rates might have been expected on the basis of the distribution of maternal age, parity, and ethnic group in mothers of different blood groups in the Jerusalem population.

TABLE I—Recorded Frequency of Toxaemia by Blood Groups

Blood Group	No. of Births	Pre-eclampsia (%)
Rh + A	3,696	1.70
B	2,101	1.91
AB	866	1.73
O	3,431	1.14*
Rh - A	388	0.59
B	250	2.40
AB	85	
O	363	3.03*
Total	11,180	1.58

In an earlier case-control study⁶ in which patients with toxaemia, defined by standard criteria, were matched with mothers of similar age, parity, and ethnic group there were no significant blood group differences between patients and controls (table II). Nor did the few cases of frank eclampsia show any unusual blood-group distribution. We are therefore unable to confirm any association between toxaemia and blood group A.

TABLE II—Blood Group Distribution of Patients with Eclampsia and Pre-eclampsia and Controls

Blood Group	Patients	Controls
Rh + A	39	38
B	16	25
AB	5	7
O	45	40
Rh - A	3	2
B	3	2
AB	1	0
O	1	1
Unknown	7	4
Total	121	121

It is not unlikely that in Britain, too, women of different blood groups differ in respect of ethnic background, social class, age, parity, and religion. Women delivered in particular hospitals may also provide a very biased population sample. Since the distribution of these variables affects the incidence of pre-eclampsia it would be important to take them into account in studying the relationship of the disease to blood groups.—We are, etc.,

SUSAN HARLAP
A. MICHAEL DAVIES

Department of Medical Ecology,
Hebrew University-Hadassah Medical School,
Jerusalem

- 1 Page, E. W., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1972, 79, 883.
- 2 Jack, H., et al., *Lancet*, 1969, 1, 539.
- 3 Mourant, A. E., Kopec, A. C., and Domaniewski-Sobczak, K., *Lancet*, 1971, 1, 223.
- 4 Sterk, V. V., et al., *Israel Journal of Medical Sciences*, 1971, 7, 1248.
- 5 Davies, A. M., et al., *Israel Journal of Medical Sciences*, 1969, 5, 1095.
- 6 Davies, A. M., et al., *Israel Journal of Medical Sciences*, 1970, 6, 253.
- 7 Davies, A. M., *Geographical Epidemiology of the Toxemias of Pregnancy*. Springfield, Thomas, 1971.

Coxsackie Viruses and Diabetes Mellitus

SIR,—Dr. D. R. Gamble and his colleagues (3 November 1973, p. 260) have presented evidence associating infection with Coxsackie B4 virus and the onset of insulin-dependent

Admission and Convalescent Antibody Titres Shown Side by Side for Coxsackie B1—B5 and Mumps

Case No.	Age (Yrs)	Sex	Month of Presentation	Coxsackie B1	Coxsackie B2	Coxsackie B3	Coxsackie B4	Coxsackie B5	Mumps
1	13	M	January	—	—	—	—	—	—
2	5	M	January	—	—	—	—	—	—
3	5	M	February	—	—	—	—	—	—
4	6	F	July	—	—	—	—	—	—
5	12	F	August	—	—	—	—	—	—
6	5	F	September	—	—	—	—	—	1/4 1/16
7	9	M	October	—	—	—	—	—	—
8	11	F	October	—	—	1/64 1/64	—	—	—
9	11	M	November	—	—	—	—	—	1/16 1/16
10	13	M	December	—	1/64 1/64	1/16 1/16	—	—	—
11	7	F	December	—	1/16 1/16	—	—	—	—

diabetes in young adults. However, as they point out, among the patients aged 9 years or less there was no significant difference in the incidence of neutralizing antibodies to Coxsackie type B4 when compared with a control group of normal children. They suggest that other viruses may possibly initiate diabetes in childhood.

We have looked for evidence of recent virus infection in 11 children who presented with diabetes over the past year. The mean duration of symptoms for this group of patients was seven weeks; no child had a history suggestive of a premonitory illness in the preceding three months. Sera were taken for virological studies at the time of the acute admission and subsequently at an outpatient visit. The mean time interval between the two samples was 6.8 weeks. The age, sex, and month of presentation of the cases are shown in the table, together with the results of the tests for neutralizing antibodies to Coxsackie B group viruses 1-5 and complement fixing antibodies to mumps.

