that the medical profession in Britain will urge the relevant authorities there to support this amendment. The wellbeing and probably the longevity of patients with coeliac disease is at stake.

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Toxocara infections and kennel workers

SIR,—It is now widely recognised that the embryonated ova of the common roundworm of the dog, *Toxocara canis*, are infective for man. Surveys have shown that approximately 2% of normal healthy adults in the UK possess demonstrable antibodies to toxocara antigen and in certain circumstances infection can produce clinical disease.^{1,2} Borg and Woodruff³ reported that 24.4% of 800 soil samples from public places all over Britain contained *Toxocara* spp eggs, but still heavier contamination may be found in special situations such as, for example, some breeding kennels and dog training schools.⁴

One such location was identified during a recent study of the helminth parasites of greyhounds.⁵ The establishment in question consists of a central complex of buildings, including administrative offices and residential quarters for dog handlers. These are surrounded by kennels and 56 hectares (140 acres) of exercising paddocks and walks. More than 500 adult greyhounds are kept for racing and 12.5% of these carry patent *T. canis* infection—that is, about 19m eggs are deposited on the estate each day. Examination of 10 g of soil taken from each of 12 sites on the estate revealed the presence of *T. canis* ova in 11, with up to 18 eggs per sample. In addition, grass collections from small exercising paddocks yielded up to 828 ova per kg of foliage, although only 0.7% of these were embryonated.

It was considered that this high degree of contamination might present a potential health hazard to the 70 employees working at the kennels, particularly as the majority also live on the estate. An attempt was therefore made to evaluate the degree of risk involved. Serum was collected from 34 volunteers and examined by a fluorescent antibody test using a *T. canis* larval antigen.⁶ It was found that none of the 24 people who had worked at the kennels for between 6 months and 10 years showed evidence of infection (see table), but two workers with 22 and 43 years' service respectively had positive antibody titres. A further two long-term employees were, however, negative.

Although no general conclusions can be drawn from this one small study the results suggest that, at least in this instance, infection with *T. canis* is not readily acquired by kennel

staff maintaining a reasonable standard of personal hygiene. The health hazard, although constantly present, would seem to be slight.

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¹ Woodruff, A W, *British Medical Journal*, 1970, 3, 663.

² Woodruff, A W, *Environmental Health*, 1976, 1, 29.

³ Borg, O A, and Woodruff, A W, *British Medical Journal*, 1973, 4, 470.

⁴ Jacobs, D E, Pegg, E J, and Stevenson, P, *Journal of Small Animal Practice*. In press.

⁵ Jacobs, D E, and Prole, J H B, *Veterinary Parasitology*. In press.

⁶ Bisserru, B, and Woodruff, A W, *Journal of Clinical Pathology*, 1968, 21, 449.

Local gold toxicity

SIR,—I am not wholly convinced that Dr J A N Rennie (27 November, p 1294) has in fact described one of "the more readily avoidable toxic reactions" to gold. Nor do I think that his advice to clinicians that they "should not forget to ask the basic question about local gold toxicity" is of any practical value.

Contact dermatitis due to gold has certainly been described, but it is so rare that individual case reports still make news.¹ This contrasts with the prevalence of nickel dermatitis. Nickel is said to be the commonest sensitising agent encountered by dermatologists in Britain² and is the cause of the vast majority of skin reactions associated with the wearing of gold jewellery. It would be most interesting to know the results of patch testing to nickel and gold in the patient described by Dr Rennie. One of the features of contact sensitivity to gold as previously described³ has been the persistence of the eruption for many weeks or even months after the elimination of all contact with gold. The speedy resolution of his patient's rash clearly differs from this experience.

It would be a great pity if patients who might benefit from gold were to be denied this opportunity on account of a history of sensitivity to jewellery without at least the confirmation of gold sensitivity by patch testing. I believe that the case against gold in his patient is unproved. It seems to me to be almost as likely that his patient had a recurrence of a nickel dermatitis due not to the gold in the syringe but to the nickel in the needle that delivered it.⁴

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¹ Young, E, *Dermatologica*, 1974, 149, 294.

