We can only agree with Dr Skrabanek that there are considerable difficulties with the concept of schizophrenia, syndrome, disease, or behaviour pattern, and he is correct in concluding that we would like to discover an organic cause or, for that matter, a psychological one. However, our observations on typhoid catatonia had little bearing on that question and our argument was directed to showing that typhoid catatonia does not resemble schizophrenia, in contrast to other conditions such as the psychoses associated with epilepsy² or amphetamine abuse,³ which very closely resemble schizophrenia and for that reason have been called "symptomatic schizophrenias." The notion of symptomatic schizophrenia seems to us to be useful in the present state of knowledge and ignorance. "Schizophrenia," like "dropsy," may indeed, in course of time, become redundant, but nevertheless describes something real.

The implication in Dr Skrabanek's second paragraph that physicians "in our part of the world" have somehow superior ethical or conceptual understanding is not only insulting but peculiarly inappropriate, as the senior author had his entire medical, psychiatric (and ethical) training in Ireland. We simply hope that our observations and experience in India may also be of some interest and value to physicians elsewhere.

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¹ Bleuler, E, *Dementia Praecox or the Group of Schizophrenias*, trans J Zinkin, p 248. New York, International Universities Press, 1950.

² Slater, E, Beard, A W, and Glithero, E, *British Journal of Psychiatry*, 1963, **109**, 95.

³ Connell, P H, *Amphetamine Psychosis*. London, Oxford University Press, 1958.

Catatonia after malaria

SIR,—The article on catatonia after typhoid fever by Drs W R Breakey and A K Kala (6 August, p 357) was most interesting. Enteric fevers are common in Rhodesia in the rainy season, but I have never observed this phenomenon.

I have, however, often seen catatonia after malaria, especially cerebral malaria. The subjects are usually African teenage girls. One sees withdrawal, mutism, and waxy flexibility, sometimes with statue posturing. On one occasion I had two young African girls standing on either side of the ward all day like sentries. Delusions and hallucinations do not seem to be present and I have never elicited echopraxia or echolalia. Peripheral blood-films are not always positive in the cerebral cases (as observed by Manson-Bahr) but fever responds to intravenous quinine infusion. Dexamethasone is usually given in these cases. Catatonia has always responded to chlorpromazine in about two weeks and no relapses have been observed.

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Inhumanity to man

SIR,—Your leading article (3 September, p 591) correctly states that under the 1959 Mental Health Act the ultimate responsibility

for admitting patients to psychiatric units is that of the responsible medical officer. However, various other agencies such as social workers, general practitioners, and the police influence the pattern of psychiatric referrals and hence admission. The police, for example, have the option of taking a person who appears to be suffering from a mental disorder and to be in need of care and control to a "place of safety." Alternatively, if an offence has been committed the individual can be charged.

Working in the psychiatric unit of an inner London general hospital, we formed the impression that a disproportionate number of patients admitted under section 136 of the Mental Health Act (that is, directly via the police) or section 60 (that is, from prison) were members of ethnic minority groups. Review of 46 consecutive section 136 patients confirmed this, as 35% were born in the New Commonwealth, while the percentage of New Commonwealth immigrants in the borough aged over 15 was only 13 (1971 Census¹). Sims and Symonds² in Birmingham have reported a fourfold over-representation of West Indians in their series of 252 section 136 admissions.

The same trend was even more obvious in the analysis of 88 consecutive admissions to the hospital under section 60, since 50% of these patients were New Commonwealth immigrants. Many of the immigrant patients had committed relatively trivial offences (such as travelling on the underground without a ticket or failing to pay for a cup of tea in a snack bar), as had a smaller proportion of the United Kingdom-born patients.

Research is required to explain the over-representation of Black patients admitted under section 136 and section 60. The more florid acute psychotic states observed in patients from the Caribbean and Africa³⁻⁶ may account for at least some of the section 136 admissions. This explanation would not apply to the section 60 patients, however, since the majority of these patients had classical (Kraepelinian) schizophrenia.

