a dosage of up to 400 mg daily. This drug is used for its muscle-relaxing properties in tetanus, but it was first introduced into medical practice as a vermifuge. The worm absorbs the drug but, unlike its host, is unable to metabolize or excrete it. The anthelmintic property may be related to an anticholinesterase action which upsets the neuromuscular apparatus of the worm or alternatively to an effect on redox systems. These mechanisms might be expected to paralyse the worm, but our experience is that they retain their motility on expulsion in cases of tetanus.

It would be helpful if further light could be thrown on this problem.—We are, etc.,

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1 Smith, M. H., *Nature*, 1969, 223, 1129.

Was it a Drug?—Forceval Protein

SIR,—Your report (*Supplement*, 1 June, p. 86) has once again drawn my attention to the recommendation of the Clayton Committee that Forceval Protein should be regarded as a food except "when prescribed for biochemically proven hypoproteinaemia."¹

As I have previously pointed out,² hypoproteinaemia occurs (a) as a result of defective manufacture, as in liver disease, or (b) as a result of excessive loss, as in nephrotic syndrome or carcinoma. It does not occur in simple starvation. No amount of feeding of protein by mouth will affect hypoproteinaemia unless the cause can be remedied. If it is remedied, then the body will once again retain protein from the diet and regain its nutritional protein status and the blood protein level will rise. It follows that any suggestion that Forceval Protein or any other protein given by mouth is not really a food but an endeavour to treat hypoproteinaemia cannot be acceptable. I now challenge any member of the Clayton Committee to justify its recommendation. If nobody does this time then it must be regarded as discredited and local medical committees should disregard it and use their own criteria.

Quite apart from this, the Clayton Committee perpetuates recommendations under which children (and indeed adults) born with some diseases may get nutrition free on N.H.S. prescription while those with other diseases may not. This still frequently leaves the prescribing practitioner wondering whether he may or may not prescribe what he thinks necessary for his patient's treatment and surely defeats the whole purpose of the Clayton Committee and all its predecessors.

It is my contention that all substances given by mouth which provide nothing but nutrition (even if it is a specially modified form of nutrition) are foods. If the patient is not expected to pay for them then some other means of prescribing or providing them should be instituted. We must put some limit to the intolerable situation whereby a third party sitting behind a desk can challenge what a doctor may or may not prescribe for his patient. The fact that the hospital doctor in outpatients is apparently free from this kind of control only adds to

the general practitioner's frustration.—I am, etc.,

A. LEWIS

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1 *MIMS (Monthly Index of Medical Specialties)*, 1974, no. 6, p. 207.
2 Lewis, A. A., *British Medical Journal*, 1972, 3, 588.

Stilboestrol for Prostatic Cancer

SIR,—The value of anonymous medical articles is always questionable especially when broad generalizations are made as in your leading article "Stilboestrol for Prostatic Cancer" (8 June, p. 520). The salutary findings of the Veterans Administration Cooperative Urological Research group in 1967¹ clearly suggest that oestrogens double the death rate in prostatic cancer by their fluid-retaining and thrombogenic properties. The conclusion reached was that they should be used only for urinary obstruction or bone pain. Since the treatment is for symptoms then it is essential to define precisely what symptoms are an indication, which you singularly fail to do.

Similarly, if the subsequent work of the V.A.C.U.R.G. in 1973² states that lower doses are safer while being effective in relieving symptoms (which you appear to accept) then it is hardly rational to recommend using larger doses than theirs on purely speculative theoretical grounds.

It is disturbing that Britain's most influential medical journal should dissipate the hard-won knowledge it seeks to disseminate.—I am, etc.,

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1 Veterans Administration Cooperative Urological Research Group, *Surgery, Gynecology and Obstetrics*, 1967, 124, 1011.

2 Byar, D. P., *Cancer*, 1973, 32, 1126.

Lactose Intolerance in San Populations

SIR,—The paper by Dr. T. Jenkins and others (6 April, p. 23) raises some interesting points. However, we feel it necessary to point out some flaws in the claim that 90% of !Kung Bushmen are lactose-intolerant.

