malaria only 26 (3.9%) of about the same number of Hb SC patients were admitted under similar circumstances. The results are very highly significant ($p < 0.001$).

Though parasite counts are said to be lowest in sickle-cell homozygotes, intermediate in heterozygotes, and highest in normal homozygotes^{3,4} suggesting, perhaps, that malaria in sickle-cell anaemia patients is not severe, it is my view (and that of Edington⁵) that in Ghana malaria in sickle-cell anaemia patients can be very severe, and is a serious common precipitating cause of crisis. I have also seen death result more than once from this. Apart from the mechanisms given by Mr. Adeboye and colleagues "whereby malaria might actually lead to death in the homozygote" the role of pyrexia *per se* in causing *in vivo* sickling is known, while the accompanying hyperhidrosis, anorexia, vomiting, and diarrhoea in young children can lead to serious dehydration with massive intravascular sickling, severe erythrocyte sequestration, and instant death.

G-6-PD deficiency has not been found to be statistically significant in the incidence of crisis admissions due to malaria in Accra. Thompson⁶ found that while sickle-cell trait children were protected against *P. falciparum* sickle-cell trait adults were most often sick from this infection compared with Hb AC adults (lowest incidence) and normal homozygotes ($p < 0.05$).

Without chloroquine many of the Accra sickle-cell disease patients would have died in crisis before now. There is no difference, mortalitywise, between the sickle-cell anaemia patient who is killed by malaria and the one who dies from crisis resulting directly from malaria. No sickler gets a crisis unless there is a change in the *milieu intérieur*, and there is usually a precipitating cause to produce this change. The maxim in Accra, as in Ibadan, is never merely to "treat a sickle-cell crisis" but to search quickly and diligently for the cause and then treat that.—I am, etc.,

I am grateful to Mr. R. G. Carpenter and Miss J. Nixon of the Department of Human Ecology, Cambridge, for their help with statistical analysis of data.

F. I. D. KONOTÉY-AHULU

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SIR,—I was very interested in the report from Ibadan of "Severe Malarial Infection in a Patient with Sickle-cell Anaemia" (22 May, p. 445). On several occasions while working at Awgu in the East Central State of Nigeria I had patients with sickle-cell anaemia who died during an attack of malaria. In at least two cases the diagnosis of the haemoglobinopathy had been confirmed by electrophoresis (at Enugu General Hospital).

The usual pattern was that after diagnosis children with the SS genotype were seen in outpatients instead of at baby welfare; they were kept on routine antimalarials (Daraprim) and folic acid, and the mother was encouraged to bring the child if he had any illness at all. In the fatal cases there was usually a period of regular attendance and then the family defaulted, to be seen next with a severely anaemic child (haemoglobin usually between 10% and 15%) with high fever. Malaria parasites were found in the blood.

Unfortunately I have to write from memory as our records were lost during the civil war, but I feel it is probable that an attack of malaria is often associated with the final illness of children with sickle-cell anaemia, and that as suggested in the article it is likely that the increased resistance to malaria is confined to the AS heterozygote.—I am, etc.,

ANNE SEYMOUR

Findon,
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Insulating Kidney Perfusion Fluid

SIR,—As forecast by Calne¹ an increasing number of donor kidneys for transplantation are being supplied from hospitals peripheral to the transplantation unit. It is the responsibility of the transplant team to provide the staff and materials required to obtain these kidneys. These materials consist of pre-sterilized gowns, gloves, drapes, and instruments and a supply of cooled sterile perfusion fluids. All these materials must be unobtrusive yet readily available for time is a vital factor in obtaining donor kidneys.

Difficulties may arise in maintaining the perfusion fluids at a sufficiently low temperature and for this purpose we have used a modification of a commercially produced "cold box" (Nilo), size 16 in x 9 in x 14 in

(40 cm x 22.5 cm x 35 cm), which will normally maintain the temperature of its contents between 1° and 4°C for three to four hours. An extra layer of insulation in the form of a tailored inner lining of Evazote foam (6 mm thick) was added so that the contents of the box remained at the same low temperature for a longer period of time. To this insulated box is added three bottles of perfusion fluid, a giving set, and a low-reading thermometer and the whole unit stored unsealed in a cold room (4°C) in the transplantation unit.

