was also inoculated intracerebrally into a strain of mice known to be susceptible to acanthamoebae, but amoebae were not isolated.

Dr Griffin also suggests that repeated nasopharyngeal specimens should have been examined for acanthamoebae. In fact this also was done. Nose and throat swabs taken two days after admission and two and four days later were inoculated into HeLa cell cultures, on to klebsiella-agar plates, and intracerebrally into mice, again with negative results.

It is possible, as Dr Griffin suggests, that these cultures of CSF and nasopharyngeal secretions were negative because the sulphadiazine received by the patient for two days before the specimens were taken had inactivated the amoebae. However, the amoebae were seen to be still moving, although very sluggishly, in a specimen of CSF taken four days after the first, after the patient had received both sulphadiazine and amphotericin B.

The high cell count in the CSF is more typical of naegleria than of acanthamoeba infection. Dr Griffin suggests that *N gruberi*, which is sensitive to febrile temperatures, would cause little damage in a patient with a fever. In fact our patient had a fever of 39·1°C on admission, but thereafter his temperature never rose above 37°C.

There is thus no evidence for Dr Griffin's theory that the *N gruberi* was not the primary pathogen in this case.

D C WARHURST
A P C H ROOME
S K R CLARKE

Public Health Laboratory,
Bristol

[1] Apley, J, *et al*, *British Medical Journal*, 1970, **1**, 596.
[2] Saygi, G, Warhurst, D C, and Roome, A P C H, *Proceedings of the Royal Society of Medicine*, 1973, **66**, 277.
[3] Schuster, F L, and Rechthand, E, *Antimicrobial Agents and Chemotherapy*, 1975, **8**, 591.

Squints

SIR,—Mr B Harcourt (20 March, p 703) rightly mentions in his conclusions that squints require early diagnosis and early investigation. Neglect of this, he points out, may not only lead to permanent disability but also fail to unmask serious disease.

One obstacle to this desirable goal is often the waiting list for outpatient appointments and another, partly for this reason, is the reluctance of the doctor to make a referral until he is sure the squint is "real," hence producing the very delay which is detrimental. In some areas, notably the London Borough of Barnet, this difficulty has been overcome by a very successful arrangement whereby the orthoptist in the locally dispersed children's eye clinics sees any child from any doctor in the area who is uncertain and then refers those who require it to the ophthalmologist. This system has resulted in much earlier diagnosis and treatment of real squints and spared many a mother the wearisome and pointless business of having to attend hospital because her child has a broad nose. It also brings to light visual problems in siblings and the earlier diagnosis of conditions predisposing to squints such as high hypermetropia or anisometropia.

One other point which improves the defaulting rate at an early stage is the use of 0·1% hyoscine drops in place of several days of atropine. This is as powerful a cycloplegic and only requires one hour to work, obviating the necessity of a second visit and, in some cases, the destruction of a happy relationship between mother and baby.

P A GARDINER

Guy's Hospital,
London SE1

SIR,—As with the other papers in your series "Problems of childhood", Mr Brian Harcourt's article on squint (20 March, p 703) is commendable for its clarity, conciseness, and clinical relevance. He rightly stresses the importance of early diagnosis and treatment.

However, although glad to learn that squint surgery "rarely requires over three days in hospital," I feel that it is insufficient to state that it "is not particularly uncomfortable or unsettling even for very young children." Where preschool children have to be admitted to hospital, sadly it still seems necessary to emphasise the importance of their mothers being admitted with them. A recent paper by Douglas[1] reveals not only that hospital admission without the mother can result in disturbed behaviour in preschool children on return home but also that there is a significant increase in problems of behaviour and learning during the adolescence of these children. Other writers[2 3] have shown us how the concept of the child being "settled" in hospital can be misunderstood.

Where the mother cannot accompany the child Vaughan,[3] who reported on 40 children (mean age 5·9 years) in hospital for five days for correction of strabismus, described a deliberate policy of discussing their fears with the children and explaining the procedures, which mitigated some of the adverse psychological effects of the experience. Other methods have also been described.[4 5] Vaughan also reviewed[6] the psychiatric causes of squint, which were not referred to in Mr Harcourt's article.

