CORRESPONDENCE

Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Malnutrition in the Third World

SIR,—As an organisation much concerned recently in the famine relief operation in Africa we read with interest Professor A Stewart Truswell's article (24 August, p 525). We would like to share our observations and experiences.

The priority in ensuring that the population will not deteriorate further is to provide 2000-2100 kcal (8·4-8·8 MJ) daily per head as a basic ration. This calculation is an average based on the requirements of differing age groups: a child of 4 needs 17-1800 (7·1-7·5 MJ), a young man of 19 maybe 2900 (12·1 MJ). For ease of administration and to prevent envy the rations should be seen to be the same for all. They would consist of cereal, legumes (beans or peas), and oil. A few grams of salt or spice is recommended. The distribution of milk powder is discouraged because the conditions for safe reconstitution at home do not exist. Milk is used in therapeutic feeding for severely malnourished children based on the high energy milk recommended by Oxfam.[1]

A further supplement of the same types of basic food equalling 350 kcal/day (1·5 MJ) is given to those who are growing—that is, the under 5s, breastfeeding women, and those in the last trimester of pregnancy.

Multivitamins are not used but rather the specific vitamin for an identified nutritional problem. Borderline reserves of vitamin A are used up when malnourished children begin eating. Infectious diseases are often exacerbated as the child's nutritional status improves, further depleting stocks. Because the sequelae are so serious if there is any evidence of vitamin A deficiency in a community all the children receive prophylaxis. In the Sahel scurvy has been a problem owing to nomads losing their camels' milk, which is a good source of vitamin C. In our experience anaemia is a serious problem compounded by malaria and hookworm.

While appearing to address famine, the article actually deals with individuals, particularly children with severe malnutrition. More lives are likely to be saved with measles vaccination and adequate early treatment for diarrhoea. As both of these interventions also have considerable nutritional benefit we would direct our resources to this end.

Doctors concerned in famine relief operations should have a management/organisational role, determining and guiding a locally appropriate programme aimed at lowering the mortality and morbidity of the three or four most common conditions to the level usual for that country. This is after ensuring adequate food and water. Focusing on the treatment of a few individuals often means that the major, often preventable, health problems of the majority are ignored. Therapeutic feeding, which requires individual medical supervision, considerable time, and inpatient facilities, is not an effective use of resources, especially as the results are not encouraging. If it is done we would emphasise, however, that energy density is important along with an organisation which assures six to eight feeds per child in 24 hours. Potassium replacement is easily done by adding oral rehydration salts to the high energy milk mixture.

The mid-upper arm circumference is used for rapid screening only and the cut off point we use at present is 12 cm. This is under review and will be discussed with other agencies. The use of light 850 g hanging scales encourages the more accurate weight for height survey to be done. Weight for height surveys of children aged under 5 (or 115 cm) give an indication of the nutritional status of the community. But other information must also be obtained if the intervention is to suit the problem. For example, there must be an awareness of the normal loss of weight which occurs just before the harvest[2]—the hungry season—or knowledge of recent epidemics such as measles or recent migration due to civil unrest.

Food aid can have negative effects. Donations of food may depress local prices so much that the next year's harvest has no market value. In countries where the rainfall is always borderline communities have developed ways of coping with the lean years, and shortsighted "solutions" can destroy this balance. A central distribution, convenient from the relief agencies' point of view, may create a pull effect. This encourages people to leave the shelter of their villages and congregate in large numbers and thereby increases the transmission of infectious diseases due to overcrowding and pollution of the environment. The men may be so far from their villages that they miss the rains and so do not plant in time.

The five measures Professor Truswell mentions for preventing protein energy malnutrition are aimed at child health in general. Of these five, the early and adequate treatment of diarrhoea (rather more than the use of oral rehydration solution only), breast feeding and adequate weaning, and immunisation against measles are among the most critical of the interventions that may be made in famine relief operations.

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1 Oxfam. *Practical guide to selective feeding programmes*. Oxford: Oxfam, 1984.
2 Loutan L, Lamotte JM. Seasonal variations in nutrition among a group of nomadic pastoralists in Niger. *Lancet* 1984;i:945-7.

SIR,—Professor A Stewart Truswell (31 August, p 587) describes the problem of vitamin A deficiency endemic in east Asia and certain parts of Africa and Central and South America in association with protein malnutrition.

He states that the last reported case of xerophthalmia in Britain was in 1938. It is wrong to

suppose that vitamin A deficiency is no longer encountered in this country. Although it is rarely encountered as a result of protein malnutrition, as in developing countries, it is certainly seen.

