tion may be misleading. I wish to report the case of a known drug addict who presented with unusual clinical features.

A man aged 19 returned home one evening at 9 p.m., when he appeared confused and had a staggering gait. At 10 p.m. he started shaking violently and was noticed to be foaming at the mouth. He had two further similar attacks. In between these episodes he remained confused. When seen by his doctor in the early hours of the morning he was found unconscious in bed, trembling, and with widely dilated pupils. His blood pressure was 140/80 mm Hg. There was a history of a head injury sustained in a road traffic accident two years previously with no associated loss of consciousness. As the patient was a known drug addict, although not registered as such, a drug overdose—"probably morphine"—was considered likely and he was urgently referred to hospital.

On arrival in the casualty department the patient was semiconscious. He had a stomach washout. Soon afterwards he became unconscious and developed a major epileptic fit for which he was given diazepam 10 mg intravenously. Following this he remained unconscious and, on examination, he was unresponsive to painful stimuli. Injection marks were noted on the left arm. He was perspiring profusely. Blood pressure was 140/90 mm Hg, pulse 80/min regular. There was no meningism. The pupils were constricted. Reflexes were exaggerated bilaterally. There was bilateral ankle clonus. Both plantar responses were extensor.

On the evidence of the epileptic fits, the profuse perspiration and extensor plantar response, hypoglycaemic coma was diagnosed. The serum salicylate level was reported as nil, serum sodium 132 mEq/l., potassium 2.8 mEq/l., chloride 108 mEq/l. The blood sugar level was 30 mg/100 ml. Fifty millilitres of 50% dextrose was injected intravenously. There was no immediate response, but 10 minutes later the patient suddenly regained consciousness. He admitted that 10 hours before admission he had injected himself intravenously with 2 ml of insulin of unknown strength and type to "give himself a kick." He had obtained the insulin from a diabetic friend. This was his second experience with the drug. He had made use of heroin and other drugs in the past.

Altered colour vision and visual, auditory, and olfactory hallucinations are rare symptoms of hypoglycaemia.¹⁻⁵ Insulin, however, is not commonly known to be used by drug addicts for hallucinatory purpose. It has not been possible to find reports in the literature of similar insulin abuse and it would be interesting to know if other cases have been encountered. It is suggested that hypoglycaemic coma in deeply comatose drug addicts as a result of insulin overdose should be considered in the differential diagnosis.

I wish to thank Dr. F. M. McGowan for his permission to report this case.
—I am, etc

S. RETSAS

Oldchurch Hospital,
Romford, Essex

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Adhesive Patches for Theatre Linen

SIR,—Our Area Linen Service, once known as the hospital laundry, has taken to mending theatre and possibly other linen by putting on an adhesive fabric patch, similar to the "iron-on" patches available for home use. These can be loosened during laundering and sterilization, and could easily find

their way into a wound, where they would be unsuspected and undetectable. I am glad to say that when I discussed this with those concerned they were most helpful and undertook to stop this practice, the potential dangers of which had not been appreciated.

I am reporting this as I understand that other hospital laundries may be using this method. If such a patch were discovered, say at a second laparotomy, the resulting litigation could be salutary.—I am, etc.,

J. W. DICKSON

Ipswich and East Suffolk Hospital,
Ipswich

Thymectomy for Myasthenia Gravis

SIR,—In your leading article (2 September, p. 543) you mention the occurrence of neonatal myasthenia as evidence for the existence of "a transmissible agent able to cross the placenta and cause the disease in the infant." While this may be the correct interpretation, an alternative one can be suggested.

Babies born to myasthenic mothers may suffer from a transient myasthenia-like syndrome. However, this does not always start immediately after birth but in many cases a few hours later.¹ It is possible that the delay in onset is the result of a passage through the placenta of cholinesterase inhibitor drugs taken by the mother. Almost all cases of neonatal myasthenia occur in babies born to mothers taking these drugs, and recent evidence suggests that they may have a noxious effect on the neuromuscular apparatus.^{2,3} Long-term administration to rats has been shown to produce changes mimicking myasthenia.² A similar effect could perhaps be produced in the human fetus. Possibly some of the histological abnormalities seen in non-neonatal myasthenic muscles also result from medication rather than from the underlying disease.—I am, etc.,

A. D. KORCZYN

Beilinson Hospital,
Petah Tikva, Israel

- 1 Namba, T., Brown, S. B., and Grob, D., *Pediatrics*, 1970, 45, 488.
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Malaria in Pregnancy

SIR,—At the present time malaria is being imported into the United Kingdom on an increased scale. At least 100 cases are diagnosed annually, and more may occur which are not notified under the Malaria and Dysentery Regulations (1919). Diagnosis may be dangerously delayed by language difficulties. Further difficulties may arise when a relapse of malaria is associated with pregnancy, as in the following case.

