the fact that they are hospital-based in deciding which arrangement is the better. But I am certain of one thing: that there is need for experiment and that in this most important work policy based purely on departmental decree is not good enough. If, in the broadest sense, we are to get value for money this airing of the resettlement of the disabled should be followed by a planned comparative study.—I am, etc.,

H. J. SEDDON

London W.1

SIR,—Disabled men are discharged from this unit after burns, industrial, or road traffic injuries. When they leave they know to what work they may expect to return. They leave as wage earners not as invalids. We owe this state of affairs to Mr. J. W. Daniel (30 October, p. 277), our hospital rehabilitation officer. He sees our patients in hospital as soon after injury as they are lucid, and their employers within days, while both remember the patient as a fit man. Arrangements are made then for his return to the right job, at the right time. His convalescence is a preparation for work, and the return to work the beginning of the best form of physiotherapy and psychotherapy that exists—that is, reward for effort by money rather than by the compliment of a few degrees of added movement.

By contrast district rehabilitation officers fail, and must fail, because they are presented with a man who is already psychologically an invalid and then hopeless. Their liaison with hospital doctors is non-existent. Too often, patients fit for the right job do nothing, because the challenge of something new is too great for them. The district rehabilitation officer is too late to help, often too ill-formed to advise, and too remote to relate. How can he succeed?

Every hospital dealing with trauma should have a hospital rehabilitation officer. With such measures thousands of pounds would be spent and millions saved.—I am, etc.,

ROY SANDERS

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SIR,—The excellent article of Dr. D. A. Brewerton and Mr. J. W. Daniel (30 October, p. 277) prompts me to add the following comments.

I have known Mr. J. W. Daniel since 1959 in his capacity as a resettlement officer in Mount Vernon Plastic Centre. His contribution to our patients was not only to return them to work early. On many occasions both myself and my colleagues have discussed with Mr. Daniel suitability of the particular operative procedure for patients under our care. His opinion based on the detailed knowledge of patients' work requirement has always been invaluable.

I would like to point out one line in their discussion which to me underlines the basic principle: "It is a serious error to think that rehabilitation is a separate process divorced from other medical care."—I am, etc.,

M. F. STRANG

Mount Vernon Hospital,
Northwood, Middx

Sclerosant Injection for Varicose Veins

SIR,—We have recently been concerned with a case of accidental intra-arterial injection of sclerosant during the course of injection compression treatment of varicose veins. In this instance the operator was a skilled vascular surgeon with considerable experience of the method. The varicosities were in the left ankle and lower calf regions. After the first injection had been made on the inner side of the ankle, not far behind the malleolus, the patient experienced a burning pain in the ankle. The toes soon showed signs of ischaemia and though the accident was recognized, and correctly treated by the admission of the patient for infusion of low molecular weight dextran and heparin there was ischaemic damage to the great and second toe and a large slough along the course of the posterior tibial artery proximal to the point of injection. Arteriography subsequently confirmed that the posterior tibial artery was occluded over the area in question and that the lower end of the peroneal was also occluded most probably through the communicating artery not far from the injection point. Whether this injection was into the posterior tibial itself or into the medial malleolar branch is not certain.

The purpose of our writing this letter is to draw the attention of the profession to the risk of injecting varicosities by the empty vein technique or indeed by any other for that matter in the ankle region where arteries may be quite close to the varicosities in question. We understand from colleagues that there have been at least five other cases of similar accidents so we hope that the warning will be recognized as a possible hazard of this otherwise excellent treatment.—We are, etc.,

P. G. C. MARTIN
H. H. G. EASTCOTT

London N.W.1

Manpower and Financial Incentives

SIR,—Mr. F. J. Bramble (*Supplement*, 13 November, p. 34) is reported to have "no doubt that if a financial incentive to attract doctors to unpopular areas was introduced in the hospital service—as happened in general practice—it might be effective." This statement raises the interesting question of whether financial incentives in general practice have in fact resulted in a movement of general practitioners to unpopular practice areas. The results of a large-scale investigation which this unit has just completed into patterns of geographical mobility and residential settlement among general practitioners indicate that the financial incentives which existed from 1966 to 1970 acted as effective inducements under certain limited circumstances, but that non-monetary factors were much more significant in most decisions about where to practise.

Our study, which it is hoped will be published next year, describes in some detail the complex motivations involved in the residential mobility of doctors, and clearly warns against over-simplified prescriptions in manpower planning.—We are, etc.,

J. R. BUTLER
J. M. BEVANHealth Services Research Unit,
University of Kent at Canterbury

Pupillary Mobility and Skin Colour

SIR,—Your leading article (30 October, p. 252) discusses the interesting work of Emiru,¹ who demonstrated that the pupils of Africans are less susceptible than those of Europeans to the mydriatic effects of homatropine and phenylephrine. However, your description of this research as a "pioneer study" is not correct. Readers may be interested in the study carried out by Obianwu and Rand in 1965² along similar lines.

