Thorpe Coombe Maternity Hospital is in Walthamstow in the outer London area. It has a total of 70 beds for antenatal care, first stage of labour, and the lying-in period. Since planned early discharge was introduced four years ago confinements have increased to over 2,000 annually. The hospital serves three local authorities, and for the success of the scheme close co-operation is essential. Patients for early discharge are selected during the antenatal period by a consultant. The request may come from the mother herself or her family doctor, or the hospital may ask if she is able to be nursed at home after 48 hours. Selected mothers sign an agreement and provide the hospital with the name and telephone number of their doctor and district midwife. In all cases the home is visited and assessed by a district midwife, thus ensuring her co-operation, and the general practitioner and local authority are notified. These plans having been made well in advance, discharge from hospital can proceed as a smooth routine. The antenatal care of the patient is shared by the hospital, general practitioner, and district midwife, so that she is known to all concerned.

Before discharge all mothers and infants are seen by the obstetric registrar, and in case of doubt are referred to a consultant obstetrician or paediatrician. If necessary they are detained in hospital for longer than 48 hours. Each morning the ward sister notifies a clerk of the patients for early discharge and she then informs the family doctor and midwife. The patient, who has been up and about, leaves by private transport accompanied by a relative. This has proved a satisfactory arrangement and an ambulance is seldom required. Very few patients have needed readmission to hospital. In one of the areas concerned these mothers are cared for by a retired midwife working part-time, in the other two areas by the full-time midwives. In addition to mothers booked for early discharge those booked for home confinement who are admitted in late pregnancy or labour are allowed home early provided they are fit.

The arrangements described were arrived at by trial and error accompanied by good will. They are simple and require no elaborate administrative machinery. They provide additional safety for many mothers and infants, and this makes the additional work worth while.—I am, etc.,

D. JOAN BRYSON.

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Fallacies in Four Fevers

SIR,—I support the suggestions for shortening the official period of infectivity of childish fevers (7 October, p. 41). But might it not be better to go even further?

School epidemics are poorly controlled by exclusion of children with infectious fevers. It is much less inconvenient, and often less unpleasant as well, to suffer these illnesses when young. Therefore I believe it would be better to abandon ineffective attempts at control, and instead try to infect as many children as possible. In order to do this I propose that *general policy* be one of inclusion, rather than of exclusion, of well but infectious children with rubella, mumps, and chicken-pox. Infectious measling children are not in any case well enough for school.

This policy would only be general, and schools could at any time declare a season of exclusion during important exam terms and the like. Families with special reasons for not wanting an infectious member would keep their children from school, but should in logic exclude also all uninfected members from public places during the epidemic.—I am, etc.,

JOHN E. HODGKIN.

Bury St. Edmunds, Suffolk.

SIR,—Professor R. S. Illingworth's short article (7 October, p. 41) was as usual full of common sense, and I am in complete agreement with his conclusions. With regard to school exclusion of cases and contacts of the common infectious diseases, the following recommendations were adopted in Oxford in February 1963:

Disease	*Minimum* Period of Exclusion from School *provided child appears well*	
	Patients	Family Contacts
Chicken-pox	Seven days from appearance of rash; all the scabs need not have separated.	There is no *routine* exclusion of contacts of any of these infectious diseases but individual children *may be excluded* on the advice of a general medical practitioner or a school medical officer.
Dysentery (Sonne)	Until complete cessation of diarrhoea.	
Measles	Seven days from appearance of rash.	
Mumps	Until disappearance of all swelling.	
Rubella	Five days from appearance of rash.	
Scarlet Fever	Until clinical recovery.	
Whooping-cough	Until clinical recovery.	

There has been no subsequent occasion on which there has been cause to regret departing from the more rigid criteria laid down in the Ministry of Education Memorandum 1956, which is of course advisory rather than mandatory.—I am, etc.,

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Oxford.

