be taken into the operating-theatre." Only sterile blankets should be used for this purpose, and we should not be prevented from using polypropylene because of an imaginary danger from static electricity. If a danger does exist polypropylene blankets appear to carry very little static and they could easily have woven into them (if the makers are told this is essential) an anti-static thread.

We firmly believe that cross-infection must be fought on every front even if the effect in some cases is hard to demonstrate. We have no doubt that it will be worth while converting all our hospital linen to these autoclavable materials. Polypropylene has many uses in hospitals, and these fibres may prove to be the most advantageous of all.—We are, etc.,

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Hypertensive Crisis with Eskornade

SIR,—I would be grateful if you would bring to the attention of your readers the following case of hypertensive encephalopathy following the ingestion of Eskornade spansules.

The patient, a boy aged 16, was admitted in a severely confused state within two hours of ingesting eight Eskornade spansules. He had apparently taken these because of nasal discharge before going to bed, thinking that they would help him to sleep. He awoke an hour or two later with a severe headache and became restless and excitable, talking incoherently and vomiting frequently.

On admission he was confused and very restless, with photophobia but no neck rigidity. He was apyrexial but had a tachycardia of 104 per minute. His blood-pressure was 190/150 mm. of mercury. Both femoral pulses were palpable but not delayed. There was no evidence of cardiovascular hypertrophy. There were several purple striae in each flank. The pupils were fixed and widely dilated and the retinal veins were congested, but there was no papilloedema. Urinalysis showed a trace of glucose and moderate albuminuria.

In view of the fact that the history of drug ingestion was not forthcoming for several days a provisional diagnosis of hypertensive crisis due to Cushing's syndrome or phaeochromocytoma was made. Lumbar puncture revealed clear cerebrospinal fluid at a pressure of 200 mm. of water, with 55 mg./100 ml. protein and normal globulins. Other investigations were normal.

The patient subsequently recovered fully and became normotensive and it was then that we obtained the history of drug ingestion.

Eskornade spansules (Smith Kline and French) are sustained-release capsules used for the relief of nasal congestion in vasomotor or allergic rhinitis. Each spansule contains phenylpropanolamine hydrochloride 50 mg. (a sympathomimetic amine), isopropamide iodide 2.5 mg. (a drying agent), and diphenylpyraline hydrochloride (Histryl) 5 mg. (an antihistamine). Eight capsules contain 400 mg. of phenylpropanolamine, releasing, it is claimed, about 130 mg. soon after ingestion and the rest over the next 10–12 hours. Phenylpropanolamine (norephedrine) is a sympathomimetic amine resembling ephedrine but having greater vasoconstrictor action and less central stimulating and bronchodilator action. The maximum oral dose recommended for nasal decongestion is 50 mg. t.d.s. and for hypotensive states 50 mg. parenterally. It may be assumed that 130 mg. available soon after ingestion is sufficient to produce a

severe hypertensive crisis, and this on its own or combined with the antihistamine may have produced central excitation.

My thanks are due to Dr. J. W. Paulley for permission to report this case.

—I am, etc.,

PAUL R. SALMON.

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Toxic Insecticides

SIR,—May I endorse the concern expressed by Dr. A. M. G. Campbell (26 December, p. 1658) relating to toxic insecticides in general, and organo-phosphorus ones in particular. I would confirm that the Ministry of Agriculture urges veterinary surgeons to advise their clients to use a systemic organo-phosphorus insecticide to control warble-fly infestation in cattle.

Extensive research into the usefulness and safety of this insecticide has been undertaken by the manufacturers and corroborated by the Ministry of Agriculture. Despite this, last month several cattle died within 48 hours of the approved use of this organo-phosphorus chemical. Cholinesterase blood determinations were conflicting. Death was preceded by muscular weakness and coma without any evidence of tremors, diarrhoea, or abdominal pain. Lesions in all the dead animals from more than one farm were widespread, with intense haemorrhagic inflammation of all mucous surfaces in the thoracic and abdominal cavities, and petechial haemorrhages on the serous surfaces. These symptoms and lesions are not typical of those of published organo-phosphorus poisoning in animals and man, and investigations are still continuing in efforts to determine the presensitizing factor which prompted these anaphylactic-like deaths.