² Calnan, C D, *British Journal of Dermatology*, 1956, 68, 229.

³ Comaish, S, *Archives of Dermatology*, 1969, 99, 720.

⁴ Stoddart, J C, *Lancet*, 1960, 2, 741.

Prevention of cross-infection

SIR,—How do we know that the methods we use to prevent cross-infection are effective? Statistics of comparative infection rates are seldom very convincing and anecdotes may be misleading. All the same, here is an anecdote.

A child who had been treated for acute leukaemia by a bone-marrow graft developed

a severe exfoliating skin condition. He occupied a positive-pressure-ventilated (PPV) cubicle, one of 10 in a protective isolation ward.^{1,2} His skin was infected with a staphylococcus, which regular bacteriological monitoring had shown was acquired when he was out of isolation at another hospital.

On 22 April 1976 the routine weekly settle plates were distributed round the unit. On that day there were three patients being nursed in protective isolation. A fourth, who would have been isolated according to her neutrophil count, had not proved psychologically suitable and was in an open cubicle, from which she walked into the corridor from time to time. The table shows the total colony count and PPV numbers of staphylococcal colonies on 3-h settle plates in the child's PPV cubicle, the three isolation cubicles, and the open cubicle. Air from all cubicles flows into the corridor, which is not monitored but which in these circumstances was probably contaminated fairly heavily.

Settle-plate colony counts

	<i>Staphylococcus aureus</i>	Total
Isolation cubicle A ..	0	11
" " B ..	0	26
" " C ..	4	18
Open cubicle ..	2	31
PPV cubicle ..	230	320

Samples of staphylococci from plates in all these areas were found to be of the same phage type (52/52A/80/81). Five days later, the patient who would have been isolated but was not, developed septicaemia. The identical staphylococcus was grown from her blood cultures.

One episode of this sort proves nothing, but the conclusion that protective isolation is valuable for susceptible patients is more likely to be right than wrong.

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¹ Jameson, B, *Proceedings of the R3 Association*, Gothenburg, 1975.

² Jameson, B, *et al*, *Lancet*, 1971, 1, 1034.

L-Tryptophan and sexual behaviour

SIR,—The recent report by Drs G P Egan and G E M Hammad (18 September, p 701) of an unusual side effect of sexual disinhibition in four male schizophrenic patients with depressive features and in one male patient suffering from manic depressive psychosis, all receiving a preparation of L-tryptophan together with their usual medication, is surprising as it runs contrary to all published evidence. In the series of depressed patients treated by us with L-tryptophan and reported earlier (21 February, p 460) we did not notice any such effect upon sexual responsiveness. Moreover one of us (ADB) has now treated over 500 patients with L-tryptophan and in no case has hypersexuality been apparent.

The somewhat unexpected response of the patients treated by Drs Egan and Hammad prompted us to review the relevant published literature. P-chlorophenylalanine (PCPA) interferes with the conversion of tryptophan to 5-hydroxytryptophan (5-HTP), and it is well documented

Results of fluorescent antibody test for infection with toxocara related to length of service of employees at greyhound kennels

Period of employment	Male*		Female†	
	No tested	No positive	No tested	No positive
<6 months ..	3	0	3	0
6 months-2½ years ..	10	0	7	0
2½-10 years ..	7	0	—	—
>10 years ..	4	2	—	—

*10 aged 16-19, 5 aged 20-30, and 9 over 30 years.
†7 aged 16-19 and 3 aged 20-30 years.

that it produces compulsive sexual activity in male animals from the experiments by Ferguson *et al.*,¹ Tagliamonte *et al.*,² Shillito *et al.*,³ and Gessa *et al.*⁴ The specificity of this action is demonstrated by the fact that the sexual excitement induced by PCPA is inhibited by administration of 5-HTP. Gessa and Tagliamonte⁵ have hypothesised a disturbance of 5-hydroxytryptamine (5-HT) as the causal element in sexual dysfunctions. Patients with multiple sclerosis treated by Hyypya⁶ with L-tryptophan did not show any significant change in sexual motivation. Hyypya *et al.*⁷ also administered the amino-acid to normal volunteers, and although no change in sexual behaviour or arousal was noted, transient decreases in plasma gonadotrophins were found after each dose. Finally, Sicuteri *et al.*⁸ observed that L-tryptophan was capable of diminishing excessive sexual responsiveness in patients suffering from hypersexuality.