When a donor kidney situation arises two 1 l. plastic packs of deep frozen saline (−15 to −20°C) (Allen and Hanburys Steriflex) are brought to the cold room and placed

inside the box, which is then sealed. These not only provide a means of maintaining the low temperature within the box for at least 24 hours but also provide a supply of sterile frozen saline to charge the Thermos flasks used to transport the donor kidneys. In a series of experiments using thermocouples the temperature of the perfusion fluids remained between 1° and 4°C for a period of 24 hours. In fact the temperature of these fluids was still below 4°C after 36 hours but the amount of frozen saline remaining in the 1 l. packs was insufficient for use in filling the Thermos flasks.

However, for practical purposes a period of 24 hours is quite adequate for a donor kidney situation. By replacing the cold box and its contents daily a continuous supply of cold perfusion fluids and sterile iced saline can be maintained in close proximity to the potential donor kidneys.

Nilo Cold Box is obtainable from most camping equipment shops. Evazote—expanded Vinyl Acetate—from Expanded Rubber and Plastics Ltd., Mitcham Road, Croydon, Surrey.

—We are, etc.,

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¹ Calne, R., *British Medical Journal*, 1969, **2**, 565.

Bleeding from Dialysis Shunt Sites

SIR,—I have frequently encountered the problem of persistent oozing of blood from the sites of newly created shunts in dialysis patients. These patients have usually been taking anticoagulant drugs, and oozing has persisted even when the wound has been explored for bleeding points and carefully resutured. Because of unwillingness to reverse the anticoagulation in these patients for fear of the shunt clotting, the following manoeuvre in order to arrest the haemorrhage has been developed.

The wound edges are infiltrated with 1-2 ml of bovine thrombin (200 NIH units/ml) and then a compression dressing applied for one hour. This simple technique has been used in three patients with complete success. Apart from some stinging at the site of injection, no complications have been encountered so far.—I am, etc.,

JOHN R. SAIAMAN

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Accidental Infection of Man with *Mycoplasma caviae*

SIR,—*M. caviae* is a species of mycoplasma only recently characterized¹ and apparently normally only found in guinea-pigs.^{1,2} Attempts to transmit the organism under experimental conditions to a variety of laboratory rodents and lagomorphs have been unsuccessful, and even in guinea-pigs the organism failed to show any pathogenic properties. Details of these investigations will be published elsewhere.

During the course of this work, a very

small amount of a suspension of *M. caviae* and complete Freund's adjuvant was inadvertently injected into the plantar surface of the author's thumb. Within 48 hours, the whole of the terminal joint became swollen, congested and painful; at 72 hours, oral tetracycline therapy was initiated, and by the fourth day, the whole hand was swollen. Two days later, the swelling started to subside and gradually reverted to normal during the course of the next four weeks.

Two weeks after the accident, the author had a serum antibody level of 1 in 20 to *M. caviae*, estimated by the metabolic inhibition technique.³ This level rose to 1 in 40 after a further two weeks. Two other people who had been working with the organism in the laboratory showed no detectable antibody level to *M. caviae*.

Although *M. arthritis* can produce subcutaneous abscesses in rats at the site of inoculation,⁴ the majority of mycoplasmas are not associated with localized subcutaneous lesions. This laboratory accident, therefore, demonstrates an unusual characteristic of a newly recorded species of mycoplasma. Apart from this observation of possibly little direct clinical importance, the incident emphasizes the need to instigate immediate treatment of similar laboratory accidents involving micro-organisms whose pathogenic effect to man is unknown.—I am, etc.,

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¹ Hill, A., *Journal of General Microbiology*, 1971, **65**, 109.

² Hill, A., Blackmore, D. K., Francis, R. A., *Veterinary Record*, 1969, **81**, 291.

³ Taylor-Robinson, D., Purcell, R. H., Wong, D. C., and Channock, R. M., *Journal of Hygiene*, 1966, **64**, 91.

⁴ Lemcke, R. M., *Journal of Hygiene*, 1961, **59**, 401.