The authors have confused lactose intolerance with lactose malabsorption. As they point out, none of their sample had symptoms following ingestion of 50 g of lactose, though blood glucose levels did not rise significantly in 39 of the 40 subjects. Thus a high incidence of lactose malabsorption and not lactose intolerance has been shown.

The fears concerning the possibility of the introduction of milk powder as a form of dietary supplementation are likewise unfounded. Milk powder is used mainly as a food supplement in the paediatric population, particularly in the age group that has just been weaned and at risk from protein energy malnutrition. This group as a whole should have adequate lactase levels in the small-bowel mucosa. It has also been shown in patients with kwashiorkor that the low lactase levels do not necessarily correlate with lactose intolerance but rather with lactose malabsorption.¹ It is unlikely in adults that anywhere near 1½ litres of reconstituted cow's milk a day (equivalent to 50 g lac-

tose) would be consumed. Thus if lactose intolerance did not occur with the lactose tolerance test it is doubtful if it will occur with milk supplementation.

Too much importance has been placed on a flat lactose tolerance curve. Milk remains a very good source of protein and calories, and therefore should not be discarded because of an abnormal blood result, but rather only if a high incidence of lactose intolerance in a population group is shown. This was not demonstrated in the sample studied.

Another contentious point is the authors' comment that the !Kung Bushmen tested were not undernourished. Yet a survey done on a similar group a few miles away had definite evidence of undernutrition.² In the presence of chronic undernutrition and lack of exposure to milk after weaning from the breast, can a genetic cause for the lactose malabsorption justifiably be postulated in the !Kung Bushmen on present evidence?—We are, etc.,

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1 Prinsloo, J. G., et al., *Archives of Disease in Childhood*, 1971, 46, 474.

2 Truswell, A. S., et al., *American Heart Journal*, 1972, 84, 5.

Treatment of Vulval Candidiasis with 5-Fluorocytosine

SIR,—Fungicidal agents effective in the treatment of systemic candidiasis are little used for localized vulval infections because of the dangers of toxic effects of therapy and the apparently trivial nature of the disease. Nevertheless, there is a place for such treatment in severely affected women who have proved resistant to conventional methods.

A patient aged 42 had a 20-year history of vulval candidiasis varying in intensity and often disabling, with intense irritation. Numerous courses of treatment, including local and oral nystatin, gave only temporary relief. She was a diabetic, well controlled on insulin. The vulva had an inflamed, macerated appearance and swabs from this area grew *Candida albicans* sensitive to 5-fluorocytosine (5-FC) in a concentration of 0.2 µg/ml.

She was admitted for treatment, and, renal function proving normal, was given 2 g of 5-FC six hourly for 12½ days together with 5-FC cream locally. Blood 5-FC levels were 6 µg/ml three hours after starting treatment and 20 and 32 µg/ml on subsequent occasions. There was a dramatic subjective improvement within 18 hours of starting treatment and complete freedom from vulval symptoms within three days. She developed transient abdominal cramp and mild diarrhoea which lasted for one month. Following treatment she felt more fit than she had for many years and has remained symptom-free, with negative cultures, for nine months.

Before undertaking treatment with 5-FC it is essential to make sure that the yeast is a sensitive strain, and an adequate blood level will lessen the likelihood of secondary resistance developing.

Gastrointestinal upsets occur in 5-10% of cases; laboratory abnormalities include increased serum transaminases, alkaline phosphatase, and blood urea and pancytopenia. These have mainly been found in severely ill patients and may reflect underlying infection or treatment with drugs such as immunosuppressive agents rather than drug toxicity.

Such side effects rule out the use of 5-FC for vulval candidiasis except where this is disabling. Such cases do, however, occur, and the patient described above has been transformed from an anxious, depressed woman to a happy housewife, all her family sharing the benefits.

