thromboembolism. Four of these patients had normal pulmonary "wedged" pressures (indirect left atrial pressures) and in one there was a slight increase. In the remaining six the pulmonary "wedged" and pressure record could not be analysed because of difficulty in identifying an appropriate waveform. This could have been due to technical imperfection, but an alternative explanation might be pulmonary venous hypertension without left atrial hypertension. The occurrence of pulmonary venous constriction following experimental pulmonary embolism was demonstrated by Daicoff et al. 1 This mechanism could account for pulmonary oedema in the absence of left heart failure and might also account for the difficulty in obtaining an adequate pulmonary "wedged" record.

Although in man the mechanism is obscure, there is no doubt that pulmonary oedema can occur as a result of pulmonary thromboembolism. We are, etc.,

W. J. WINDENBAK
F. MORAN
Centre for Respiratory Investigation, Royal Infirmary, Glasgow

2 Smith, G. T., Dester, L. and Dammin, G. J., in Pulmonary Embolic Disease, ed. A. A. Sembera and M. Stein, p. 120. New York, Grune and Stratton, 1965.
4 Dester, L., Bulletin of the New York Academy of Medicine, 1960, 36, 92.

Pathology of Malignant Hyperpyrexia

Sir,—There are two minor inaccuracies which should be corrected in your otherwise excellent leading article (3 February, p. 249).

Firstly, malignant hyperpyrexia was first recognized as a complication of general anaesthesia in the 1930s. 2 At that time ether was probably the agent most frequently responsible. Secondly, Bradley and Murchison 3 studied muscle biopsy specimens from six patients at risk in two families, not four as stated. At a recent meeting of the Royal Society of Medicine, the pathologist and biochemist of the post-mortem and in vivo biochemical studies commented on malignant hyperpyrexia were discussed in greater detail.—I am, etc.,

W. G. BRADLEY
Newcastle General Hospital,
Newcastle upon Tyne

3 Proceedings of the Royal Society of Medicine, 1973, 66, 63.

Thiocyanate Metabolism in Human Vitamin B12 Deficiency

Sir,—The letter from Dr. A. G. Free man (27 January, p. 231) has reminded me of an article entitled "Optic Atrophy and Pernicious Anaemia; with Special Reference to Sex Distribution and Aetiology," published in 1915, in the "medical" literature. Two cases were described and the sex, age of onset, and smoking habits in 21 others reported in the literature were analysed. Of the total, 22 patients were males; in the only female the diagnosis was in doubt. A positive history of smoking was obtained from 11 males; in the remaining cases no details were given. In the paper attention was drawn to the fact that this rare complication of pernicious anaemia predominantly affects males, and the aetiology of this chronic form of retrobulbar neuritis was discussed with special reference to the role of tobacco.

I have published in an obscure and short-lived journal (under my editorship) provides ample reason why this article has passed unnoticed.—I am, etc.,

DOUGLAS MCLAIN
Marnhull, Dorset

1 Mclain, D., and Goldsmith, A. J. B., "Obiter of the Middlesex Hospital, 1951, 1, 109.

Medical Aspects of Ambulance Design

Sir,—I was very interested in the article on ambulance design by Dr. Roger Snook (2 September, p. 574). Recent experiences have underlined what he says about the motion of the vehicle affecting the condition of some patients and the inadequacies of some equipment. Several of patients requiring life-support treatment. It would seem that giving a shocked patient a ride in a modern ambulance may well finish him off. How often do we read in the paper "The patient died on the way to hospital!" I know there are many problems attached, and money is not the least of them, but in this age of science could we not design an ambulance?—I am, etc.,

R. N. SEYMOUR
Street, Somerset

Staffing of Accident and Emergency Departments

Sir,—I was suggested by the suggestion of Mr. J. C. Scott (3 February, p. 292) that casualty work should be an essential prerequisite for entry into general practice. Had such service not been a requirement for the F.R.C.S., then the staffing crisis of casualty departments would have occurred many years ago. I differ from Mr. Scott in that the conditions of work of casualty officers as have taken place recently would have been inevitable 20 years ago. Accident departments should be staffed by doctors keen to work there, not by men compelled to do six months' service in inadequate departments with inadequate training and cover. —I am, etc.,

R. K. HOLDSWORTH
Rugby

Sir,—My friend Mr. P. A. M. Weston (13 January, p. 114) must have been living in Thameside if he believes that the typical family doctor's work consists in caring for the patient as a whole and in continuity. The stresses put upon the general practitioner by the extended group of the "N.H.S. have tended to have quite the opposite effect. Many doctors will reluctantly agree that they have time only to treat the presenting symptom. With regard to continuity, witness the late hours, the patient must be content with the service of any doctor. Witness the town doctor system where on a Wednesday afternoon, no doctor is on call for the community. And witness the lock-up surgeries where the agency doctor has no access to the patient's records.

