I have a vivid recollection of one of my teachers whose habit was as I was about to divide the second cranial nerve in an enucleation to say, "it was the right eye, wasn't it, Payne?" One knew very well that he would say this, but it never failed to send a shiver down the spine, and I have always felt compelled to verify the laterality by all possible means in the theatre immediately before operations of this sort.—I am, etc.,

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Staffing of Casualty Departments

SIR,—The recent crisis in the staffing of casualty departments throughout the country makes one look at the future—and at the past. The explanation given that the number of overseas graduates, who usually staff these departments, is drastically falling, produces a situation which is unlikely to improve. In the "good" (or "bad") old days there were no overseas graduates. Casualty departments in small hospitals were staffed by a competent sister, who would call on the house-surgeon when she was in trouble. Since the advent of the N.H.S., however, it seems to have become legally essential that every case attending hospital must first be seen by a "qualified" doctor. This doctor, for the sake of argument, may be totally inexperienced, come from some remote foreign medical school whose training is quite different from ours, have only spent a minimal time in Britain, and may only speak a smattering of English. This, however, is medicolegally more acceptable than that the patient be seen by a fully competent sister with many years of casualty experience in this country behind her—surely an absurd state of affairs?

We are constantly giving lip service to improvement of the status of the nursing profession. We change their names, we change their uniforms, we change their pay, but as soon as it comes to giving them clinical responsibility—surely the acid test of our opinion of their worth—we hold up our hands in horror.

A lamentable situation has arisen in which many casualty departments throughout the country are being closed at evenings and weekends. Surely the time has come when the Department of Health, the regional boards, and perhaps the judiciary, should reconsider as a matter of urgency the whole question of whether or not casualty departments may remain open even though there is no medical officer immediately available. Are we to deny the public a service which could so easily be given just because of the constant fear of possible litigation?—I am, etc.,

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Benzene Hexachloride Poisoning

SIR,—I am writing to record a case of nearly fatal poisoning in an 8-year-old boy resulting from ingestion of biscuits found on a rubbish tip. This had been sprayed on the previous night with Cooper's Tip Dressing, which contains 4% benzene hexachloride of which only 0.5% was of the gamma isomer.

The patient was brought home from school at 3 p.m. on 4 June vomiting and drowsy. He complained that he felt unwell, and after further vomiting fell into a restless torpor. At the time of admission to hospital at 9.30 p.m. he was in a stuporous state, but passing urine, vomiting, and protesting on examination. He had a suggestion of neck stiffness; pupils were dilated and sluggish in their reaction to light, and his fundi were thought to be normal. Tendon reflexes were reduced and difficult to elicit; plantar responses were flexor and abdominal reflexes absent. He had a bradycardia of 52 per minute with some sinus arrhythmia. The clinical diagnosis of presumed poisoning was made, and he was treated by aspiration of the stomach contents and put on half-hourly observation throughout the night.

By morning he had begun to twitch and his general state of consciousness had lapsed so that he no longer responded to painful stimuli and his pupils were quite fixed, dilated, and diverging. An intravenous infusion of 500 ml. of 10% mannitol was established to combat presumed cerebral oedema. We now learned that at noon on the day of admission he had eaten chocolate biscuits taken from a rubbish tip, which had been sprayed with Cooper's Tip Dressing Powder on the previous evening. He had taken about a dozen biscuits, and the wrappings proved to contain benzene hexachloride, which is present in 4% concentration in the tip dressing powder.

Investigations carried out included examination of his urine on admission for presence of sugar, salicylates, and phenothiazines which gave negative results: blood sugar 131 mg./100 ml.; pseudocholinesterase levels were at the lower range of normal, being 37 Rappaport units/ml. on admission and 41 Rappaport units/ml. 12 hours after admission. His serum electrolytes showed some salt depletion with a sodium level of 111 mEq/l., chloride 88 mEq/l., blood urea 20 mg./100 ml.

