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 Edington[5]) 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. Adeloye 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. Thompson[6] 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. KONOTEY-AHULU

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1 Diggs, L. W., *American Journal of Clinical Pathology*, 1965, **44**, 1.
2 Edington, G. M., and Gilles, H. M., *Pathology in the Tropics*, p. 379, London. Arnold, 1969.
3 Vandepitte, J., and Delaisse, J., *Annales de la Société Belge de Medicine Tropicale*, 1957, **37**, 703.
4 Raper, A. B., *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1959, **53**, 110.
5 Edington, G. M., *British Medical Journal*, 1953, 2. 957.
6 Thompson, G. R., *British Medical Journal*, 1962, 1, 682.

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

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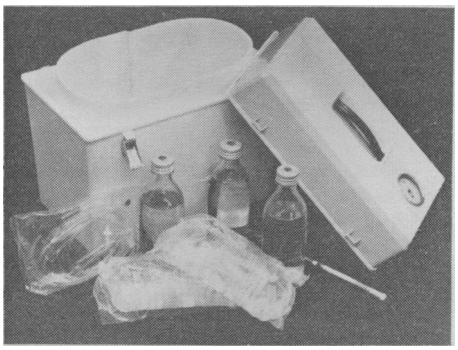
Insulating Kidney Perfusion Fluid

SIR,—As forecast by Calne[1] 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.,

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Accidental Infection of Man with Mycoplasma caviae

SIR,—*M. caviae* is a species of mycoplasma only recently characterized[1] 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