A woman of Asian extraction was admitted to hospital on 15 August 1972. Her doctor wrote "... temperature tonight 105°F, arrived from India two weeks ago. ? Malaria." The history was obtained through an interpreter.

The patient complained of vomiting, severe generalized headache, and fever for three days. She was pregnant, expecting her third baby on 27 October. For the past year she had had episodes of fever, but malaria had been excluded.

On admission the patient was feverish but not desperately ill, with a temperature of 106°F, (41.1°C), pulse 128/min, respirations 38/min,

and blood pressure 70/40 mm Hg. Neck stiffness was minimal. The spleen was not palpably enlarged. The fundus uteri corresponded to the period of amenorrhoea.

Initially, subarachnoid haemorrhage was suspected because of blood in the cerebrospinal fluid but later this was shown to be due to contamination. Pyelonephritis was considered despite the absence of loin tenderness. A blood film, however, showed *Plasmodium vivax*.

Treatment with oral chloroquine, 600 mg as a loading dose then 300 mg daily for four days, was followed by primaquine 15 mg daily for two weeks. The fever, seen to be classically tertian, soon settled. Later, an x-ray revealed the presence of twins. At no time did anaemia occur; the haemoglobin at 38 weeks' gestation was 11.5 g/100 ml. The twins were delivered vaginally at the 39th week. Both babies thrived and there was no evidence of neonatal malaria.

Obstetricians will do well to recall that malaria can occur in any pregnant woman who has been out of the United Kingdom.^{1,2} Pregnancy may lead to a relapse of vivax infection, which can occur up to two years after the initial attack. High fever, especially if tertian, in a pregnant Asian should suggest the diagnosis, which can be confirmed by examination of a suitable blood film. Vivax infection may mimic pyelonephritis of pregnancy and bacteraemic shock, while the fever may cause fetal death or precipitate premature labour. Falciparum infection in pregnancy may cause acute renal failure and in its cerebral form may mimic eclampsia by causing coma; early recognition is essential to save life.

Relapses of vivax infection respond quickly to treatment, preventing premature labour, even, as shown above, in multiple pregnancy. The fetus is not affected by antimalarial drugs apart from quinine, which may cause fetal death. Chloroquine can cause abdominal discomfort and vomiting, while primaquine, when taken daily to prevent a relapse of vivax infection, may lead to haemolytic anaemia if the red cells are deficient in glucose-6 phosphate dehydrogenase.

Congenital malaria is known, but the incidence is very low, probably less than 0.1%.³ If patients in a malarious area took proguanil daily and for four weeks after entering the United Kingdom, relapses of malaria would be entirely prevented.

I would like to thank Dr. V. W. Johnson, under whose care this patient was diagnosed, treated, and cured, for permission to use his clinical records and Dr. W. S. A. Allan, consultant pathologist, for his opinion and advice.—I am, etc.,

ALAN M. SMITH

New Cross Hospital,
Wolverhampton

- 1 Barnes, C. G., *Medical Disorders in Obstetric Practice*, 3rd edn., Oxford, Blackwell, 1970.
- 2 Shute, P. G., and Maryon, M., *British Medical Journal*, 1969, 2, 781.
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Malaria Mortality in Children

SIR,—Your issue of 14 October 1972 has reached me today (7 December). (This is a good example of "modern" postal facilities.) On p. 112 it is stated that Sagnet *et al.*¹ reported a death rate of 47% from the Congo among children under 3 years of age from malaria. I agree with Dr. M. J. Colbourne that a death rate of this sort is hard to believe. Did these children have malaria and nothing else? My own experi-