Adrenaline in the treatment of anaphylaxis

SIR,—We would agree with Dr L M McEwen's observations (13 September, p 649) that prophylactic steroid cover is unlikely to be of any help in the treatment of anaphylaxis, but we cannot possibly agree with his statement that "when faced with profound anaphylactic collapse most doctors give adrenaline intravenously, using 0·5 ml of 1/1000 solution, diluted if possible, given slowly."

We consider the risks of intravenous adrenaline are so great that no general practitioner should ever give it in this way. Besides the very severe subjective symptoms that will arise, the more serious effects of cerebral haemorrhage and cardiac arrhythmias preclude its use intravenously except in exceptional circumstances, when it must be adequately diluted.[1]

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1 Goodman, L S, and Gilman, A, *The Pharmacological Basis of Therapeutics*, 4th edn, p 496. London, Macmillan, 1970.

Allergic reactions to tetracosactrin

SIR,—The Committee on Safety of Medicines has recently drawn attention[1] to the rare but serious reactions which have occurred in patients receiving tetracosactrin; in two cases the reaction was fatal. I wish to report three further patients who have developed allergic reactions to tetracosactrin injections.

A woman aged 25 had a five-year history of multiple sclerosis. She had had six previous courses of tetracosactrin for relapses of her condition. In June 1974 she had a further course; after the third injection she developed acute polyarthritis, an itchy rash, and wheezing. No further injections were given and the reaction settled with antihistamines.

A woman aged 27 had a four-year history of multiple sclerosis and had had three previous courses of tetracosactrin without any trouble. In February 1975 she gave herself a further course of tetracosactrin and after the seventh injection developed general malaise and severe local swelling and itching at the injection site.

In July 1975 a woman aged 68 developed a postvaccinial polyneuropathy and was treated with tetracosactrin. After six injections she developed an acute urticarial rash. The injections were stopped and the rash subsided.

None of these patients had a previous history of allergy, nor were they taking other medication. It is clear from the report of the Committee on Safety of Medicines that further injections could cause serious or even fatal reactions. This can be a problem in patients with multiple sclerosis, who may require repeated courses of corticotrophin. It is therefore noteworthy that the first two patients have received subsequent courses of corticotrophin in a base of carboxymethylcellulose (Crookes) without any reaction.

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1 Committee of Safety of Medicines, *Current Problems*, No 1, September 1975.

Infective agent in infantile enteritis

SIR,—The suggestion in your leading article (6 September, p 555) that most cases of infantile enteritis in Britain are of viral origin stems from a recent spate of publications reporting the presence of virus-like particles in the faeces of infants with enteritis. Most of the investigations described have included examination for the presence of enteropathogenic *Escherichia coli*, but it is likely that only a limited range of enteropathogenic serotypes could be recognised.

Epidemiological studies using serotyping techniques have implicated many *E coli* serotypes as causes of epidemics, but the set of antisera available to diagnostic laboratories has never included the complete range. For example, *E coli* O114 was first described as a cause of epidemic infantile gastroenteritis in Birmingham in 1956,[1] but antiserum for its identification did not come into routine use until further epidemics had occurred in the Manchester area in 1968-9.[2] Similarly, *E coli* O142 had been recognised as a cause of infantile diarrhoea in Indonesia in 1960[3] and in Mexico in 1965,[4] but antiserum was not widely available in Britain until 1971 following several outbreaks in Glasgow[5-7] and Dublin.[8] *E coli* O91 was described as the causative organism in an outbreak of infant diarrhoea in Winchester in 1968,[9] but antiserum for this O group has never been included in the routine range.

E coli isolated from patients in an outbreak may possess O antigens which are not included in the full international serotyping scheme. In one such outbreak strains isolated from all the affected babies were unidentifiable with antisera for *E coli* O groups 01-0157, but further studies showed that the O antigens of all the isolates were the same. A new O group, 0158, was established, but antiserum for its identification is not widely available.

Routine investigation of infantile enteritis frequently fails to find a pathogen. It is important to remain aware that the disease may be due to *E coli* serotypes recognised as enteropathogens but not included in the routine range of sera, to serotypes not yet recognised as enteropathogens, or even to new serotypes.

When outbreaks of infantile enteritis occur and the routine laboratory fails to find a bacterial pathogen it should not be assumed that the disease is of viral origin. Strains of *E coli* should be sent to the reference laboratory for complete examination, which will include full serotyping and in the future may include tests for enterotoxin production. Those investigating the role of viruses in enteritis should be aware of these principles.