It is difficult, therefore, to understand the cause of the hypersexuality seen after L-tryptophan in Drs Egan and Hamad's patients. It should be noted, however, that four out of five of them were suffering from schizophrenia and were being treated with phenothiazines in addition to the amino-acid. Bender⁹ has observed that phenothiazines have a complex action on serum tryptophan in schizophrenics; it appears that in such patients they reduce brain serotonin synthesis, whereas in rats they have the reverse effect. It might be postulated that, in similar fashion, administered L-tryptophan may have an atypical effect in schizophrenic patients receiving phenothiazines.

L-Tryptophan has so far been mainly indicated for the treatment of affective disorders and we believe that it has been used very little in the management of schizophrenia. Smythies¹⁰ has recently drawn attention to its possible application in schizophrenic patients. If his advice is followed hypersexuality may become a more commonly reported side effect.

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¹ Ferguson, J, *et al.*, *Psychophysiology*, 1969, 6, 221.

² Tagliamonte, A, *et al.*, *Science*, 1969, 166, 1433.

³ Shillito, E E, *British Journal of Pharmacology*, 1970, 38, 305.

⁴ Gessa, G L, *et al.*, *Nature*, 1970, 227, 616.

⁵ Gessa, G L, and Tagliamonte, A, *Sexual Behaviour: Pharmacology and Biochemistry*. New York, Raven Press, 1975.

⁶ Hyypya, M T, *British Medical Journal*, 1976, 2, 1073.

⁷ Hyypya, M T, *et al.*, Presentation at International Congress of Pharmacology, Helsinki, 1975.

⁸ Sicuteri, F, *et al.*, Presentation at International Congress of Pharmacology, Helsinki, 1975.

⁹ Bender, D A, *Lancet*, 1976, 2, 427.

¹⁰ Smythies, J R, *Lancet*, 1976, 1, 136.

Community self-care

SIR,—For the past two or three years we have had a community care scheme which is organised by the local inhabitants. It has provided a great deal of help in our practice in that there is a panel of volunteer drivers who will take visitors to hospital, collect prescriptions for the housebound, and also bring patients to the health centre and even provide immediate companionship in a family crisis.

We try to make reasonable use of their services and not abuse them. The local people are encouraged to offer their services for driving, gardening, phone-answering, etc. There is a phone number (easily remembered) to be used for offers and requests. It is not intended as a "them and us" arrangement but one in which there are times for giving help and times for receiving it (in one form or

another). Finally, it is not used as a system for avoiding proper medical responsibilities but as an aid to improving care. It is known as LINK (presumably "the bond between").

The details are beyond the scope of a short letter, but further inquiries are welcomed.

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Ferrous sulphate: danger to children

SIR,—We were disappointed to receive a recommendation from the DHSS recommending the use of ferrous sulphate. The *British National Formulary* 1976-1978 is quoted as saying: "The dosage of elemental iron should be at least 100 mg daily. Ferrous sulphate in tablet form is the preparation of choice."

Although ferrous sulphate is the cheapest iron preparation, it is also the most toxic. The green, sugar-coated tablets attract small children, who may suffer haemorrhagic necrosis of the gastric or jejunal mucosa, liver damage, and sometimes death after swallowing them. A very common sequence is that the toddler gains access to his mother's iron tablets, prescribed for her when she is pregnant again.

In 1966 four infants under 3 years of age were admitted to our children's ward with ferrous sulphate poisoning. All were the offspring of our antenatal patients. Three were severely shocked and had gastric haemorrhages and one was left with an hour-glass constriction of the stomach. The ferrous sulphate tablets had been dispensed with a warning in red on the bottle that the tablets were dangerous to children.