The 5-PC was provided by Dr. J. Garrod of Roche Products Ltd. Microbiological investigations were performed by Dr. A. T. Willis and blood 5-FC levels estimated at the Mycology Reference Laboratory.

—I am, etc.,

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Cot Deaths

SIR,—In your leading article (2 March, p. 341) on cot deaths, also referred to as the sudden infant death syndrome (S.I.D.S.), it was stated that "post-mortem examination of infants with S.I.D.S. has shown an adequate cause of death, such as pneumonia, acute bronchiolitis, or gastroenteritis in 25% to 50% of cases." I feel that it would have been more appropriate to define S.I.D.S. precisely in your article and adhere to the definition throughout the course of your discussion. Thus both semantic confusion and the inflation of the incidence of S.I.D.S. by 25-50% would have been avoided.

At present a widely recognized definition of S.I.D.S. is "the sudden death of an infant or young child, unexpected by history, in which a thorough postmortem examination fails to demonstrate an adequate cause of death."¹ Prior to 1970 to most investigators S.I.D.S. was the death of an infant or child who was thought to be in good health or whose terminal illness proved to be so mild that the possibility of a fatal outcome was unexpected. However, unless we adhere to a generally acceptable definition, studies on S.I.D.S. either to evaluate its true incidence or to elucidate the cause and/or mechanism of death are bound to have a limited significance.—I am, etc.,

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¹ *Sudden Infant Death Syndrome: Proceedings of the Second International Conference on Causes of Sudden Death in Infants*, ed. A. B. Bergman, J. B. Beckwith, and C. G. Ray. Seattle, University of Washington Press, 1970.

Meningioma Mimicking Features of a Pheochromocytoma

SIR,—Drs. R. Gabriel and B. D. Harrison (11 May, p. 312) report a case of supratentorial meningioma mimicking the clinical and biochemical features of pheochromocytoma in which the raised vanilmandelic acid (VMA) excretion returned to normal after removal of the tumour. The explanation offered, that raised intracranial pressure had produced these features through stimulation of the sympatheticoadrenal pathway, is not the only possible one and not perhaps the most likely.

Since the time of Cushing¹ vasomotor changes have been accepted as part of the clinical picture of raised intracranial pressure; such changes occur usually only with a rapid pressure rise² and then in-constantly and mainly in children with

lesions that directly compromise the posterior fossa.³⁻⁵ In the case described no mention is made of papilloedema and the lumbar cerebrospinal fluid pressure was only 270 mm.

The association of pheochromocytomas and meningiomas with von Recklinghausen's disease is well accepted, the link probably lying in the common neural-crest ancestors of these tumours. Other neural tumours have been reported as secreting VMA.⁶ Is it not probable that the meningioma may on occasion have this capacity? After all, other intracranial tumours, the haemangioblastomas, secrete hormones and the latter is found in association with pheochromocytomas. It would have been of interest to know the cytochemical characteristics of this patient's tumour.—I am, etc.,

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- 1 Cushing, H., *American Journal of Medical Sciences*, 1902, 124, 376.
- 2 Pickering, G. W., *High Blood Pressure*, p. 520, 2nd edn. New York, Grune and Stratton, 1968.
- 3 Browder, J., and Meyers, R., *Archives of Surgery*, 1938, 36, 1.
- 4 Johnston, I. H., Johnston, J. A., and Jennett, B., *Lancet*, 1970, 2, 433.
- 5 Plum, F., and Posner, J. B., *Diagnosis of Stupor and Coma*, 2nd edn., p. 32. Philadelphia, David, 1972.
- 6 Kogut, M. D., and Kaplan, S. A., *Journal of Pediatrics*, 1962, 60, 694.

The Heart during Sleep

SIR,—With reference to your leading article (13 April, p. 70) in the beginning of open-heart surgery in the 1950s it was recognized from first principles and practical experience that pain, fear, and exhaustion predisposed to death from arrhythmia, and the protective value of providing adequate sleep was quickly appreciated.