What happens when an animal breaks a leg soon after application, or for other reasons has to be slaughtered in an emergency? Can and will organo-phosphorus residues be detected in the carcass before it is sold to the unsuspecting housewife, and will she or her family suffer ill-effects from pre-existing sensitization of which both she and they may be unaware?

Have, in fact, the "clearing agencies" paid sufficient attention to synergism or potentiation between differing toxic chemicals which may or may not be related to one another, but which the agricultural community is pressed to buy and use under the allegation that these will save them money and produce bigger and better crops?

A letter was published in the *Veterinary Record* in October drawing attention to the dangers to cattle and humans, and before these recent deaths transpired. Animals and birds may be more sensitive to these toxic chemicals than man, and perhaps these unfortunate deaths might be regarded as a "barometer" when considering the effects on man.

There is little doubt that deaths and illnesses from toxic chemicals escape members of my profession, and until one has become personally involved with strong circumstantial evidence, and carried out patient investigations to sift the weed from the corn, it is relatively simple to arrive at an inaccurate diagnosis, where an apparently more realistic aetiology springs to mind, because it may be more commonplace and acceptable to orthodox training. I was guilty of issuing three

inaccurate post-mortem certificates last year, because symptoms and lesions caused by certain toxic chemicals were indistinguishable from more common ailments, both in terminal symptoms and at post-mortem examination.—I am, etc.,

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Tenterden, Kent.

F. D. T. GOOD.

Iron Content of Infant Milk Foods

SIR,—In your articles on child care I fail to find any reference to the iron content of milk foods used for the artificial feeding of young infants. It would appear, on a comparison of national dried milk with various proprietary foods, that the former, on which so many of the infants belonging to our industrial populations are fed, is the lowest in iron content, with approximately 0.15 mg. per ounce (0.005 mg./g.). One proprietary product, emphasized to be "humanized," has 0.3 mg. per ounce (0.011 mg./g.), while two well-known proprietary foods, made from cows' milk, contain approximately 1 mg. per ounce (0.036 mg./g.), as also does a popular proprietary infant food made from dried skimmed milk.

I am informed that the official attitude is against adding iron to national dried milk because the healthy baby is provided with sufficient iron at birth to last until weaning. On the other hand, two of the firms making proprietary foods refer to the important work of Mackay,¹ on hypochromic anaemia of infants as their authority for reinforcing the very low iron content of natural cows' milk, which is the basis of their product. Although most babies are born with enough iron to satisfy their physiological needs, others may be deficient because of prematurity or other factors, and may therefore suffer from infantile anaemia, with a resultant increased morbidity rate. Questions that arise from this consideration are the desirability of adding iron to infant foods and the amount, if any, to be added. A subsidiary question concerns possible harm in giving small amounts of iron to infants who do not require it.

As a fortified milk food with 20 mg. of iron per ounce (0.72 mg./g.) has been used successfully for the treatment of infantile anaemia, it would seem unlikely that any harm would accrue from all baby foods having a minimum content of 1 mg. of iron per ounce as a contribution towards the prevention of infantile anaemia, which my own experience suggests is not uncommon.—I am, etc.,

Health Department,
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JAMES GRANT.

REFERENCE

- ¹ Mackay, H. M. M., *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, No. 157, 1931. H.M.S.O., London.

Need for Family Histories

SIR,—It is agreed that the reports of Clinicopathological Conferences from the Postgraduate Medical School at Hammer-smith justly deserve their popularity. They read easily and entertain while instructing gently. However, it seems inconceivable that year after year so much can be said about patients and yet so little about human beings with loves and hates, feelings and fantasies, and, above all, families. Clearly a full social