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¹ Office of Population Censuses and Surveys, *Census of Great Britain 1971, Greater London County Report Part 1*. London, HMSO, 1974.

² Sims, A C P, and Symonds, R L, *British Journal of Psychiatry*, 1975, **127**, 171.

³ Constant, J, *Psychopathologie Africaine*, 1972, **8**, 169.

⁴ Lambo, T A, *British Medical Journal*, 1960, **2**, 1.

⁵ Royes, K, *Proceedings of the Third World Congress of Psychiatry*, 1961, vol 2, p 1121. Toronto, University of Toronto Press, 1962.

⁶ Sutter, J-M, et al, *Encyclopédie Médicale et Chirurgicale (Psychiatrie, 4)*. Paris, Editions Techniques, 1974.

SIR,—May I stress the obvious, that after every kind of medical, paramedical, social, and other help large numbers of people with senile dementia, damaging schizophrenia, or severe mental handicap due to brain disease, injury, or deficit need skilled, devoted care for life?

Accepting that steady efforts must go forward to strengthen available supports outside hospitals, nevertheless absolute priority should be given to enhancing the morale and status of nurses who undertake the permanent care of the most demented, chronically psychotic, and severely mentally handicapped.

Your leading article "Inhumanity to man" (3 September, p 591) was good, although 15 years late. One week later your leading article

"Day services for mentally handicapped adults" (10 September, p 652) welcomes the first move to substitute teachers for nurses and a shift to less beds and more care in the same "community" which has failed the old and the psychotic by reducing beds.

This is a plea that we hasten slowly, lest domestic catastrophes, violence, and horror situations, now avoidable, become inescapable. If the calling to life-long care is demeaned further, irreparable damage could easily follow. Most hospitals for the mentally handicapped of 500 beds or less, preferably about 250, will bear the closest scrutiny as centres of happy, genuine community life and it is these which should form the keys to future care and not the renamed training centres. It would be justifiable to change the name of such communities to "hospitals for the severely brain damaged."

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Radiation and growth hormone deficiency

SIR,—We would like to comment on your recent leading article (27 August, p 536) on the possible effects of brain irradiation in children on future growth and development. We have seen a patient with abnormalities of both growth hormone (GH) and prolactin secretion after a course of radiotherapy some eight years earlier.

A 5-year-old girl presented with slight proptosis of the right eye, which was found to be blind. There was enlargement of the optic foramen on x-ray. At surgical exploration a right optic nerve glioma was demonstrated extending back to the chiasm. Subsequently she was treated with external irradiation to the region—4290 rads in 13 treatments.

She was later investigated at age 13·7 years for short stature, with height at the 3rd percentile. She had normal secondary sex characters and periods had started two years before. A GH deficiency was demonstrated, with the plasma GH concentration not rising more than 3·0 IU/l during insulin hypoglycaemia (blood glucose 1·8 mmol/l (32 mg/100 ml)). The plasma cortisol concentration rose from 335 to 593 nmol/l (12 to 21 µg/100 ml) during this test, and there was a normal response of thyrotrophin to thyrotrophin-releasing hormone (TRH) injection (rising from 1 mU/l to 21 mU/l at 20 min after TRH). The basal plasma luteinising hormone level was 6·0 mU/l, while the plasma prolactin concentration was raised to 20 µg/l. Bone age was not retarded. The patient is at present being assessed for human GH treatment.

The GH abnormalities in this patient correlate with the results described by Shalet et al,¹ who showed that if a threshold of about 3000 rads radiation dose to the brain is exceeded there is an absence of GH response to hypoglycaemia. In other reports^{2,3} there was little evidence of other endocrine deficits except occasional reduced gonadotrophin responses. Serum prolactin was not measured by these workers and it is therefore of interest that an elevated level was found in this patient. An increased serum prolactin concentration would be in keeping with the idea that the effects of irradiation on pituitary secretion are the result of a hypothalamic disturbance.

