

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°).<sup>3</sup> 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°.<sup>4</sup> 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.<sup>5</sup> It is of further interest that these levels are unchanged after acute and chronic administration of allopurinol.<sup>6</sup>—I am, etc.,

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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 aware<sup>1</sup> 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.<sup>2</sup> 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.,

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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.,

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### 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 case<sup>1</sup> 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.<sup>2</sup> 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.<sup>3</sup> 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 sarcoidosis<sup>4</sup> may continue long after the granulomas have "healed." Unfortunately, the late lesions are diagnosable only by exclusion.—I am, etc.,

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