Mr. Weston states that G.P.'s are suitable to staff small hospital accident units. This is a move in the right direction but, as I argued in my letter (9 December, p. 607), I believe that the only solution to the staffing problem is the universal use of doctors of this genre.

Mr. Weston's main point is that staff (and I presume he means casualty consultants) should be asked to do the accident departments to train the G.P.'s. I believe that all departments need doctors with the G.P.-type of training who will be sensible enough to know what up to was played by the consultant staff to deal with the seriously ill or injured. No special training is necessary beyond that learned as an undergraduate and as a house surgeon, except perhaps an acute psycholgy could have played a major role.

I am, etc.,

J. P. TUNNEY
Whitewaven, Cumberland

Disappearance of Diphtheria

Sir,—We appreciated your leading article on "Infectious Disease" (13 January, p. 63) and welcome the series it introduces. In the light of our experience in a laboratory with a special interest in diphtheria we feel that the information that diphtheria strains may merit some further comment. It is a common assumption that the disappearance of diphtheria was due to immunization, but we do not know why diphtheria declined so rapidly, nor do we know what upward shift of the susceptible groups in the prevalent types of strain.

The isolation of atypical and non-toxigenic strains first became fairly common about 1935, before either extensive immunization or large group practice that had played an important rôle. Two changes are noteworthy. There has been a steady fall in the incidence of virulent strains of each type isolated 1 and a change in the incidence of the biotypes, so that today endemic mutants strains, the majority of which are non-toxigenic, are commonly found, but epidemic group strains are uncommon and interemedius is rarely seen.

There is a familiar ring about the prediction that with continuing immunization diphtheria must cease to exist. The hope expressed by Logan in 1952 that diphtheria could disappear within 10 years was not realized. If we examine the situation in the United Kingdom, we find that the most vulnerable group, the underprivileged, have the poorest acceptance rate of immunization. Experience in areas such as Alberta, Canada, draws attention to the necessity for diphtheria bacilli in the skin of a section of the population, with occasional cases of fatal or classical diphtheria in their associates. Moreover, some of the patients with typical classical diphtheria have been fully immunized. Although the declining world incidence of classical diphtheria is a hopeful sign, we must remember that as the incidence of the disease has fallen, the mortality rate has risen steadily from 5% in the 1930s to 20% in 1970 in the United Kingdom. Since many laboratories became aware of the importance of looking for the organism, it appears that the diphtheria
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 of the first kind 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

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 reading 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.,

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

Treatment of Malaria

Sir,—Dr. Alan N. Smith (30 December, p. 799) 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 erythrocytic 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 erythrocytic 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.?—I am, etc.,

W. Peters
Department of Parasitology
Leped School of Medical Science

Complication of Internal Jugular Venepuncture

Sir,—The hazard described by Dr. Sally Arnold and her co-workers in their excellent report (21 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 subclavian 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 support couch or table. The head should be a little over the edge and be turned, either to the right or to the 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 floor or 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 was left behind 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 producing a liquid 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, cases of chemical pneumonitis and chemical-induced 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 polymethylacrylate is washed with methanol, 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 and producers of polymethyl methacrylate may contain 3-5%, of low molecular polymers and some monomer. The monomer has an oral LD50 of 84 ml/kg for rats and 7 ml/kg for rabbits. It is corrosive to mucous membranes and the rabbit skin causes local erythema. Workers exposed to finely powdered methacrylate dust complained of irritation of the respiratory tract,3 and Harris thought the polymer powder also possessed allergic properties similar to the monomer. Monitoring of Plexiglass gives rise to sweat vapours containing monomers if local cooling is not applied.4 Karpov5 observed irritability, tiredness, drowsiness, dryness of the angora, and headache in workers exposed to monomer vapours. Irritation of the mucous occurred at 62 p.p.m. He suggested therefore a maximum permissible level of 12.5 p.p.m. Rainey found this value too high since at exposures to 12.5-5 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 varied between 50 and 100 p.p.m. at the American Conference of Governmental Industrial Hygienists is 100 p.p.m. Harris believes that sawing Plexiglass may set free unreacted methyl methacrylate which causes conjunctival irritation.

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.