In a large proportion of the patients who have received cerebral irradiation there seems to be a marked sensitivity of regions responsible for GH secretion. In view of this it is obviously important to measure accurately the heights of children at risk of growth retardation after irradiation at subsequent follow-up visits. If growth velocity⁴ is estimated any tendency

for a reduced final height attainment will be determined and assessed early. Treatment with human GH should be started as soon as possible to obtain satisfactory results. In our patient there was a delay in the institution of investigations. If bone age is advanced at the time of treatment the chance of great benefit is slight.

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¹ Shalet, S M, *et al*, *Clinical Endocrinology*, 1976, 5, 287.

² Shalet, S M, *et al*, *Lancet*, 1975, 2, 104.

³ Harrop, J S, *et al*, *Clinical Endocrinology*, 1976, 5, 313.

⁴ Tanner, J M, *et al*, *Archives of Disease in Childhood*, 1966, 41, 454.

Lithium and thyrotoxicosis

SIR,—In my letter (17 September, p 765) commenting on the statement in your leading article (6 August, p 346) that "there is at least one report of lithium-induced thyrotoxicosis"¹ I omitted to state that there had been other reports of thyrotoxicosis in patients on long-term lithium therapy for manic-depressive illness.²⁻⁵

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¹ Franklin, L M, *New Zealand Medical Journal*, 1974, 79, 782.

² Cubitt, T, *Lancet*, 1976, 1, 1247.

³ Bujaque, H H, and Myers, D H, *New Zealand Medical Journal*, 1974, 79, 1409.

⁴ Rosser, R, *British Journal of Psychiatry*, 1976, 128, 61.

⁵ Brownlie, B E W, *et al*, *Australian and New Zealand Journal of Medicine*, 1976, 6, 223.

Increased incidence of poliomyelitis

SIR,—In a previous letter (Drs J Nagington and D Rubenstein, 26 February, p 573) attention was drawn to the increased incidence of paralytic poliomyelitis in Britain at the end of 1976. A clearer picture of the change has now emerged.

Up to the end of July this year there have been 13 cases of confirmed paralytic poliomyelitis compared with four in the same period the previous year. It is noteworthy that all 13 were in children aged 7 years or under—that is, a return to "infantile paralysis." Ten of the 13 were caused by type 1 virus, two by type 2, and one by type 3, and not a single patient had received any polio vaccine at any time. Since all but two were over 6 months of age vaccine, if given, should have protected 11 of them.

It is evident that there is need to intensify efforts to achieve the maximum possible vaccination of children under 5 years of age.

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Dextrostix/Eyetone in the insulin hypoglycaemia test

SIR,—An acceptable method of monitoring insulin tolerance tests would enhance both

their efficiency and their safety by ensuring adequate but not dangerous hypoglycaemia. The Dextrostix/Eyetone system as proposed by Dr M A Preece and Mr R G Newall (16 July, p 152) is "tantalisingly close" to an adequate monitor, as Dr A M Bold and others point out (13 August, p 459). We feel that this system has a place in monitoring hypoglycaemia provided its limitations are recognised and certain practical procedures observed.

Even though venous blood gives more reliable results than capillary blood, we found that Dextrostix/Eyetone systematically overestimated venous plasma glucose concentrations less than 3.3 mmol/l (60 mg/100 ml). The discrepancy was greatest with the lowest plasma glucose concentrations. Clinical features of hypoglycaemia should not be ignored because of reassuring Dextrostix readings both because of this bias and also because of occasional incorrect high Dextrostix values. Although venous Dextrostix/Eyetone and conventional determinations correlate reasonably, the correlation for capillary blood is not as good. Moreover, if the finger is pricked when still wet from the alcohol preparation dangerously misleading results can occur (for example, 12.2 mmol/l (220 mg/100 ml) instead of 2.3 mmol/l (41.5 mg/100 ml)).^{1 2}

The Dextrostix/Eyetone system should be set up correctly. The two-point calibration system described by Dr Preece and Mr Newall is inadequate, as is their method of detecting deteriorated Dextrostix. The following simple operating procedure is accepted by nursing staff and gives reasonable precision (coefficient of variation 5.4% at 2.5 mmol/l (45 mg/100 ml)).

