sleep both before and after surgery over the ten days of the study (see figure). More detailed analysis of the sleep patterns showed that the deficit was principally due to early awakening, though there was also more delay in getting to sleep. Patients also recorded a decrease in the quality of the sleep, increased wakenings, and, in the first three postoperative days, much disturbance by both noise and pain. Only two-thirds of the severe group and one-third of the mild group received intramuscular analgesia on the first postoperative night.



Sleep duration in hospital.

Discussion

We have provided evidence to support anecdotes that acute hospitals with open wards are not places in which it is easy to obtain a sound night's sleep. Two other points are suggested by the results of this study: firstly, that pain relief receives less attention than it should; and secondly, that the further decrease in sleep after injury may, because it is long lasting, represent a primary effect of surgery and anaesthesia on the brain.

Copies of the sleep questionnaire and the detailed data are available from HD.

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Failure of heparin to alter the outcome of pneumococcal meningitis

Pneumococcal meningitis continues to carry a high death rate, especially in tropical Africa.[1] The reason for this is not clear.[2] Post-mortem studies[3] and the presence of raised concentrations of fibrin degradation products (FDP) in the cerebrospinal fluid (CSF) of patients suggest that cerebral vascular thrombosis may be important. We therefore tried treating pneumococcal meningitis with heparin.

Patients and treatment

From November 1976 to March 1977 15 adults with proved pneumococcal meningitis were admitted to this hospital. They were allocated randomly to control or heparin-treatment groups. All patients received intravenous penicillin in a dose of 24 megaunits/24 hours for five days, followed by a smaller intramuscular dose for a further five days. Patients treated with heparin received an initial intravenous dose of 10 000 units followed by 30 000 units/24 hours for five days given by continuous intravenous infusion. Clotting times, checked regularly, were always less than twice the control.

Detailed clinical observations were made daily on all patients. Blood and CSF were collected on admission, one, two, three, and five days later and, in the survivors, 24 hours after ending penicillin treatment. All CSF samples were examined by Gram stain and cultured on blood agar. CSF leucocyte and bacterial count, protein content, and lactate concentration were measured by standard techniques, and serum and CSF were tested for pneumococcal antigen by countercurrent immunoelectrophoresis.[4] Platelet counts, FDP concentrations, and kaolin cephalin clotting times were measured by standard methods.

The presenting clinical features in the two groups were similar (table). Four patients treated with heparin died but among the controls only two deaths occurred. Persistent blood staining of CSF was observed in the daily samples of three of the patients treated with heparin but in none of the controls.

Two patients in the control group showed laboratory, but no clinical, features of mild disseminated intravascular coagulation and one of these patients died. Gram-positive cocci were seen in the CSF of four of five surviving heparin-treated patients three days after the start of treatment and in two of six surviving controls. The results of all cultures were negative at this time. Cerebrospinal fluid cell and bacterial counts, protein, lactate, and FDP concentrations were all high on presentation and fell progressively during treatment but no significant differences were found between the heparin-treated and control patients.

Comment

Intravenous heparin in the dose that we used is of no value in treating pneumococcal meningitis. Four of the six patients who died were treated with heparin and new focal neurological signs developed in four heparin-treated patients but in only two controls.

The persistent blood staining of the CSF in three patients treated with heparin suggests that heparin may be dangerous in this condition. Owing to local beliefs a necropsy could not be performed to see if heparin had contributed to the death of patients in this treatment group. In one patient, however, bilateral burr holes were made after the development of a hemiplegia, but no cerebral bleeding was found.

Disseminated intravascular coagulation is a recognised but rare complication of severe pneumococcal infection.[5] It is unlikely, however, that mild disseminated intravascular coagulation of the type seen in two of our patients, or its prevention by heparin, could be important in determining the outcome of the infection.

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Clinical features and clinical course of patients with pneumococcal meningitis treated with heparin and of control patients

	No of cases	Mean age in years (range)	Patients with coma on admission	Patients with focal neurological signs on admission	Deaths	Days to full consciousness in survivors (SD)	Patients with new focal neurological signs developing during treatment
Heparin group	7	24 (13-40)	4	4	4	6·3 ± 5·0	4
Control group	8	28 (12-50)	3	2	2	6·0 ± 2·5	2