In the early 1960s coronary care units appeared and some of us introduced, and continued to find useful in threatened and acute myocardial infarction,¹⁻⁴ the sleep regimens that we had developed in the surgical wards.—I am, etc.,

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- 1 Nixon, P. G. F., *et al.*, *Lancet*, 1968, 1, 726.
- 2 Nixon, P. G. F., in *Recent Advances in Medicine*, ed. D. N. Baron, N. Compston, and A. M. Dawson. London, Churchill, 1968.
- 3 Nixon, P. G. F., in *Seventh Symposium on Advanced Medicine*, ed. I. A. D. Bouchier. London, Pitman Medical, 1971.
- 4 Nixon, P. G. F., *Practitioner*, 1973, 211, 5.

Blind Babies

SIR,—I was pleased to read your leading article (8 June, p. 518) on xerophthalmia, the blinding disease due basically to a lack of vitamin A in the food. It is a scourge in many countries yet little is taught about it in medical schools in the West. This is particularly unfortunate at present as preventive schemes are starting in many parts of the world and accurate diagnosis is essential. The Governments of India, Indonesia, and Bangladesh are distributing, each six months, a large dose of vitamin A (300,000 IU) in oil by mouth to children under 5 years old in the hope that this will maintain a satisfactory level of vitamin A in the liver and blood. The results of these measures are not yet available, but one hopes that the impact will be great and that the stream of malnourished children with xerophthalmia

coming daily to paediatric and ophthalmic outpatient clinics will be reduced.

However, such children, who are the most at risk of going blind, can be effectively treated at a relatively small cost. At Madurai in Southern India, Dr. Venkataswamy directs a nutrition centre for children suffering from malnutrition and xerophthalmia where the child and parent (usually mother) are given food which provides vitamin A and which is both local and cheap. In practice this means provision of about an ounce (28 g) a day of well-boiled and mashed carrots or dark green leafy vegetables such as spinach, amaranth, drumstick leaves, or turnip tops. The rest of the food is designed to supply a good level of protein and calories. The parents do the marketing and cooking and are encouraged to continue the food when they go home. The results have been sufficiently encouraging for the work to be extended into surrounding villages.

The Xerophthalmia Club exists to help preventive measures. A current activity is an attempt to encourage governments and charitable bodies which send dried skim milk to disaster areas to ensure that all such milk is fortified with vitamin A. If protein food is given to a malnourished child already deficient in vitamin A the last remnants of vitamin A in the body are used up and xerophthalmia may be precipitated. Such disasters still occur.¹ Unicef and the Governments of the United States and the Netherlands have already agreed that all dried skim milk exported shall contain vitamin A. We hope this will become universal.

The Xerophthalmia Club is financed entirely by the Royal Commonwealth Society for the Blind. There is no membership subscription for those seriously interested in the prevention of xerophthalmia.—I am, etc.,

ANTOINETTE PIRIE

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Girton College, Cambridge

¹ Oomen, H. A. P. C., *Xerophthalmia Club Bulletin*, No. 5, 1974.

Forensic Neuropsychiatry

SIR,—I disagree with some of Dr. M. F. Cleary's comments in his Personal View (1 June, p. 498) about the psychiatrist in court. Of course psychiatry is a woolly subject, but it need not be quite as woolly as some psychiatric witnesses make it seem.

As Cuthbert¹ has pointed out, serious crime may be a manifestation of brain disease, especially when the offence is not readily "understandable." In this situation it is not enough to order a neurological examination and E.E.G., both of which may give entirely normal results in the presence of significant disease.^{2,3} I believe it is essential, if justice is to be done, to offer a thorough neuropsychiatric investigation. In practice this will usually require a brain scan, air encephalography, psychometry, and in some cases a sphenoidal E.E.G. as well. In this way, significant brain disease is unlikely to be overlooked. Indeed, there is no other way of assessing the cerebral atrophy which frequently accompanies alcoholism,² and the relationship between crime and alcoholism is well known. Some tentative diagnoses of temporal lobe epilepsy are clinched when the air encephalogram shows marked temporal atrophy.³ Retrospective diagnosis,