Nyctalopia, the less severe form of vitamin A deficiency, can occur in malabsorption syndromes such as short bowel syndrome in Crohn's disease[1] and after intestinal bypass surgery for obesity.[2] It has also been described in patients with primary biliary cirrhosis.[3]

There have been three cases of dietary induced keratomalacia reported. In the United States Gombos *et al* described a patient who voluntarily eliminated all fresh fruit and vegetables from her diet to try to reduce her exacerbations of ulcerative colitis.[4] In Britain Bohrs and Fells described a young man who deliberately omitted all foods containing vitamin A from his diet in the belief that this would prevent his grand mal epilepsy.[5] I have described a case of bilateral keratomalacia in a strict vegan who adhered to a diet of pulses which was totally devoid of dairy produce, vegetables, and fruit.[6]

Because vitamin A deficiency does occur in Britain in people with malabsorption, severe liver disease, and unusual diets clinicians should be aware that patients may present with it.

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1 Main ANH, Mills PR, Russell RI, *et al.* Vitamin A deficiency in Crohn's disease. *Gut* 1983;**24**:1169-75.
2 Brown GC, Felton SM, Benson WE. Reversible night blindness with intestinal by-pass surgery. *Am J Ophthalmol* 1983;**89**: 776-9.
3 Walt PR, Kemp CM, Lyness L, Bird AC, Sherlock S. Vitamin A treatment for night blindness in primary biliary cirrhosis. *Br Med J* 1984;**288**:1030-1.
4 Gombos GM, Hornblass A, Vendeland J. Ocular manifestations of vitamin A deficiency. *Ann Ophthalmol* 1970;**2**:680-4.
5 Bohrs F, Fells P. Reversal of complications of self-induced vitamin A deficiency. *Br J Ophthalmol* 1971;**55**:210-4.
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Timing of lumbar puncture in severe childhood meningitis

SIR,—I was very glad that you published Dr J R Harper's letter on the hazards of lumbar puncture in young children (7 September, p 651) together with the four replies showing an intriguing spectrum of opinion.

As a junior hospital paediatrician who has worked mainly in district general hospitals without local facilities for computed tomography or cerebral pressure monitoring I count myself extremely lucky not to have caused brain stem coning by the many lumbar punctures I have both done and sanctioned. A traditional teaching is that the lumbar puncture is safe if there is no papilloedema. This is patently untrue because cerebral oedema may occur acutely without papillodema. Unfortunately the signs of acute cerebral oedema (not only due to advanced meningitis but also, for instance, to Reye's syndrome or a space occupying lesion) are not easily distinguishable from those of meningitis, especially in the very young child.

In a centre with paediatric intensive care and neurosurgical facilities (such as where I now work) there is a tendency to criticise referring hospitals for inappropriate lumbar puncture, since the small proportion of patients who deteriorate rapidly have to be transferred and tend also to do badly. There is, however, no easy answer to the problem of when or when not to do a lumbar puncture in a sick young child, and it is important that the debate should be brought into the open. It would be helpful for those actually doing lumbar punctures to have guidelines on this, ideally from a body such as the British Paediatric Association, but the four responses you obtained to Dr Harper's letter suggest that consensus would be difficult to achieve.

QUENTIN SPENDER

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SIR,—Dr J R Harper describes a case of meningococcal septicaemia with meningitis in a 20 month old boy who died despite prompt treatment. It is more likely that this child had a cardiovascular collapse rather than a pressure cone. Acute meningococcaemia is often associated with cardiovascular collapse secondary to myocardial dysfunction[1]; this may in turn be due to a direct effect of endotoxin on myocardial performance.[2]

Furthermore, it is theoretically possible that antibiotics, especially bactericidal ones which cause bacterial death and disruption, could precipitate a massive endotoxinaemia.[3] This has been confirmed in very low birthweight babies with other Gram-negative infections (APJ Thomson, unpublished observations).

Methods for detecting endotoxin are now quantitative and more reliable,[4] and antiendotoxin treatment, such as plasmapheresis or anti-lipopolysaccharide antibody infusion, has been successfully used in meningococcal[5] and other infections.[6] Yet the mortality of meningococcal disease has remained unchanged for 20 years.[7]

We should be looking at endotoxin levels in meningococcal septicaemia and evaluating the immediate effects of antibiotic treatment. By avoiding massive endotoxinaemia bacteriostatic antibiotics such as chloramphenicol might paradoxically be more appropriate than bactericidal antibiotics in a fulminant case such as Dr Harper describes. Nevertheless, the sooner the antibiotic treatment is started, whether or not other investigations have been performed, the better the prognosis.

