bacilli are not quite as rare as people might imagine.

With a relatively low level of immunization among those most at risk, the appearance of a highly virulent strain could again give rise to an outbreak, the first few cases of which might well be diagnosed at necropsy. Until we know the precise reasons for the decline of diphtheria in the United Kingdom we should be cautious of writing its obituary.—We are, etc.,

M. G. MCENTEGART
I. ZAMIRI

Department of Medical Microbiology,
University of Sheffield

- ¹ Brooks, G. F., *Journal of Infectious Diseases*, 1969, 120, 500.
- ² Logan, W. P. D., *Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service*, 1952, 11, 50.
- ³ Jellard, C. H., *Journal of Hygiene*, 1972, 70, 503.
- ⁴ Dunnet, W. N., Schallibaum, E. M., and Scott, T. G., *British Medical Journal*, 1960, 1, 251.

Dyslexia as Cause of Psychiatric Disorder

SIR,—With regard to the paper by Drs. W. A. Saunders and M. G. Barker (30 December, p. 759), although I agree that dyslexia could be a cause of psychiatric disorder, I disagree as to the differential diagnosis between dyslexia and illiteracy. The World Federation of Neurology definition of dyslexia includes more than was stated in the above paper. It reads "A disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence, and sociocultural opportunity. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin" (my italics).

In the cases quoted it is difficult to say that the patients were of normal intelligence since none achieved an I.Q. of more than 100, which puts them in the dull average and some in the educationally subnormal range. Similarly, cultural or scholastic opportunity is not mentioned, nor are modality deficits. It is not possible to decide, therefore, whether these patients were in fact dyslexic or were illiterate for other reasons, and I feel that the distinction should be more carefully drawn.—I am, etc.,

BEVÉ HORNSBY

Word Blind Clinic,
St. Bartholomew's Hospital,
London E.C.1

Treatment of Malaria

SIR,—Dr. Alan N. Smith (30 December, p. 793) draws attention to the possibility that a relapse of malaria may occur in association with pregnancy. One point in this letter, however, does require clarification. Dr. Smith is perfectly correct in recommending that patients who have been in an endemic area should continue taking an antimalarial for four weeks after entering a non-malarious country. The main purpose of this recommendation is to ensure that infection with *Plasmodium falciparum* which may have been contracted within the last few weeks, and is still in the incubation stage, does not become patent. A daily tablet of proguanil (100 mg) or a weekly dose of chloroquine (300 mg base) taken for four weeks after entering the non-malarious country will

ensure protection against this species of parasite or a radical cure of any developing parasitaemia. It will not, however, prevent relapses of malaria due to *P. vivax*, *P. ovale*, or *P. malariae*. Such relapses have their origin in the cryptic secondary exoerythrocytic stages in the liver. In order to ensure that relapses caused by these parasites do not occur (that is, a radical cure) it is necessary to give primaquine in the dosage referred to by Dr. Smith—namely, 15 mg base daily for 14 days. Secondary exoerythrocytic stages do not occur in *P. falciparum* so that a radical cure can be obtained by the sole administration of a blood schizonticide such as proguanil or chloroquine taken as prescribed. However, in the treatment of an overt malarial attack the drug of choice normally would be chloroquine, irrespective of the species concerned, since the action of proguanil is undesirably slow.

In relation to the dangers inherent in areas where drug-resistant parasites are present, may I draw the attention of your readers to an earlier article published in the *B.M.J.*¹—I am, etc.,

W. PETERS

Department of Parasitology,
Liverpool School of Tropical Medicine

- ¹ Peters, W., *British Medical Journal*, 1971, 2, 95.

Complication of Internal Jugular Venepuncture

SIR,—The hazard described by Dr. Sally Arnold and others in their excellent report (27 January, p. 211) would seem to be inherent in the technique they described for puncturing the internal jugular vein. The point of introduction of the needle is correct, but the needle should not be directed towards the suprasternal notch.