(1) Turn on the Eyetone 30 min before use.

(2) At the beginning of each week set the reflectance strips with the adjustment screws, making sure the white line on the thumbwheel is in the middle of the visible region.

(3) Set up calibration standard to 7.2 mmol/l (130 mg/100 ml) with the thumbwheel, following the instructions supplied by Ames for use of Dextrostix.

(4) Check the machine calibration by testing one of the three Tek-Chek controls. These should read 2.5, 5.0, and 13.9 mmol/l (45, 90, and 250 mg/100 ml) $\pm$ 10% respectively.

This procedure is repeated weekly. In between the machine is left on continuously and steps 3 and 4 carried out whenever the Eyetone has not been used for two or more hours or if there is a change of operator.

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¹ Phillips, P J, *et al*, *Medical Journal of Australia*, 1977, 1, 790.

² Ball, S G, and Hughes, A S B, *British Medical Journal*, 1976, 1, 1279.

A tennis elbow support

SIR,—Tennis elbow is a painful condition and stops an otherwise active and healthy adult from taking part in sports or even from pursuing his work. In most cases a local steroid injection will effect a "cure," but any return to strenuous activity like tennis or gardening can cause a relapse of the tennis elbow.

I suffer from tennis elbow myself. I am also rather interested in tennis. Last summer,

after two local steroid injections with only temporary relief, I found that a handkerchief tied around the forearm just below the elbow enabled me to play tennis and yet did not restrict the movements of my elbow. I followed this idea up and got a surgical appliance firm to make a tennis elbow support which would grip the upper part of the forearm without interfering either with the elbow joint or with the blood circulation of the limb. The material used was a special type of rubber with surface cross-striations and was fairly rigid when stretched. This was held together by Velcro fastening. The exact specifications were: white calendered rubber gauge 40/1000, width 2½ in (5.25 cm), length 13 in (33 cm), with twin Velcro fastening 4 in (10 cm) long giving a 3-in (7.5 cm) adjustment.

The tennis elbow support is placed around the upper part of the forearm just below the head of the radius. This anatomical landmark is easily identified by palpation. The forearm muscles are relaxed and the support applied without stretching on to the bare forearm. The special texture of the rubber ensures that it does not slip. Sweat holds it more firmly in place. When the patient grips anything, as during the act of holding a tennis racket, the forearm muscles contract and are supported near the attachment to the epicondyles of the humerus. This support is sufficient to ease the symptoms of tennis elbow. It can be worn the whole day or during any strenuous activity. It has been used on occasions in cases of golfer's elbow.

As the preliminary results are encouraging I thought it would be useful to publish this. Fifty of these prototype tennis elbow supports were initially supplied by M Masters and Sons Ltd, of London. Within three months another 100 supports were made available for trial use. Seventy-five of these supports have been used by patients in the Enfield district who have had recurrence of their symptoms. Fifty of these patients were personally reviewed by me; 46 (92%) have been helped by the tennis elbow support, having had less pain and being able to carry out most of their activities without any symptoms. They are able to play sports like tennis and squash.

I feel the preliminary results are encouraging enough for any surgeon interested in the condition of tennis elbow to try these supports in patients with recurrent symptoms. Their use might well reduce the number of patients having surgery for resistant tennis elbow.

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Neonatal electrocardiogram and cot deaths

SIR,—With reference to the articles by Dr B R Keeton and others (3 September, p 600), I would like to report a case of actual cot death which, in retrospect, may have been caused by cardiac arrhythmia.

A female infant was delivered by emergency lower-segment caesarean section because of fulminant pre-eclampsia. The Apgar score at 1 min was one and the baby required intubation and positive pressure respiration for 10 min. On examination the gestational age was 36 weeks by the Dubowitz criteria and she was small for dates, weighing 1.28 kg. In spite of these problems her initial progress was satisfactory apart from excessive crying which required sedation with chloral.

On the evening of her 29th day she was found