"There is no such thing as tropical medicine." Often have we heard that there should be no such thing as "western medicine," "Russian medicine," or "Chinese medicine." Of course he is right: medicine is one, the whole is always greater than the part. Yet in practice we cannot manage without words that set limits to that part with which we happen to be concerned. Tropical medicine has long been a convenient if illogical term. But if, in accordance with the implications of the penultimate paragraph of Dr. Shattock's letter, it were to be replaced by terms suitable for the particular needs of every country we could finally have as many types of medicine as there are members of the United Nations, which is absurd. Tell me someone, assuming as I do that we need a geographical classification of medicine, what it should be.—I am, etc.,

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Maintenance Therapy in Myeloma: Risk versus Benefit

SIR,—The letter by Drs. Susan M. Sieber and R. H. Adamson (7 June, p. 557) drawing attention to the risks of maintenance therapy in myeloma emphasizes the current dilemma attending the treatment of patients with this disease. We have also recognized this complication[1] and, on the basis of some 100 new patients with myeloma admitted to the clinic in the past two years, we have become concerned about the whole approach to therapy.[2] Undoubtedly current treatment schedules have prolonged median survival, but we question the continued use of alkylating agents in the maintenance period when the labelling index has risen in response to treatment.[3] We have suggested that alternative chemotherapeutic regimens be employed at this time in which cycle-specific drugs are administered in an attempt to capitalize on the altered cell kinetics.[4] Acknowledging that these proposed programmes will impose a heavy load on medical staff, require additional sophisticated haematological support, and have at the present time a largely theoretical basis, we have mounted a prospective study to test this hypothesis. We would be interested to hear how other investigators currently view this therapeutic dilemma.—We are, etc.,

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1 Dubovsky, D., and Jacobs, P., *Lancet*, 1974, **1**, 1113.
2 Jacobs, P., and Dubovsky, D., *British Medical Journal*, 1975, **1**, 625.
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4 Jacobs, P., Dubovsky, D., and King, H. S., *South African Medical Journal*, 1975, **49**, 650.

False Positive Pregnancy Test in Uraemia

SIR,—Following our report (16 November 1974, p. 410) of two patients suffering from uraemia who had false positive pregnancy tests, a study of the incidence of false positive pregnancy tests in 120 uraemic patients with the Gravindex method has been completed with the following results.

(1) False positive pregnancy tests occurred in three out of 60 patients (5%) suffering from chronic renal failure (two women and one man). On haemodialysis the false positive pregnancy test reverted to normal within a period of three weeks. The false positive pregnancy test was unrelated to the degree of albuminuria, since the three patients with a positive pregnancy test only had a trace of albuminuria. (2) In 60 patients suffering from acute renal failure a false positive pregnancy test was not observed.

I suggest that a false positive pregnancy test does not occur in acute renal failure because a disturbance in the immunological state may require a period of time to develop. If a false positive pregnancy test does occur in uraemia the diagnosis would be compatible with chronic renal failure rather than acute renal failure.—I am, etc.,

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Cot Deaths in Sweden

SIR,—Several investigations have shown a rather high incidence of sudden unexpected deaths in infancy in Britain, Canada, the United States, and many other countries. Figures between 2 and 3 per 1000 live births are commonly given and for certain places and certain strata of population even higher rates have been mentioned. As a consequence, much research has been done, and is still going on, in pathology, physiology, immunology, virology, and other fields to try to reveal the cause or causes of these deaths but, as yet, with no conclusive results.

Recently, Emery and his co-workers[1-3] have brought some evidence for the belief that certain cases of sudden unexpected death in infants are due to factors which might be prevented by rather simple measures such as intensified health supervision of an empirically defined high-risk group of children and advice to their mothers on feeding and care of the babies. During the last two years, while these measures were taken, the incidence in Sheffield of sudden unexplained deaths in infants has dropped by about half.[4] In Sweden and in some other countries (Czechoslovakia, Israel, the Netherlands) a much lower incidence of sudden unexpected death has been found in infancy. For the period 1968-72 rates between 0·4 and 0·8 per 1000 live births are reported from different parts of Sweden.[5] During these years the total infant mortality after the first week of life was only between 3·7 and 2·9 per 1000 live born in Sweden. This fact supports the belief that the Swedish rate of sudden infant deaths must really be much lower than the British.