The mydriatic effect of ephedrine eye drops was compared in European, Chinese, Indian, and African students. It was found that the mydriatic response was inversely proportional to the degree of pigmentation of the iris. There is evidence that this difference is due to a more powerful reflex miosis in subjects with pigmented irises. As suggested in your leading article, this may represent an adaptation to the environment. Since the decreased mydriatic response was observed in Africans residing in the United Kingdom, the adaptation seems likely to be genetic in origin and to have become general through natural selection.—I am, etc.,

A. S. D. SPIERS

Medical Research Council Leukaemia Unit,
Royal Postgraduate Medical School,
London W.121 Emiru, U. P., *British Journal of Ophthalmology*, 1971, 55, 538.2 Obianwu, H. O., and Rand, M. J., *British Journal of Ophthalmology*, 1965, 49, 264.

Crystals in Skeletal Muscle

SIR,—In a recent publication Watts and collaborators¹ and now in your leading article (23 October, p. 185) reference is made to crystals of hypoxanthine, xanthine, allopurinol, and oxipurinol in "muscles of patients with gout who were being treated with allopurinol." The authors do acknowledge that the patients exhibit no symptoms associated with such crystals, and that "allopurinol can still be recommended,"¹ but suggest that studies more prolonged than the 8-9 years now available may be necessary to evaluate their accumulation and effects.

The data, however, show such crystals, not in muscle per se, but in specimens frozen in liquid nitrogen at -196°C or in hexane at -70°C and studied at -20°C . The elementary fact seems to have escaped attention that crystals can precipitate only when the concentration of solute exceeds the solubility point, and that the substances in question have rather high temperature coefficients of solubility. The reported levels of xanthine in specimens from xanthinuric patients may be close to the solubility limits (450 and 315 ng/mg dried tissue² (108 and 75.5 ng/mg wet weight) compared to 200 ng/mg in a saturated solution in plasma at 37°C), and precipitation may be plausible if one is willing to assume somewhat localized concentrations. Similarly, in untreated gouty patients, urate concentrations in muscle may reach levels where precipitation is possible. On the other hand, the concentrations of hypoxanthine, xanthine, allopurinol, oxipurinol, and uric acid in muscle biopsy specimens from allopurinol-treated patients are well below saturation levels in every instance, and only small fractions of the solubility in most instances. Thus, crystals of hypoxanthine are reported by Watts,¹ but

the highest concentration found in any of the specimens (12 ng/mg wet weight) was only 1.1% of the saturation level (1,150 ng/ml plasma at 37°).³ Crystals of allopurinol are mentioned; the highest concentration reported in muscle (5 ng/mg wet weight) is 0.6% of the corresponding solubility in water at 37°.⁴ It is clear that all the crystals reported in specimens from allopurinol-treated patients must be artefacts contingent on the cooling (and probable dehydration) employed in the preparation of the specimens for crystallography, and have no implications whatever for therapy with allopurinol.

It is of interest in this connexion that studies in our laboratory have confirmed the presence of xanthine in rat muscle at levels several times those of plasma, a finding consonant with an indigenous origin of muscle oxypurines.⁵ It is of further interest that these levels are unchanged after acute and chronic administration of allopurinol.⁶—I am, etc.,

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Research Triangle Park,
North Carolina, U.S.A.

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Hypoglycaemia in Infancy and Childhood

SIR,—Dr. J. C. Haworth (30 October, p. 304) quotes the incidence of neurological sequelae among 48 infants who had experienced hypoglycaemia in the neonatal period, but no mention is made of the incidence of similar sequelae among a control group. In the absence of this data it cannot be concluded that the neurological damage arose as a result of the low blood glucose levels, however tempting it may be to assume so.

In the only adequately controlled study of which I am aware¹ 41 hypoglycaemic infants were reviewed at a mean age of 51 months, and the incidence of cerebral damage did not differ from that in the control group who had been matched for age, sex, birthweight, gestational length, nutritional status, Apgar score, social class, and position in family.

Regarding the age of onset of hypoglycaemia, 85% of all infants who are going to develop hypoglycaemia do so during the first 24 hours of life, and of these 81% elevate their blood glucose levels without intravenous therapy during the same period.² Thus 69% of all infants with hypoglycaemia have their low blood glucose levels confined to the first day of life.

Dr. Haworth doubts that "true" symptomatic hypoglycaemia usually occurs after the first day of life. Though I agree that it does not invariably do so, a review of 56 such cases reported in the literature between 1959 and 1964 (Table) reveals that only six had

biochemical confirmation of hypoglycaemia (defined as a blood glucose level of less than 20 mg/100 ml) during the first day of life.