The method taught me 25 years ago by Dr. W. W. Payne when he was consultant in chemical pathology to the Hospital for Sick Children, Great Ormond Street, was as follows: The body of the child should lie at right angles to the edge of the supporting couch or table. The head should be a little over the edge and be turned, either to the right or left, so that the sternomastoid muscle is stretched and parallel with the floor. The syringe and needle are also held parallel with the floor and with the edge of the couch (that is, at right angles to the long axis of the child's body). This relationship of the needle and syringe should not be altered. In a child with an average, or more than average, length of neck the needle, when introduced at the point midway between the mastoid process and the suprasternal notch and deep to the posterior edge of the sternomastoid, will enter the internal jugular vein. If the child has a short neck, then the needle is inserted deep to the sternomastoid at a point caudal to that described above, but the relationship to the horizontal plane and the long axis of the child remains the same. In this manner it is impossible to puncture either the apical pleura or the trachea.

I estimate that I have done 1,000 venepunctures by this method in the past 25 years without mishap, apart from one baby who went into apnoea very briefly and who recovered without complications. The same technique has on one occasion proved valuable in an adult with a severe electrolyte

disturbance in whom no superficial veins could be found on which to perform venepuncture.—I am, etc.,

G. A. MATTHEWS

Hemel Hempstead General Hospital,
Herts

Possible Hazard of Contact Lens Manufacture

SIR,—Recently, when principal medical officer for occupational health in the London Borough of Camden, I saw three cases of urological disease and one case of granuloma of the upper lobe of the left lung among seven workers in a hospital department making polymethyl methacrylate contact lenses. The urological conditions were a carcinoma of bladder and two cases of cystitis of unknown aetiology. All the persons had worked in the department for more than 10 years, and the four who became ill had done so within the past two years.

It seems that very little dust or fumes are produced in making these contact lenses, and no cases of chest, kidney, or bladder disease have been reported in the past 10 years among the staff of a large British manufacturer. In the hospital department referred to above the polymethyl methacrylate is washed with detergent, dried, and moulded at a temperature of 142°C. The lens is finally shaped with a dental drill and polished with a mildly abrasive soap. The usual cleaning agent contains calcium carbonate and ammonia. Xylene and carbon tetrachloride are sometimes used for final cleaning.

Plexiglass, or Perspex, is formed by the polymerization of the monomer methyl methacrylate. The end product may contain 3-5% of low molecular polymers and some monomer. The monomer has an oral LD₅₀ of 8.4 ml/kg for rats and 7 ml/kg for rabbits. It is corrosive to mucosae and in the rabbit skin causes local erythema.¹ Workers exposed to fine polymethyl methacrylate dust complained of irritation of the respiratory tract,² and Harris³ thought the polymer powder also possessed allergic properties similar to the monomer. Machining of Plexiglass gives rise to sweetish vapours containing monomers if local cooling is not applied.⁴ Karpov^{5,6} observed irritability, tiredness, drowsiness, headache, anorexia, and hypotension in workers exposed to monomer vapours. Irritation of the mucosae occurred at 62 p.p.m. He suggested therefore a maximum permissible level of 12.5 p.p.m. Raine⁸ found this value too high, since at exposures to 12.5 p.p.m. for 20-90 minutes dizziness, drowsiness, sickness, and loss of consciousness were reported. It is therefore rather surprising to find that the threshold level value proposed by the American Conference of Governmental Industrial Hygienists is 100 p.p.m. Harris⁸ believes that sawing Plexiglass may set free unreacted methyl methacrylate which causes contact eczema.

From the above it is reasonable to assume that moulding of Plexiglass at high temperatures will cause an evolution of methyl methacrylate vapour. I can find no reports of a resulting incidence of cancer or kidney disease. The illnesses of the four patients I saw might have been purely coincidental, but I would be interested to hear of other cases.