The sera were screened at a dilution of 1/16 for the Coxsackie B group antibodies and any positive at this titre was titrated. Eight of the patients were negative at this dilution. Cases 10 and 11 had a titre of 1/16 or greater for Coxsackie B2 and cases 8 and 10 for Coxsackie B3, but in no child was there a rise in titre to support a diagnosis of recent infection.

In one of the two cases with demonstrable mumps antibodies (case 6) there was a rise in the S antibody titre indicative of very recent mumps. This might be considered a normal occurrence among a group of 11 children.

No significant titre or four-fold or greater rise in titre was found in complement fixation tests for Q fever, psittacosis/ornithosis, *Mycoplasma pneumoniae*, influenza A or B, parainfluenza, or infection with adenoviruses, respiratory syncytial virus, cytomegalovirus, herpes simplex, and varicella. Most children had a high titre of antibody to measles virus, as might be expected at their age.

Tissue cultures were inoculated with throat swab extracts from seven children; and tissue cultures and newborn mice with stool specimens from four children, collected when the children were first admitted to hospital. A rhinovirus was isolated from one child and adenovirus type 7 from the stool of the same child; all other specimens were negative on tissue culture and in mice.

We conclude from these observations that Coxsackie B4 virus played no aetiological role in diabetes in these 11 children and none of the other agents or viruses in eight of them. It is not possible to state categorically whether or not Coxsackie B viruses 2 or 3 in three cases or mumps virus in two cases may have damaged the pancreas.

We wish to thank the staff of the Virology

Laboratory, Radcliffe Infirmary, for their great assistance with this study.—We are, etc.,

J. D. BAUM
A. AYNLEY-GREEN

University Department of Paediatrics,
John Radcliffe Hospital

F. O. MACCALLUM

Virology Department,
Radcliffe Infirmary,
Oxford

Confusion of Tongues

SIR,—The Duke of Cumberland referred to the Highland clansmen as speaking "with their Irish tongues," a comment which has no doubt endeared him even less to the descendants of those slain at Culloden. Drs. P. A. Emerson and M. S. Lewis (1 June, p. 488) refer to the language of Northern Belgium as Dutch. Sir, the language of Northern Belgium is Flemish. Such is the friendship and courtesy of our colleagues in Ghent and Antwerp, and moreover their realization of the linguistic incompetence of the British, that they would be ready again to welcome Drs. Emerson and Lewis as they have done before. Nevertheless I think an apology to them for the error would not be out of place.—I am, etc.,

GEORGE DAVISON

Cramlington, Northumberland

The Killer Worm

SIR,—Tetanus unfortunately is all too common in India, up to 30 or 50% of cases proving fatal. Even more prevalent is the intestinal nematode *Ascaris lumbricoides*. That there could be any connexion between the presence of ascaris in the bowel and the outcome in a case of tetanus may at first seem unlikely. However, our observation has been that a severe or fatal laryngeal spasm in tetanus often directly precedes the passage of a roundworm from the nose or mouth. The worm presumably stimulates vagal afferent fibres in the pharyngeal mucosa in the neighbourhood of the epiglottis which initiates reflex spasm of the laryngeal muscles. A true cough is seldom possible in severe tetanus because of the rigidity of the muscles involved in the reflex.

What encourages the worm to migrate upwards from its position in the bowel is open to speculation. The constantly high intrathoracic and abdominal pressures caused by muscle contraction could interfere with oxygen entry into the bowel and create an environment unfavourable to the worm.¹ Escape downwards would prove difficult in the tetanus patient, who is frequently constipated. Another factor to be considered is that we give these patients chlorpromazine in

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.,

F. D. DASTUR
G. H. KASHYAP
K. G. NAIR

Department of Medicine,
K.E.M. Hospital,
Bombay

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

London W.9

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.,

MALCOLM C. BATESON

Department of Medicine,
Dundee University

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.,

J. M. PETTIFOR
JOHN D. L. HANSEN

Metabolic and Nutrition Unit,
University of the Witwatersrand,
Johannesburg

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.