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1 Boucek MM, Boerth RC, Artman M, *et al.* Myocardial dysfunction in children with acute meningococcaemia. *J Pediatr* 1984;**105**:538-42.
2 Rao PS, Dahm CH, Ritter HA, *et al.* Impairment of myocardial performance in endotoxic shock. *J Mol Cell Cardiology* 1978;**10** (suppl 1):86.
3 Hopkin DAB. Frapper fort ou frapper doucement: a Gram-negative dilemma. *Lancet* 1978;ii:1193-4.
4 Harris R, Stone PCW, Stuart J. An improved chromogenic substrate endotoxin assay for clinical use. *J Clin Pathol* 1983;**36**: 1145-9.
5 Bjorvatn B, Bjertnaes L, Fadnes HO, *et al.* Meningococcal septicaemia treated with combined plasmapheresis and leucapheresis or with blood exchange. *Br Med J* 1984;**288**: 439-41.
6 Ziegler E, McCutchan J, Fierer J, *et al.* Treatment of Gram-negative bacteraemia and shock with human antiserum to a mutant Escherichia coli. *N Engl J Med* 1982;**307**:1225-30.
7 Slack J. Deaths from meningococcal infection in England and Wales in 1978. *J R Coll Phys Lond* 1982;**16**:40-4.

SIR,—Dr J R Harper and others discussed a very important subject. At the Red Cross Children's Hospital in Cape Town we have had a fair experience of meningitis in infancy and childhood and have offered the following guidelines to our junior and registrar paediatric staff.

The diagnosis of meningococcal septicaemia, which is often associated with meningitis, is made when the central nervous system, the circulatory system, and the skin or mucous membranes are affected simultaneously. The child presents with confusion, stupor, or coma. Meningism is present, as also is a tense fontanelle. Lumbar puncture is contraindicated because it may precipitate coning from the cerebral oedema that is invariably present. A positive culture from the blood or a Gram stain of an imprint smear taken after scraping a typical skin infarct will show intracellular Gram negative diplococci.

An infant or child presenting with suspected meningitis, whose level of consciousness is severely affected or who is in status epilepticus, should not undergo lumbar puncture at the onset. In these cases there is cerebral oedema and lumbar puncture may precipitate coning. The absence of papilloedema does not exclude cerebral oedema. Early death may be from cerebral oedema not meningitis. Mannitol intravenously or steroids (dexamethasone) should be given before lumbar puncture. Again a blood culture may be positive.

We have learnt from necropsy studies the hazards of performing a lumbar puncture in any patient admitted with suspected meningitis.

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SIR,—One week ago we had a similar experience to that of Dr J R Harper with a 16 month old boy. Over 24 hours he had become progressively more drowsy and disinclined to be handled. On admission he was very unwell with a rectal temperature of 39·2°C, neck stiffness, and a positive Kernig's sign. There were no petechiae. The initial impression was of a definite bacterial meningitis. We followed the conventional practice of carrying out a diagnostic lumbar puncture, and 2 ml of turbid cerebrospinal fluid was removed. Microscopy confirmed the diagnosis of bacterial meningitis with numerous Gram negative coccobacilli seen. Treatment with intravenous penicillin and chloramphenicol was started.

The child's condition remained unchanged until 12 hours after the lumbar puncture, when he had a respiratory arrest. Resuscitation was prompt, but afterwards the child had fixed dilated pupils and no spontaneous respiration. We started mechanical ventilation but stopped this after confirming brain stem death 48 hours later.

Blood cultures grew *Haemophilus influenzae*. Postmortem examination confirmed haemophilus meningitis with coning.

This child's death may have been a consequence of the lumbar puncture. Conventional teaching has been that in the presence of bacterial meningitis there should be no risk from lumbar puncture. Is this really true? Lorber and Sunderland mention three children who died with cerebellar coning after lumbar puncture.[1] In cases in which the diagnosis of meningitis is in doubt lumbar puncture can be justified as a diagnostic exercise. When the diagnosis is certain on clinical grounds, are we putting our patients at unnecessary risk by carrying out an investigation unlikely to alter the clinical management?

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1 Lorber J, Sunderland R. Lumbar puncture in children with convulsions associated with fever. *Lancet* 1980;i:785-6.

SIR,—We have seen three children with meningococcal septicaemia who suffered a respiratory arrest several hours after lumbar puncture. None had any pre-existing clinical evidence of increased intracranial pressure before lumbar puncture. All three children were resuscitated after presumed coning but died after several days' respiratory support. Two children underwent necropsy and coning was confirmed as the cause of death in both. Meningococcus was isolated from blood cultures in all three cases.

Although it cannot be proved that coning would not have occurred had no lumbar puncture been