After the infusion of mannitol his level of consciousness improved so that he responded to painful stimuli. Grand mal convulsions supervened and he was given 5 ml. paraldehyde intramuscularly and intravenous dexamethasone. The boy's condition remained critical until 48 hours after admission, but by 72 hours he seemed to have made a full recovery.

Though benzene hexachloride was not identified in the vomitus, the circumstantial evidence for this being the cause of his illness is strong. Kay and Kuder¹ have described eight cases of poisoning following the ingestion of food containing 4% benzene hexachloride, three of whom died within seven hours of consuming the food, after having convulsions, coma, and pulmonary oedema. (Vomiting was thought to have prevented serious illness in others). Necropsy additionally showed cerebral congestion and oedema and renal ischaemia with haemorrhage.

Benzene hexachloride has nine stereoisomers, of which gamma-hexachlorocyclohexane is the least toxic to humans and the most effective insecticide.² Even with the gamma isomer fatalities have been reported by Attygalle and Fernando,³ where the undiluted insecticide was drunk in mistake for alcohol.

I feel that this case merits publicity to prevent recurrences elsewhere.—I am, etc.,

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- 2 Martindale, *Extra Pharmacopoeia*, ed. R. G. Todd, 25th edn., p. 525, 528. London, Pharmaceutical Press, 1967.
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Treatment of Typhoid

SIR,—The three recent papers and your leading article (8 August, pp. 318, 320, 323, and 297 respectively) on the treatment of enteric fever with trimethoprim-sulphamethoxazole prompt us to record a proved relapse of *Salmonella paratyphi B* infection following such treatment.

A man aged 29 developed fever on 26 February 1969, after a holiday in the Canary Islands. Five days later he was admitted to hospital and the suspected diagnosis of enteric fever was established by isolation of *S. paratyphi B* from blood cultures. Treatment with trimethoprim-sulphamethoxazole—two tablets six hourly—was started one week after the onset of his illness. His fever subsided over the next six days. Treatment was continued for a total of two weeks. No ill effects of this large dose were noted. Before discharge seven negative stools and six negative urine cultures were obtained.

Six weeks after the onset of the initial illness, and three weeks after the end of treatment, he was readmitted with a recurrence of fever of three days' duration. Blood and stool cultures again grew *S. paratyphi B*. The relapse was treated by chloramphenicol and his recovery was uneventful.

Two other patients with enteric fever, one caused by *S. typhi* and the other by *S. paratyphi B*, were treated similarly during the summer of 1969 and neither relapsed. Relapse is an important complication of enteric fever, and any treatment which could be shown significantly to reduce the incidence of relapse would be of great value. Relapse rates should therefore be recorded carefully in trials of the treatment of enteric fever, and we look forward with interest to Dr. S. A. Kamat's next report, which will include these important data for his large series. We have not come across another report of relapse following this treatment.—We are, etc.,

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Folate and Vitamin B₁₂ in Epilepsy

SIR,—I was interested to read your leading article on "Folate and Vitamin B₁₂ in Epilepsy" (27 June, p. 774). In subsequent correspondence Dr. N. S. Gordon (25 July, p. 226) has drawn attention to the association of reduced serum folate with the gradual mental deterioration observed in some children during the prolonged administration of anticonvulsant drugs.

The majority of studies have confirmed the threefold concentration of folate in the spinal fluid compared with serum, and this ratio has been shown to be maintained in treated epileptic patients in the presence of reduced serum folate concentrations. Whether these reduced levels of serum and C.S.F. folate reflect alterations in cerebral folate activity, as suggested by some reports describing the neurological and psychiatric complications of folate deficiency, is important but difficult to define in man.

Recent studies in rats,¹ gerbils,² and cats³ have indicated that brain tissue is capable of maintaining a normal level of folate activity, principally in the form of 5-methyltetrahydrofolate, in the presence of diphenylhydantoin.