General Hospital) of left facial palsy in tetanus. In almost all these cases the lesion was incomplete or partial on the same side of the injury to the face and recovery eventually occurred.

Electrophysiological studies of facial palsy in tetanus by Bagratuni⁸ gave normal results. Park⁶ noted normal nerve conduction time of the involved facial nerve and abnormal complex activity of the involved muscles on electromyography and suggested a nuclear lesion of the facial nerve. In Farquharson's case⁷ electromyography showed positive waves but no fibrillations. Recently Garcia-Mullin *et al.*,⁹ reported the results of electrophysiological studies of a case of left facial palsy with tetanus which resulted from an injury to the left side of the face. There was prolonged distal latency of the left orbicularis oculi after stimulation of the left facial nerve which became normal after clinical improvement. Electromyography of the orbicularis oris and orbicularis oculi showed abnormal muscle potentials at rest which disappeared after lignocaine block of the facial nerve (suggesting that the origin of the muscular activity was proximal to the block) and during sleep (indicating a central or nuclear lesion of the facial nerve). The prolonged distal latency of the orbicularis oculi could have been due either to a peripheral nerve lesion or to dysfunction of the larger motor neurones in the facial nucleus. There was no evidence of a neuromuscular transmission defect. In Kaeser and Saner's case³ neuromuscular block due to curare cannot be excluded. However, if neuromuscular block does occur in some cases of tetanus, theoretically guanidine hydrochloride may help the presynaptic release of acetylcholine and thereby improve muscle paralysis.

Baker¹⁰ described degenerative changes in cranial nerve nuclei in tetanus. I therefore consider a temporary and partial lesion of the facial nerve nucleus involving motor neurones in groups due to centripetal transport of tetanus toxin to be the most likely explanation for partial or segmental facial paralysis on the side of injury.—I am, etc.,

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- 1 Boudouresques J., Khall R., Vigouroux R. A., Daniel F., and Gosset A., in *Handbook of Clinical Neurology* ed. P. J., Vinken, and G. W., Bruyn, Ch. 13, Vol. 7. Amsterdam, North Holland publ. Co.
- 2 Kaeser, H. E., Muller, H. R., and Friederich B., *European Neurology*, 1968, 1, 17.
- 3 Kaeser, H. E., and Saner A., *European Neurology*, 1970, 3, 193.
- 4 Watkins, A. L., *Archives of Neurology and Psychiatry* 1939, 41, 788.
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- 7 Farquharson, R. G., *Proceedings of the Royal Society of Medicine* 1972, 65, 875.
- 8 Bagratuni, L., *British Medical Journal* 1952, 1, 461.
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Who'd be a Physiotherapist?

SIR,—While feeling duly grateful for what we are about to receive in the shape of a rise in doctors' pay, I wonder if we might not spare more than a passing thought for one group of hospital ancillary workers

whose salary scale has long been niggardly and is now falling comparatively farther and farther behind—the physiotherapists.

A would-be physiotherapist has to have obtained not only a minimum of five G.C.E. "O" levels but one or preferably two "A" levels, which is not so far removed from qualification for university entry, before being acceptable for training. Unless grant-aided, she then has to pay for her training for three years—during 2½ years of which she can be treating patients under supervision—and pay also for accommodation, uniform, books, etc., studying numerous scientific and technical subjects, including anatomy to near medical standard, to make her a specialist in her own treatment field, before finally paying also examination fees when she qualifies as M.C.S.P.

Yet she will then find herself paid less than a staff nurse (though nurses require no "A" levels, fewer "O" levels, and are paid a living wage during free training); less than the younger clerk who perhaps books in the patients for the physiotherapy department; less than the overtime-working domestic of sometimes minimal schooling who cleans out the department (while most physiotherapists get paid nothing for their overtime on emergencies or at weekends); less even than the traffic warden who may book her for incautious parking of the Mini, which is as much as she is likely to be able to afford, if she has snatched a few minutes out for shopping.

