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.,

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I. ZAMIRI

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- 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.,

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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

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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.

I am grateful to Dr. Wilfrid G. Harding, medical officer of health, London Borough of Camden, for his interest and encouragement.—I am, etc.,

R. ROUTLEDGE

Shenfield, Essex

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- 6 Karpov, B. D., *Gigiena i Sanitariya*, 1954, No. 10, 25.
- 7 Raines, L. A., *Gigiena Truda i Professional'nye Zabolevaniya*, 1957, 1, 56.
- 8 Harris, D. K., *British Journal of Industrial Medicine*, 1959, 16, 221.

Making Hospital Geriatrics Work

SIR,—It is interesting to read that one of the consequences of Drs. H. M. Hodkinson and P. M. Jeffery's approach to "Making Hospital Geriatrics Work" (2 December, p. 536) is the maintenance of the morale and professional satisfaction of "ancillary staff." May I, as a physiotherapist working in this particular arena, put a different point of view?

Paramedical staff involved in the geriatric service frequently experience feelings of impotence and frustration. This is one of the major causes for poor recruitment in this field of medicine and I do not believe that the situation can be remedied by increasing patient turnover.

Rehabilitation of the elderly patient is a lengthy and detailed procedure. Generally it is not so much the acute medical problems that concern the therapist but, to quote Drs. Hodkinson and Jefferys, "the unavoidable [case load] of patients with greater disability." If such patients are to return once again as viable members of their community and maintain optimum independence there, then much time needs to be given by all those involved in their reablement. In these situations, the therapist will need medical support over a long period rather than medical "pressure" to carry out such a programme effectively. Drs. Hodkinson and Jefferys may be able to concede "less than complete therapeutic results," but in rehabilitation of older patients there should be no such compromise. The process cannot be hurried. The rate of turnover may be slowed down in consequence, but the alternative for the patient is breakdown at home and eventual hospital readmission, and for the therapist the knowledge that one has been prevented from giving one's professional best.

After three months in hospital, in Drs. Hodkinson and Jefferys's view, discharge becomes very much harder because of "institutionalization" and "the withering of the patient's ties with the community." One might venture to suggest that perhaps it is those with the greatest disability who tend to have the longest stay in hospital. If a patient has adequate incentive and his reablement programme is both realistic and appropriate, it has not been my experience that "institutionalization" occurs, however prolonged his hospital stay may be. Moreover—and this is essential—if relatives, friends, and supporting social service personnel are involved from the beginning,

need there be this inevitable "withering" of community ties? Rehabilitation after all, is an exercise in vivo and not in vitro.—I am, etc.,

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Edgware, Middlesex

Sickle-cell Anaemia

SIR,—Sickle-cell anaemia was unknown to medicine until 1905, when James B. Herrick of Chicago, an experienced haematologist in a country with a large negro population, seems to have got the shock of his life on seeing the sickling phenomenon in the blood film of a negro with severe anaemia.¹ I have been informed by a haematologist that of course sickle-cell anaemia was well known before 1905. However, at the beginning of the century there were numerous books on haematology with coloured illustrations of the bloods of all known anaemias, and at least in British, American, French, and German books there is no mention of sickle-cell anaemia or thalassaemia, with its almost equally arresting blood picture. These two words seem to have been unknown to the official nomenclatures of those countries. Can anyone refer me to any evidence in black and white in any language that these two diseases were known to exist before 1905?—I am, etc.,

H. GALL

Richmond upon Thames,
Surrey

¹ Herrick, J. B., *Archives of Internal Medicine*, 1910, 6, 517.

Nitrazepam: Another Interesting Syndrome

SIR,—A 55-year-old woman for many years accustomed to taking five 100 mg tablets of butobarbitone during the night was admitted to hospital for treatment for her alcoholism. She was immediately changed to nitrazepam 10 mg at bedtime, something I had been unable to achieve in her home environment. Six weeks later for the first time, and after her discharge home, she complained of pins and needles in both hands and that the fingers were clumsy and losing their power. Her symptoms were worse in the morning and gradually improved as the day progressed. On examination there was no evidence of peripheral neuritis, and no other obvious cause was found. The severity of her symptoms increased, and when she was woken during the night by the tingling in her hands she resorted to taking another nitrazepam tablet to obtain sleep.

Her hands became useless in the mornings; she had to stop knitting and could not hold a needle to sew on a button. By late evening her hands were almost back to normal. One night she omitted her nitrazepam because she had to see her son off at 5 a.m. She noticed for the first time in weeks that her hands felt normal. She took her nitrazepam at 5 a.m., and within two hours she woke with her hands tingling and feeling numb once more. Nitrazepam was immediately stopped and she has had no recurrence of the symptoms since.

I am drawing attention to this case because when my patient was wakened by her symptoms she took another dose of nitrazepam,

thereby inadvertently aggravating them. I did not elicit the history of improvement as the day wore on until afterwards. It would appear that when a patient complains of acroparaesthesiae for which no other cause can be found an inquiry into the taking of nitrazepam should be made.—I am, etc.,

HAMISH MACLEAN

Gairloch, Rossshire

Internal Fixation of Secondary Deposits in Long Bones

SIR,—We would like to question the finding of Campbell,¹ quoted by Mr. Malcolm Fidler (10 February, p. 341), that malignant tissue is not disseminated along the shaft of a long bone by the insertion of a Küntscher nail.

It is our clinical experience to have discovered, in some cases, further neoplastic deposits at the opposite end of the bone to the point of entry of the internal splint, some months after the operation and local radiotherapy. Because of this it has been our policy for some years now to irradiate (preferably with megavoltage) the whole length of the shaft postoperatively.—We are, etc.,

W. H. BOND

A. J. BANKS

WILLIAM G. JONES

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Birmingham

¹ Campbell, C. J., in *Palliative Care of the Cancer Patient*, ed. R. C. Hickey, p. 313. Boston, Little, Brown and Co., 1967.

Anthrax

SIR,—Dr. Robert Lamb's article on anthrax (20 January, p. 157) was most valuable for doctors working in the U.K. but did not cover the full spectrum of the disease as seen elsewhere.

During my service a good many years ago as a pathologist in the Gold Coast (now Ghana) it was not very uncommon to encounter anthrax in the necropsy room, although a malignant pustule was never seen. The story was usually that the body had been found under a tree, supplemented occasionally by evidence that the deceased was one of many people who had eaten the meat of a cow which had been killed when moribund. The ratio of reported fatalities to incidents was usually unity, and it seemed likely that the common mode of infection was by ingestion and that the infectivity of the organism by this route was very low.

Soon after my arrival at the Accra laboratory I was taught, by Dr. George Robinson, that it was a good plan to remove the skull-cap before opening the trunk when doing a necropsy. A bulging purple dura with haemorrhagic meningitis was suggestive of anthrax, which could be confirmed by puncturing the membranes and withdrawing a drop of cerebrospinal fluid for examination; in this way further opening of an infected body could be avoided. My curiosity once led me to do a full necropsy on one of these patients, in whom large masses of mesenteric lymph nodes, which looked like ripe Victoria plums, convinced me that the gut had indeed been the portal of entry of the infection.

On one occasion only was I able to diag-