

benoxaprofen damage was reduced; this may be due to a subclinical photosensitivity reaction, as occurs with onycholysis, which may not be associated with obvious sun sensitivity. It is not yet clear whether the hypertrichosis is simply secondary to the photosensitivity, as is seen in porphyria<sup>4</sup> and when alopecia areata is treated with ultraviolet B or psoralen-ultraviolet A, or whether a different mechanism is entailed, as in hypertrichosis associated with minoxidil.<sup>5</sup>

We thank Dr M Tobin and Dr W E B Preston for permission to report their cases.

- <sup>1</sup> Mikulaschek WM. Longterm safety of benoxaprofen. *J Rheumatol* 1980; 7, suppl 6: 100-7.
- <sup>2</sup> Taylor AEM, Goff D, Hindson TC. Association between Stevens-Johnson syndrome and benoxaprofen. *Br Med J* 1981;282:1433.
- <sup>3</sup> Fenton DA, English JS. Toxic epidermal necrolysis, leucopenia and thrombocytopenic purpura—a further complication of benoxaprofen therapy *Clin Exp Dermatol* (in press).
- <sup>4</sup> Kaufman BM, Vickers HR, Rayne J, Ryan TJ. Congenital erythropoietic porphyria. *Br J Dermatol* 1967;79:210-20.
- <sup>5</sup> Burton JL, Marshall A. Hypertrichosis due to minoxidil. *Br J Dermatol* 1979;101:593-5.

(Accepted 20 January 1982)

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## Haemophilus influenzae type b resistant to both chloramphenicol and ampicillin in Britain

It is now standard practice to treat severe infections due to *Haemophilus influenzae* with either chloramphenicol alone or a combination of ampicillin and chloramphenicol. We describe a patient who twice became infected with a strain of *H influenzae* type b that was resistant to both ampicillin and chloramphenicol.

### Case report

A 9-year-old boy was diagnosed in 1979 as having dermatomyositis, which was treated with prednisolone with a satisfactory initial response. To reduce his dependency on steroids, salicylates, sodium etidronate, and penicillamine were introduced sequentially, starting in January 1981. In April a calcinotic lesion medial to the left knee became infected and discharged pus.

Routine swabs were taken and treatment started with flucloxacillin 50 mg/kg 24 hours and fusidic acid 20 mg/kg 24 hours. *Staphylococcus aureus* sensitive to erythromycin, methicillin, gentamicin, co-trimoxazole, and cefuroxime but resistant to fusidic acid and penicillin, and *H influenzae* sensitive to erythromycin, gentamicin, and cefuroxime but resistant to ampicillin and chloramphenicol were isolated. Flucloxacillin was therefore stopped and intravenous erythromycin started. This caused painful phlebitis, and cefuroxime 56 mg/kg/day was substituted two days later. Intravenous antibiotics were given for a total of nine days. Flucloxacillin 40 mg/kg/day and cephalexin 40 mg/kg/day were given by mouth for a further 12 days. The treatment of his underlying dermatomyositis was then changed to daily high-dose prednisolone and weekly methotrexate because of progressive muscular weakness.

Six weeks after stopping antibiotics he had a general anaesthetic for insertion of grommets and simultaneous plastic surgery to repair a persistent skin defect over the left knee. One week later he developed pneumonia with production of purulent sputum. Chest radiography showed no acute changes. Sputum samples grew *H influenzae*, again resistant to ampicillin and chloramphenicol but sensitive to gentamicin. This was successfully treated with intravenous gentamicin and flucloxacillin. His general condition continued to deteriorate, however, and despite intensive treatment including plasmapheresis he died one month later.

**Bacteriology**—On both occasions the organism was initially identified as *H influenzae* by its morphological appearance when cultured on blood and chocolate agar. This was confirmed by its nutritional requirements of haeme and diphosphopyridine nucleotide (X and V factors). The isolate from the sputum was cultured on chocolate agar and then slide agglutination testing performed (Wellcome Foundation Ltd) which showed it to be serotype b. Beta-lactamase activity was shown by the method of McGhie *et al.*<sup>1</sup> The minimal inhibitory concentration was tested by the agar dilution method of susceptibility testing, which is not critically dependent on the inoculum

(Adatah, Mast Laboratories Ltd). The minimum inhibitory concentration of chloramphenicol was 16 mg/l (control strain <2.0 mg/l) and that of ampicillin 32 mg/l (control strain <1.0 mg/l). All the other sensitivities were determined by routine antibiotic disc sensitivity testing (Mast Laboratories Ltd). The disc strength of cefuroxime was 30 µg.

### Comment

Chloramphenicol-resistant, ampicillin-sensitive *H influenzae* was first reported from the United States in 1972.<sup>2</sup> In 1977 a child in Oxfordshire survived meningitis caused by *H influenzae* with the same antibiotic sensitivities.<sup>3</sup> In 1979, however, *H influenzae* type b resistant to both ampicillin and chloramphenicol caused an outbreak of meningitis in Bangkok in which three children died.<sup>4</sup> In the same outbreak one child with *H influenzae* type b meningitis was successfully treated with rifampicin and co-trimoxazole. The organism in our patient was sensitive to co-trimoxazole, and also to cefuroxime, which should be regarded as a therapeutic alternative when treating meningitis due to a similarly resistant organism as it penetrates adequately into the cerebrospinal fluid.<sup>5</sup> If this strain of *H influenzae* becomes more common the recommended antibiotic treatment for conditions such as epiglottitis and meningitis will have to be altered.

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(Accepted 20 January 1982)

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## Hypersensitivity to local anaesthetics: a direct challenge test with lignocaine for definitive diagnosis

Local anaesthetics hold a key position in medical and dental practice. When hypersensitivity (allergy) to them is suspected an accurate diagnosis must be established or a local anaesthetic found which the patient can take safely. Many tests have been advocated for this, but unfortunately, in-vitro tests have proved unreliable, and other procedures, such as nasal challenge and skin tests, have not been validated by controlled series. Indeed, in our own studies on skin testing with lignocaine a false-positive rate of one in four occurred among atopic subjects (unpublished observation).

We present here the details and results of a direct challenge test with lignocaine on eight patients with histories of hypersensitivity reactions attributable to this drug. Similar regimens have been reported<sup>1,2</sup> but mainly to evaluate a suitable alternative local anaesthetic to that suspected as an allergen. In our regimen a solution of lignocaine was administered in saline without preservative or vasoconstrictor, thereby avoiding any possible contribution from these substances.

### Patients, methods, and results

Seven women and one man (aged 18-56 years) were investigated after one or more suspected reactions to lignocaine with at least one of the following features: swelling of the lips and cheek, urticaria, wheeze, severe dyspnoea,