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1 Rogers, K B, and Cracknell, V M, *Journal of Pathology and Bacteriology*, 1956, **72**, 27.
2 Jacobs, S I, *et al*, *Archives of Disease in Childhood*, 1970, **45**, 656.
3 Ørskov, F, *et al*, *Acta Pathologica et Microbiologica Scandinavica*, 1960, **48**, 48.
4 Olarte, J, and Ramos-Alvarez, M, *American Journal of Diseases of Children*, 1965, **109**, 436.
5 Rowe, B, and Gross, R J, *Lancet*, 1971, **1**, 650.
6 Love, W C, *et al*, *Lancet*, 1972, **2**, 355.
7 Kennedy, D H, *et al*, *Journal of Clinical Pathology*, 1973, **26**, 731.
8 Hone, R, *et al*, *Journal of Medical Microbiology*, 1973, **6**, 505.
9 Hughes, M H, Greaves, J L, and Bettelheim, K A, *Journal of Clinical Pathology*, 1968, **21**, 387.
10 Rowe, B, *et al*, *Journal of Clinical Pathology*, 1974, **27**, 832.

Hyperthyroidism after use of contrast medium

SIR,—We should like to report a further case of contrast-medium-induced hyperthyroidism. The patient was receiving treatment for his thyrotoxic state at the time Drs B J Fairhurst and N Naqvi reported their cases (13 September, p 630).

A 44-year-old man was diagnosed in January 1972 as having a small symptomless atrial septal defect. He was also suspected of having thyrotoxicosis as he exhibited exophthalmos and a tachycardia of 100/min. There was no goitre, however, and his serum protein-bound iodine (PBI) level was 520 nmol/l (6·6 μg/100 ml) (normal 276-630 nmol/l (3·5-8 μg/100 ml)). In April 1975 he was admitted to a psychiatric hospital for treatment of a schizophrenic illness and while there was found to have spastic quadriparesis with a right foot drop. He was referred for investigation to the neurological unit of this hospital, where he was again suspected of having a hyperthyroid state. Thyroid function studies were once again normal, however, his serum thyroxine (T4) level being 144 nmol/l (11·2 μg/100 ml) (normal 64-154 nmol/l (5-12 μg/100 ml)) and normalised thyroxine ratio (NTR) 1·04 (normal 0·88-1·1). Cervical myelography was performed on 22 May and a dose of 6 ml of iophendylate injection (Myodil) (2268 mg iodine) was given. No space-displacing lesion was found and he was returned to the psychiatric hospital.

When seen in the department of rheumatology and rehabilitation on 6 August for assessment and the prescription of a caliper he showed agitation, tremor, warm hands, and tachycardia of 110/min. A small goitre was palpable but no bruit was heard. A shrewd physiotherapist remarked that he had lost a lot of weight over the weeks she had been treating him. Further thyroid function studies were as follows: serum T4 171 nmol/l (13·2 μg/100 ml), NTR 1·12, confirming the clinical diagnosis of hyperthyroidism. No thyroglobulin antibodies as detected by the tanned red cell and thyroid antigen complement fixation tests were found. Treatment with carbimazole 10 mg thrice daily was begun. Although control of sympathetic overactivity with regression of the thyroid enlargement was obtained within four weeks there has so far been no weight gain.

Our patient exhibited exophthalmos without laboratory confirmation of thyrotoxicosis for 3½ years. We believe overt thyrotoxicosis was precipitated by the iodine dose contained in iophendylate injection. It is known[1] that iodine is slowly released from this oily preparation over many years and that serum PBI levels as high as 126 nmol/l (16 μg/100 ml) occur as long as 15 years after myelography, while in the early weeks considerably higher levels of PBI occur. The cases reported by Drs Fairhurst and Naqvi and this further case suggest that iatrogenic thyrotoxicosis may be less rare than we normally suppose and illustrate the need for caution in the use of these media in patients with goitre or suspected hyperthyroidism.

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1 White, A G, *British Journal of Radiology*, 1972, **45**, 21.

SIR,—In a recent report Drs B J Fairhurst and N Naqvi (13 September, p 630) described two patients who were diagnosed as having hyperthyroidism shortly after the administration of sodium ipodate for cholecystography. The hyperthyroidism was