Treatment of Poisoning by Antidepressant Drugs

SIR,—Dr. A. B. Masters (30 September, p. 866) and Dr. C. A. Fuge (14 October, p. 108) both draw attention to sudden relapse and death after seeming recovery from the effects of an overdose of a tricyclic drug. In their original paper (9 September, p. 663) Dr. C. M. Steel and his colleagues also noted this unusual outcome.

It is of great practical and theoretical interest to know the cause of these sudden relapses. They may perhaps be directly caused by persistence of the drug or one of its metabolites, but it is also possible that they result rather from some special feature of the drug-induced illness, and only indirectly from the drug itself. Thus Dr. Steel and his colleagues warn against the risk of precipitating circulatory collapse by allowing patients to be physically active as soon as they seem to have recovered from the overdose. The drug leaves a cardiovascular sensitivity behind it, and physical strain might cause relapse.

I wish to draw attention to the possibility of permanent brain damage which may arise where there is a long delay in instituting treatment. Status epilepticus, hypotension, and hyperpyrexia damaged the brain in the following case:

A normal boy weighing 12.7 kg. and aged 14 months was able to crawl and use five distinct words. He opened a screw-cap bottle and took four to eight tablets of desipramine hydrochloride (Pertofran, Geigy, 100–200 mg.). His parents put him to bed, where next morning he was found to be unrousable. When he reached hospital, 15 hours after his overdose, he was in status epilepticus, with clonic convulsions every five minutes, a weak irregular pulse (40/min. at apex), blood pressure unrecordable, and a rectal temperature of 41° C. Electrocardiogram showed a chaotic mixture of ventricular and supraventricular tachycardia with wide QRS complexes.

Intravenous thiopentone and 100 mg. hydrocortisone were given. Fourteen hours after admission fits had ceased, the blood pressure was 80/50 mm. Hg, pulse was 190 at the apex, and the E.C.G. showed regular ventricular tachycardia. Procaine amide, 50 mg. intravenously over five minutes, produced a slower but irregular ventricular rhythm. Next morning, however, systolic blood pressure was 100 mm., pulse 120 regular, and E.C.G. showed sinus rhythm, but the patient remained unrousable. Two days later convulsions returned, and 15 were noted in the next 13 hours in spite of renewed thiopentone. They ceased when 14 g. urea was given intravenously over half an hour (as 30% "Urevert") to reduce cerebral oedema.

Thereafter the child made only a partial recovery. After 19 days an E.E.G., kindly performed by Mr. D. Lee (Warneford Hospital), showed a medium voltage arrhythmic slow wave pattern in all leads without evidence of any cortical activity. When seen five months later he lay sucking his thumb, rhythmically emitting a constant feeble whine if it was pulled out of his mouth. He could take his weight on his legs, but could not maintain his balance for standing or sitting. His pupils reacted to light, but there was no comprehension of visual stimuli. He opened his mouth to receive food when a spoon touched his lips. Hunger and discomfort caused wailing. Pain made him turn from supine to prone in evasion.

The special feature of this case was the long delay in instituting treatment, as in Dr. Masters's case.

I am grateful to Dr. Desmond MacCarthy for permission to publish this case.

—I am, etc.,

W. R. EDWARDS.

Woodstock, Oxford.

Acute Necrotizing Encephalitis

SIR,—In your leading article (30 September, p. 812) referring to a recent report by two of us[1] on eight cases of acute necrotizing encephalitis you state that, "If an exploratory biopsy is carried out, however, necrotic brain only will be found. . . ." This might seem to imply that cerebral biopsy is of no value in establishing the diagnosis of acute necrotizing encephalitis, when in fact the reverse is the case. In four of the five original cases, and in two more recently encountered, examination of a cerebral biopsy disclosed the characteristic histological features of acute necrotizing encephalitis.

You also observe that it is still uncertain if acute necrotizing encephalitis is due to direct virus invasion or represents yet another antigen-antibody reaction, and whether herpes virus is the only cause or the only one so far demonstrated. We have now encountered 10