ANTHONY G CARROLL

Department of Child and
Family Psychiatry,
Regional Hospital,
Galway, Ireland

[1] Douglas, J W B, *Developmental Medicine and Child Neurology*, 1975, **17**, 456. Vol. 17, No 4, August, 1975, p 456.
[2] Wolff, in *Children under Stress*, p 90. Harmondsworth, Pelican Books, 1973.
[3] Vaughan, G F, *Lancet*, 1957, **1**, 117.
[4] Becker, R D, *International Journal of Child Psychotherapy*, 1972, **1**, 65.
[5] MacCarthy, D, *Developmental Medicine and Child Neurology*, 1974, **16**, 279.
[6] Vaughan, G F, in *Little Club Clinics in Developmental Medicine No 9*, ed V H Smith. London, Heinemann, 1963.

Occupational hazard in preparation of polyacrylamide gels

SIR,—Polyacrylamide gel electrophoresis is used very widely for the analysis of proteins. The toxicity of the acrylamide monomer is well known.[1 2] We have, however, encountered recently a case of an acute allergic response to one of the reagents used in the preparation of polyacrylamide gels. The substance, *N,N,N′ N′*-tetramethylenediamine (TEMED), $(CH_3)_2NCH_2CH_2N(CH_3)_2$, is used to promote polymerisation of the gel.[3 4]

A research worker in this laboratory, in his late twenties and otherwise in good health, complained of insomnia which was associated with intense generalised pruritus. Skin testing showed that the agent responsible was TEMED. Five millilitres of TEMED was applied to the subject's forearm; within 30 seconds local vasoconstriction occurred and was followed by a suffused vasodilatation proximal to the site of application. Within 20-30 minutes the vasodilation had spread to the skin of the face and giant urticarial weals were apparent. Intense generalised pruritus and a pricking sensation of the conjunctivae lasted for approximately 12 hours. The application of reagent to the forearm of a control subject elicited no reaction.

The reaction could be adequately controlled with chlorpheniramine maleate. The allergic response occurred probably as the result of a type I hypersensitivity reaction (anaphylactic, reagin-dependent; classification of Gell *et al*[5]) mediated by the TEMED acting as a hapten and involving homocytotropic IgE antibodies. Satisfactory control was achieved by asking non-sensitive colleagues in the laboratory to handle the material in a fume hood.

The problem of toxic and allergic responses to materials found in research laboratories is an area in which recent legislation has defined the responsibilities of both employer and employee more clearly than in the past. Potential allergens commonly found in research laboratories include small-molecule sensitizers, of which TEMED is an example, and purified enzymes used for analytical purposes.

R A KLEIN

Medical Research Council Biochemical
Parasitology Unit,
Molteno Institute,
University of Cambridge

[1] Fullerton, P M, *Journal of Neurology, Neurosurgery and Psychiatry*, 1969, **32**, 186.
[2] Fullerton, P M, *Proceedings of the Royal Society of Medicine*, 1969, **62**, 201.
[3] Weber, K, and Osborn, M, *Journal of Biological Chemistry*, 1969, **244**, 4406.
[4] Gordon, A H, *Electrophoresis of Proteins in Polyacrylamide and Starch Gels*, p 43. Amsterdam, North Holland, 1969.
[5] Gell, P G H, Coombs, R R A, and Lachmann, P J, *Clinical Aspects of Immunology*, 3rd edn. Oxford, Blackwell Scientific, 1974.

Steroids and hypostatic eczema

SIR,—I want to thank Drs S Selwyn and P W M Copeman for their clarifying remarks on the treatment of patients with hypostatic eczema with Miol lotion and cream (14 February, p 399).

One explanation of the fact that I have seen adverse effects of treatment with strong steroids only in exceptional cases of this condition might, of course, be the choice of steroid. Approximately 90% of my patients were treated with betamethasone valerate or betamethasone dipropionate. In controlled studies we also used other steroids, and noted a temporary erythema of the skin and a burning sensation after, for example, fluclorolone acetonide or flumethasone.

Drs Selwyn and Copeman are concerned about the use of antibiotics in skin medicaments without bacteriological monitoring. I entirely agree that antibiotics are very seldom needed, either in leg ulcers or hypostatic eczema. Fairly extensive investigations in our clinic have shown not only that an antibiotic may change the growth pattern entirely but also that healing progresses independently of the bacterial flora. Skin conditions on the basis of venous incompetence of the leg should *not* be treated with antibiotics, either locally or systemically, unless there is a severe clinical