No doubt the Department of Health and Social Security is reluctant to pay more when they have for years been able to get this particular sweated-labour service so cheaply. Physiotherapists do not have a powerful, militant union to fight for them and hitherto they have voted almost unanimously against strike action, even when other, already better paid groups, have taken such action. But it would be shameful if doctors, either through ignorance or I'm-all-right-Jackmanship, failed to support one of the most dedicated of all ancillary groups of hospital workers.—I am, etc.,

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Treatment of Acute Intermittent Porphyria

SIR,—With reference to the article by Dr. A. D. Beattie and others (4 August, p. 257) it may be of interest to report the case of a patient with acute intermittent porphyria whom I began to treat with guanethidine in 1964.¹ He recovered completely symptomatically, and, whereas he was considered totally unfit for work in 1963, he became able to take up his studies to become a merchant captain and is now working as an officer of a merchant ship. His urinary excretion of porphobilinogen and δ -amino-laevulinic acid remains high.

On one occasion when the patient had attacks of moderate abdominal pain an attempt was made to alleviate this symptom with repeated intravenous injections of 2 mg of the beta-blocking agent oxprenolol. This caused no change in the patient's condition, but when the dose of guanethidine was temporarily increased the abdominal pain eventually disappeared. He now takes 75 or 100 mg of guanethidine daily and is completely free from symptoms, apart from

a tendency to frequent bowel movements in contrast to the severe constipation that he suffered from before the guanethidine treatment was begun. He has no orthostatic hypotension.

The treatment has now been continued for nine years without reappearance of the symptoms of the disease. It is understood, of course, that the treatment is purely symptomatic.—I am, etc.

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¹ Wahlberg, P. I., *Lancet*, 1964, 2, 817.

Type IV Hyperlipidaemia in Cord Blood

SIR,—Hyperlipidaemia is now clearly recognized as a risk factor in coronary heart disease¹ and it can be found in young, clinically healthy individuals—namely, children of patients with premature atherosclerosis.² You have recently emphasized the importance of serum cholesterol determinations in young people in a leading article (24 March, p. 690), and it clear that the early detection of hyperlipidaemia, eventually from birth, may become a useful step in the primary prevention of juvenile atherosclerosis.

For this reason we have started a programme of study of the serum lipid pattern of the newborn. Blood samples are collected immediately after birth from the umbilical cord. Serum total cholesterol levels were determined by the method of Watson,³ serum triglyceride by the method of Eggstein and Kreutz,⁴ and lipoprotein electrophoresis was carried out in cellulose acetate.⁵

Among the 78 cases observed up to now we have found six cases of type IV hyperlipidaemia (see table). Glueck *et al.*⁶ have reported the detection of type II hyperlipidaemia in cord blood, but as far as we know these are the first cases of type IV hyperlipidaemia in the newborn to be reported here.

Case	Serum Total Cholesterol (mg/100 ml)	Serum Triglycerides (mg/100 ml)	Lipoprotein Electrophoretic Pattern
1	91	157	IV
2	96	171	IV
3	90	186	IV
4	102	171	IV
5	96	186	IV
6	100	171	IV

Our finding of a high prevalence in the newborn of this hyperlipidaemia, which is usually regarded as characteristic of the adult, may well be correlated with the great prevalence of type IV hyperlipidaemia observed in our first screening of the Portuguese population.⁷ Determination of the future serum lipid pattern of these babies, study of their clinical evolution, and further investigation of their families are needed to evaluate the meaning of these results.—We are, etc.,

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- ¹ Kannel, W. B., Castelli, W. P., Gordon, T., and McNamara, P. M., *Annals of Internal Medicine*, 1971, 74, 1.
- ² Martins, F. M., et al., in *Proceedings of the 6th European Congress of Cardiology*, Madrid, 1972.
- ³ Watson, D., *Clinica Chimica Acta*, 1960, 5, 637.
- ⁴ Eggstein, M., and Kreutz, F. H., *Klinische Wochenschrift*, 1966, 44, 262.
- ⁵ Canal, J., and Ricquois, M., *Feuillets de Biologie*, 1971, 12, 53.
- ⁶ Glueck, C. J., Heckman, F., Schoenfeld, M., Steiner, P., and Pearce, W., *Metabolism*, 1971, 20, 597.
- ⁷ Miguel, M. J. P., et al., *Annales de Biologie Clinique*, 1973, 31, 147.