Two questions seem to arise from these facts. (1) Is it justified to think that the so much lower rate of cot deaths in Sweden may be due partly to a more intense child health service which has managed to reach also most babies in the high-risk group defined by Emery? In Sweden virtually all children are followed by the child health service at least during their first year of life. Out of 112 273 babies born in 1972, 110 467 were supervised and had an average of 11·7 contacts with the doctor or nurse of the service during the first year. The rate of babies being entirely breast-fed for at least two months was still 30·7% that year. (2) Is the group of babies who still die suddenly and unexpectedly in Sweden, or their families, in any respect different from the corresponding group in Britain? In other words, are there two (or more) types of sudden infant death syndrome, one preventable by better care and feeding (plus, probably, certain socioeconomic factors), the other not preventable by these means? In our opinion, answers to these questions would be of great value.—We are, etc.,

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1 Protestos, C. D., *Archives of Disease in Childhood*, 1973, **48**, 835.
2 Emery, J. L., and Carpenter, R. G., *Proceedings of the F.E. Camps International Symposium on the Sudden Infant Death Syndrome*, p. 97. Toronto, Canada, 1974.
3 McWeeny, P. M., and Emery, J. L., *Archives of Disease in Childhood*, 1975, **50**, 191.
4 Emery, J. L. Personal communication.
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Glibenclamide-induced Hypoglycaemia

SIR,—We would like to draw attention to the dangers of accidental ingestion of the hypoglycaemic agent glibenclamide. Cases of poisoning with glibenclamide have been reported but complete recovery seems to have occurred.[1] In the following case of a patient who was admitted to hospital after accidentally taking glibenclamide sequelae still persisted 12 months after ingestion.

A 30-month-old, previously normal child was admitted 48 hours after ingesting an unknown but small number of 5-mg tablets. Since taking them he had been drowsy and delirious and had had screaming attacks and difficulty in feeding. On admission he was pale, limp, perspiring freely, and had a generalized convulsion with right focal features. The Dextrostix test was non-reactive for blood glucose. He was promptly given a bolus of 50% dextrose solution intravenously and a slow intravenous infusion of 10% dextrose solution. He did not recover consciousness after the convulsion and two hours after starting therapy his blood glucose was 0·38 mmol/l (7 mg/100 ml) and cerebrospinal fluid glucose 1·38-2·77 mmol/l (25-50 mg/100 ml). Further 50% dextrose was given statim. Ten hours later he was more deeply unconscious. His pupils were dilated and fixed and the plantar responses extensor, though he responded to painful stimuli. There was continual multifocal twitching. Though the fundi appeared within normal limits his deterioration was believed to be due to cerebral oedema, and intravenous dexamethasone, mannitol, and glycerol were given sequentially. His conscious state improved but fluctuated over the next 48 hours. After 72 hours he was conscious but very irritable. Plasma insulin was estimated daily for several days from the third day after admission. The values were not raised and were appropriate for the simultaneously measured plasma glucose levels. Blood samples were not available for plasma insulin before the third day.

When discharged 12 days after admission the patient had a persisting left third nerve palsy but he was alert and happy. There had been no attacks of drowsiness or twitching for seven days. He was maintained on anticonvulsant therapy. When last seen 12 months later the left third nerve palsy was still present, there seemed to be a diminution of visual acuity in the left eye, and the left optic disc appeared paler than the right. He was dyslalic and his vocabulary was limited for his age, but his parents thought his comprehension was normal. He had had a number of epileptic seizures, a few grand mal but mainly minor motor seizures, including akinetic seizures. Plasma glucose estimations performed at these times had been within normal limits.

The hypoglycaemic reaction to glibenclamide in adults has been reported as not