Since 1967 the prescription of ferrous sulphate for pregnant women has been forbidden in our obstetric department and local general practitioners have been encouraged to use other preparations. Between January 1967 and December 1975, of 16 children admitted after ingesting iron tablets only three had taken ferrous sulphate and no case was severe.

We are convinced that, at least for pregnant women with young children, it is wise to avoid prescribing ferrous sulphate.

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FEIBA in haemophiliacs with factor VIII inhibitor

SIR,—Dr G D O Lowe and others (6 November, p 1110) report the successful use of prothrombin complex to secure haemostasis in a haemophilic with an inhibitor to factor VIII. Others have used "factor VIII inhibitor bypassing activity" (FEIBA) in such patients with apparent success,^{1,2} prompting us to report our experience with this product.

A 32-year-old man sustained extensive anterior myocardial infarction in 1975. Intractable angina of effort developed subsequently and coronary arteriography revealed stenosis of the anterior descending branch of the left coronary artery. Femoral arterial puncture for this procedure led to extensive bruising of the groin and thigh. Excessive bleeding had also occurred after dental extractions

in 1967. Coagulation screening revealed a prolonged activated partial thromboplastin time (APTT) of 70 s (control 34 s) with an otherwise normal profile. The prolonged APTT showed little correction in mixing tests with normal plasma, suggesting the presence of a rapidly acting inhibitor. Levels of all recognised clotting factors were normal when assayed at high dilution.

The APTT of plasma containing moderate numbers of platelets shortened during standing at room temperature for several hours; the association between platelets and the contact phase of coagulation led us to investigate the possibility that the inhibitor might be an abnormal variant of the physiological "anti-Xla."³ We found that the inhibitor could destroy contact activation product prepared from the patient or from normal subjects very rapidly; it differed in many respects from physiological "anti-Xla."

It seemed likely that FEIBA would bypass the activity of this inhibitor. A trial dose of 15 U/kg body weight was given. The APTT shortened by 12 s and this effect lasted at least 72 hours, without evidence of intramuscular coagulation. At operation a vein graft was used to circumvent the stenosis under cardiopulmonary bypass. While on bypass the patient was partially exchange-transfused with six units of bank blood. During closure, after heparin neutralisation, FEIBA 40 U/kg was given together with fresh blood, platelet concentrate, and fresh frozen plasma. Excellent haemostasis was achieved, with complete normalisation of the APTT, again without evidence of intravascular coagulation although the contribution of FEIBA to haemostasis is uncertain. Postoperative recovery was uneventful and in the four weeks since surgery the APTT has risen slowly to 70 s.

We will report this case and the properties of the unusual inhibitor in more detail elsewhere.

We thank Mr D Ross, who kindly gave us permission to report this case.

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¹ English, P J, Sheppard, E M, and Wensley, R T, *Lancet*, 1976, 1, 1299.

² English, P J, Sheppard, E M, and Wensley, R T, *Lancet*, 1976, 2, 207.

³ Nossel, H L, and Niemi, J, *Blood*, 1965, 25, 712.

Low molecular weight dextran

SIR,—While in agreement with three of the rules quoted by Dr T G Feest (27 November, p 1300) for the use of low molecular weight dextran (LMWD), I would disagree with the fourth. He states that LMWD should not be infused at a rate faster than 1 l/day. However, this would exclude the very useful effect of dextran 40 in patients who have polycythaemia secondary to chronic hypoxic lung disease, as reported by Harrison *et al.*¹ This technique involves withdrawing 2 l of blood in 1 h and giving 1 l of dextran 40 simultaneously and another 1 l over the next 6-8 h. This has great symptomatic benefit for secondarily polycythaemic patients, reducing their packed cell volume (PCV) to near normal levels for as long as six months, when the procedure may be repeated. Not only are headaches and lethargy improved, but also pulmonary artery pressure may be reduced and thus right ventricular strain. It has been shown experimentally in dogs that the pulmonary vascular resistance rises exponentially with a rise of PCV.² Simple venesection without dextran 40 may lead to an increased risk of clotting.