Psychiatric Nurse as Therapist

SIR,—Dr. I. M. Marks and his colleagues are to be congratulated on their well-designed paper (21 July, p. 156) which raises a number of topical issues, especially for those engaged on similar ventures.

At this unit we have, since 1961, operated an 18-month postgraduate course for nurses who hold as their basic qualification at least the S.R.N. or R.M.N. The regional hospital board allocates four bursaries (equivalent to a staff nurse's salary) and, in conjunction with the Portsmouth Polytechnic, a special diploma is awarded to candidates after successful completion of the training. Despite obvious differences in clinical approach and setting, our aims are not unlike those outlined by Dr. Marks—for example, the use of behaviour modification through the repeated coupling of two individuals¹ or small "transactional" groups for mothers and pre-school children, involving social comparison processes, especially modelling.² Our curriculum seeks to impart a substantial body of theoretical knowledge, authenticated by continuous practical involvement and the sharing of learning experience with other team members by means of study groups, tutorials, and seminars. There is increasing emphasis on extramural work, follow-ups, and crisis intervention.

Because much of our enterprise deals with multidimensional family systems rather than individuals, it is harder to apply "accurate measurements" to a comparison of therapeutic effectiveness. Frequently a nurse therapist replaces absent or "invalid" parents; or he or she might have to feed back specific activity cues into the home of an autistic child. Nonetheless, we would agree with all the general comments made about the value and functional aspects in the training of nurse therapists. A recent cost analysis points to a possible reduction in drug expenditure; and a prospective study of "breakdown recurrence," now under way, suggests no significant difference between patients treated by nurses and other therapists. The problem of grading on the Salmon scale seems to us of paramount importance. If so much effort is being devoted to training and skilled nurse therapists become ultimately sidetracked into administration, one might perhaps question if this constitutes "the most rational deployment of N.H.S. personnel."—I am, etc.,

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- ¹ Haffner, C., *Deutsche Schwesternzeitung*, 1968, 10, 601.
- ² Mundy, L., *Journal of Child Psychotherapy*, 1972, 3, 77.

SIR,—I found the paper by Dr. I. M. Marks and others (21 July, p. 156) very interesting.

I am aware of a somewhat similar attempt in Canada which was not too successful. However, in this instance attempts were made to utilize the Canadian equivalent of state enrolled assistant nurses, probably because they were the only staff readily available at the time. I note, too, that Dr. Marks and his colleagues were able to select their five trainees out of 80 applicants.

Secondly, the authors say that "qualified therapists need to be linked either to a psychiatrist or to a psychologist." I wonder how they would perform if they found themselves in a post where the psychiatrist or psychologist was either not as enthusiastic as the authors for utilizing nurses in this way or, possibly more important, was not as well informed in behaviour therapy principles and practice as the authors (or possibly even the nurse-therapist)? I am now a little out of touch with the situation in Britain, but certainly in Canada there are still many areas where knowledge of these is somewhat limited. I am not entirely clear whether the authors feel these nurse-therapists could function adequately in such a situation. In other words, could they function where the psychiatrist acted purely as a referrer of patients, or do they require that either he or the psychologist act in a supervisory capacity?

In view of recent evidence suggestive of the advantages in some conditions of in vivo (or in practice) exposure over the use of fantasy (or imagination), the use of some such therapists becomes even more important, particularly on account of the time factor involved. I think, therefore, that it would be a pity if what potentially seems to be a very useful development were to fall into disrepute through misuse.—I am, etc.,

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Drug-induced Antiplatelet Antibodies

SIR,—Thrombocytopenia is a well-recognized complication of treatment with phenylbutazone and generally results from marrow hypoplasia. However, in a recent case in this hospital a specific platelet antibody was also present, and as far as we are aware this has not previously been recorded.