Certainly if infants are both hypoglycaemic and symptomatic, an intravenous therapeutic diagnostic test dose of glucose is indicated, and as I have pointed out (21 August, p. 475) even asymptomatic cases are probably best treated prophylactically after the first day of life. This course of action would have resulted in the treatment of the case reported in Dr. Haworth's letter long before symptoms commenced at the age of 75 hours.

Finally, I am sure that Dr. Haworth would agree that the first aim of treatment should be the prevention of hypoglycaemia by ensuring an adequate calorie intake and by the avoidance of hypothermia.—I am, etc.,

A. D. GRIFFITHS

Nevill Hall Hospital,
Abergavenny, Mon

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Need for Asylums

SIR,—In your leading article entitled "Mental Hospital Revolution" (30 October, p. 249) you refer to the latest report on mental hospital patient turnover for the period of 1956-69. There is much in your article to raise a quiver in the nostril of an old mental hospital war-horse, and I write as one who superintended a mental hospital of 2,500 beds for over 20 years. But I deal with one point only—namely, your reference to the increasing number of offenders remanded for psychiatric reports. Since my retirement from the National Health Service I have gained some years' experience as a visiting psychiatrist to a prison that houses recidivists on long sentences. In this occupation I have been truly horrified by the considerable number of so-called criminals who are chronic schizophrenics and mental subnormals of I.Q. ranging from 50 to 70. When it comes to the time for release of these poor socially non-viable people I write to the psychiatrists who are best placed to attend to their essential after-care, only to be given a rebuff in terms such as "I cannot see my way to accepting this case." Now, this might not be so bad were the psychiatrist and the patient strangers to each other, but, alas, it generally is the case that the psychiatrist concerned is the very man who started the patient on his "criminal" career by turning him out of his only refuge.

I have come to believe it is useless to appeal to any mental hospital psychiatrist

these days to provide the kind of services the public require, as distinct from the role that the psychiatrist envisages for himself. I suggest that, above all, the public feels the need for restoring the asylum. An asylum, in my view, should be a place that, in the main protects the inmate from the demands of society, and to a minor degree protects society from the vagaries of the inmates. It thus needs a boundary wall or fence, and such a thing is abhorrent in a modern mental hospital. We do not need the boundary wall to incarcerate dangerous criminals. Such people would scale any boundary wall. No; the greatest liability is the care of geriatric patients, who invariably will choose a foggy night in winter to wander away, only to be found dead from exposure the next day.

Let us get it clear from the start that the re-creation of the asylum is a matter for social workers, not for medical or paramedical personnel. It does not need to be staffed by psychiatric nurses, psychiatrists in training, or other professional staff, for such people would obviously clash with inmates who do not seek any medical help.—I am, etc.,

O. W. S. FITZGERALD

Salcombe,
South Devon

Renal Tubular Acidosis

SIR,—The interesting case described by Mr. N. R. Farid and Dr. D. C. Evered (23 October, p. 233) could not be diagnosed fully because the pathology presumably had altered after five years. The residual findings were hyperglobulinaemia, positive antinuclear and rheumatoid factors, and lymphorrhages with fibrosis in the kidney. A similar chronic case¹ showed at renal biopsy tubular atrophy with calcification and macrophages as well as round cells interstitially. The patient, who earlier showed hepatosplenomegaly, had nephrogenic diabetes insipidus and histological evidence of Sjögren's syndrome. The authors suggested that "an unrecognized substance may have acted as an allergen and a nephrotoxin." Six out of nine cases of Sjögren's syndrome had interstitial nephritis, and one also had lymphocytic infiltration of the thyroid and biliary cirrhosis.² A man who was ill for only a few months developed nephrogenic diabetes insipidus, and microgranulomas without giant-cells were found in the renal biopsy.³ His symptoms cleared up in less than a year, but he had generalized sarcoidosis. He was given prednisone for three months, and the hilar lymphadenopathy regressed considerably.

The first two cases also may have had sarcoidosis, which healed incompletely, leaving behind the lymphocytic infiltration and persisting hyperglobulinaemia. The defensive immune reactions of sarcoidosis⁴ may continue long after the granulomas have "healed." Unfortunately, the late lesions are diagnosable only by exclusion.—I am, etc.,

G. A. MACGREGOR

Chilworth, Surrey

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No. of Cases Studied	No. with onset within the first 24 hours of life	Comment
8 ^a	None	
6 ^a	None	
10 ^a	1	There was no biochemical confirmation of hypoglycaemia during the first day of life in this case
8 ^a	None	
24 ^a	9	There was no biochemical confirmation of hypoglycaemia during the first day of life in three of these cases.