Allergy to Iprindole (Prondole) with Hepatotoxicity

SIR,—Various reports exist in your correspondence columns incriminating iprindole as the causal agent of jaundice in patients under treatment for a depressive illness (7 February 1970, p. 367; 25 April 1970, p. 238; 7 November 1970, p. 368; 21 November 1970, p. 494). No reports appear to exist, however, where the liver cells could be considered to be undamaged prior to the drug being administered, as demonstrated by normal serum transaminases.

In this practice we have noted several cases of hepatotoxicity demonstrated clinically as jaundice or subclinically with raised serum transaminases after administration of iprindole. One such patient had been investigated for chest pain four months previously and at that time had a normal aspartate transferase level of 95 units/100 ml. After taking iprindole for two weeks the aspartate transferase was 150 units/100 ml and the patient presented with shivering, and a dark urine.

No permanent changes seem to have occurred in any patients, and we have found iprindole an excellent antidepressive drug. In view of its otherwise noted freedom from side effects it would appear important to establish the seriousness of possible liver damage after this drug in assessing its true place in the physical treatment of depressive illness.—I am, etc.,

ANTHONY D. CLIFT

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Congenital Dislocation of the Head of the Radius

SIR,—Two sisters with this anomaly occurring bilaterally in the absence of any other abnormality have recently been seen. A review by Almquist, Gordon, and Blue¹ made no mention of the isolated anomaly occurring as a familial characteristic. The girls were aged 7 years and 18 months when first seen. Both had been full-term normal deliveries following normal pregnancies. The mother had noticed a deformity of the elder child's elbows but had accepted this as normal. In neither case was there any history of trauma and the deformities caused no disability. Seven other siblings were normal, with the exception of a brother aged 16 years with a post-traumatic subluxing shoulder. Two paternal first cousins had congenital dislocation of the hips but examination of close relatives showed no other abnormalities.

Clinically both children showed cubitus valgus of 30° in the elder girl and of 40° in the younger (Fig. 1). The displaced radial heads were not visible but were fairly easily

Fig. 1. The two sisters aged 7 years and 18 months respectively, showing the marked cubitus valgus.

Fig. 2. Radiographs of the elbows of the elder child showing the anterior dislocation of the radial heads. The articular surfaces are convex. The capitula are well developed.

palpable. In both girls flexion was from 40° to 140°, pronation was two-thirds, and supination was only one-third of normal. No joints in either child were hypermobile. The patellae were normal and there were no abnormalities of skin or nails.

X-rays showed bilateral anterior dislocation in both cases. In the elder child the radial epiphyses were incompletely modelled and were convex on their articular surface. The capitula appeared well-developed (Fig. 2). In the younger child neither the radial nor capitular epiphyses had appeared (Fig. 3).

Fig. 3. Radiographs of the elbows of the younger child showing anterior dislocation of the radial heads. The epiphyses have not yet appeared.

A review of the literature provides very few reports of the familial occurrence of congenital dislocation of the head of the radius. Abbott² reported seven cases of anterior dislocation occurring in one family over four generations. No other abnormalities were described. Cockshott and Omololu³ described posterior dislocations in a father and daughter. These were thought by McKussick⁴ to be part of the Ehlers-Danlos Syndrome. In the discussion of a paper by Fox and Griffin, Lambert⁵ mentioned that he had seen two sisters with the anomaly. It would appear that as an isolated familial lesion congenital dislocation of the head of the radius is very rare.

I am grateful to Mr. Malcolm Swann for permission to report his cases, which were also presented at a clinical meeting at the Royal Society of Medicine.

—I am, etc.,

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Ascot, Berks

¹ Almquist, E. E., Gordon, L. H., and Blue, A. I., *Journal of Bone and Joint Surgery*, 1969, **51A**, 1118.

² Abbott, F. C., *Lancet*, 1892, **1**, 800.

³ Cockshott, W. P., and Omololu, A., *Journal of Bone and Joint Surgery*, 1958, **40B**, 483.

⁴ McKussick, V. A., *Heritable Disorders of Connective Tissue*, 2nd edn., St. Louis, Mosby, 1960.

⁵ Lambert, R. G., *Journal of Bone and Joint Surgery*, 1960, **42A**, 530.