The patient, a 58-year-old Caucasian woman, was admitted with one week's history of bruising. Six weeks before admission she had been prescribed phenylbutazone 100 mg thrice daily for low back pain, which she had continued up to the time of admission. Otherwise she had had no recent medication of any kind, and her job as a finisher in tailoring had not exposed her to any unusual chemicals.

On admission she was found to have widespread bruising on the limbs, but there was no evidence of bleeding elsewhere and the spleen was not palpable. The initial blood count showed: haemoglobin 10.9 g/100 ml, W.B.C. 2,800/mm³ (neutrophils 330/mm³), platelets 4,600/mm³, E.S.R. 39 mm in one hr. The blood film was normocytic and normochromic and the reticulocyte count was 1.4%. Routine investigations showed normal blood urea and electrolyte levels, liver function tests, and chest x-ray and negative rheumatoid factor, latex and antinuclear factor tests. Serum proteins were 6.2 g/100 ml (albumin 3.7 g/100 ml, globulin 2.5 g/100 ml); protein electrophoresis demonstrated an increase in the α -globulin and quantitative

immunoglobulin estimations an increase in IgM (IgG 510 mg/100 ml, IgA 165 mg/100 ml, IgM 430 mg/100 ml). Plasma haptoglobin level was 200 mg/100 ml and the direct Coombs test negative. The sternal marrow aspirate showed some reduction in cellularity; erythropoietic and granulopoietic activity was normal, but there was a relative decrease in the number of mature granulocytes. Lymphocytes and plasma cells appeared normal, but the number of megakaryocytes was reduced.

Investigations for platelet and leucocyte antibodies were carried out. For platelets the saline agglutination test at 4°, 20°, and 37°C was negative, complement fixation test at 37°C was positive in the presence of phenylbutazone in a concentration of 1 mg/ml, and the Coombs consumption test at 20°C gave a negative result. For leucocytes the saline agglutination test at 4°C, 20°, and 37° was negative and the complement fixation test at 37°C was negative.

The patient was treated with prednisolone, 60 mg/day for 3 weeks when most of the bruising had subsided and she was discharged taking 30 mg/day, the dosage being slowly tailed off over the next four months. There was a gradual improvement in all parameters of the blood count, and at four months the haemoglobin, W.B.C., and platelet count had returned to the normal range. The patient has been entirely symptom-free since her discharge from hospital.

Though drug-induced thrombocytopenia has been described following a large variety of drugs, in only a few has a specific platelet antibody been described.¹ In this patient the general pancytopenia and the hypocellular marrow suggest that the major effect of phenylbutazone was direct marrow toxicity, and this is supported by the slow haematological response despite the use of steroids. However, both direct marrow suppression and platelet destruction by specific antibodies are recognized in the thrombocytopenia seen with thiazides.^{2,3} We are postulating a similar mechanism for the thrombocytopenia caused by phenylbutazone in our patient, and suggest that antiplatelet antibodies should be sought for in all cases of this type.

We would like to thank Dr. Maxwell Telling for permission to report this case.

—We are, etc.,

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- ¹ Aster, R. H., in *Haematology*, ed. W. J., Williams, E., Bentler, A. J., Erslev, and R. W., Rundles, New York, McGraw-Hill, 1972.

- ² Rodrigues, S. U., Leikin, S. L., and Hiller, M. C., *New England Journal of Medicine*, 1964, 270, 881.

- ³ Eisner, E. V., and Crowell, E. B., *Journal of the American Medical Association*, 1971, 215, 480.

Infectious Mononucleosis

SIR,—I have read with interest the article by Dr. H. Pullen (12 May, p. 350). Infectious mononucleosis still represents a rather frequent haemopathy with peculiar features and some obscure nosological aspects. In Dr. Pullen's article the description of the clinical symptomatology fails to include two signs which may be very useful in practice.

The first is eyelid oedema, which may cause narrowing of the palpebral fissure. According to Hoagland and Wintrobe¹ this symptom occurs in 36% of cases. But its diagnostic importance increases because of its early appearance, as confirmed by my recent observations.

The second feature missing from the article is the pain caused by the swollen lymph nodes. In certain cases of lymphaden-