CHARCOAL HÆMOPERFUSION FOR INSECTICIDE OVERDOSE

SIR,—Oxychlordane is an insecticide which is similar to and has largely replaced D.D.T. (dicophane) as an insecticide both commercially and for home use. It is toxic to the liver, kidneys, lungs, heart, and the central nervous system. Like other organochlorine compounds it is highly fat soluble and has a long half-life of three months.[1] Hæmodialysis and peritoneal dialysis are ineffectual in removal of this poison.[2] Hæmoperfusion does not seem to have been tried previously.

A 35-year-old man presented on April 19, 1977, having swallowed about 90 g of chlordane 2 h previously as a suicide attempt. The lethal dose is 6 g or more. Vomiting was induced and the patient was given oral magnesium sulphate. He then had a grand-mal seizure. Hæmoperfusion was started 4 h later, using a 'Hemocol' 300 g acrylic encapsulated activated charcoal column, manufactured by Warner-Chilcott. Hæmoperfusion was done at a flow-rate of 200 ml/min for 4 h. The oxychlordane concentrations (measured at the University of South Carolina) were, in parts per 10^9, 1800 before hæmoperfusion, 676 after hæmoperfusion, and 481 4 days later. In the general population only trace amounts of oxychlordane can be detected. In individuals occupationally exposed, the mean level[3] is 0·55 parts per 10^9.

The patient subsequently had no further neurological or other clinical symptoms. A 2+ proteinuria cleared. Neither blood-urea nor liver enzymes increased. His platelet-count fell to 110 000/μl immediately post hæmoperfusion because of charcoal absorption.

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IS AZATHIOPRINE NECESSARY IN RENAL TRANSPLANTATION?

SIR,—Dr Sheriff and his colleagues (Jan. 21, p. 118) suggest that azathioprine might not have a major role in the immunosuppressive management of renal-transplant patients. Our experience supports this conclusion to some extent. Azathioprine was interrupted on 26 occasions in twenty-one cadaver graft recipients. Withdrawal was decided because of jaundice (bilirubin $\geqslant$ 2 mg/dl) due to hepatic disease, between 2 and 109 months after transplantation (beyond 24 months in 11 out of 26 occasions). Interruption lasted between 1 and 5 months on 20 occasions in sixteen patients. One acute rejection episode was observed in three patients and successfully reversed by steroids with an eventual return of serum-creatinine to pre-rejection values. In the 17 other instances the serum-creatinine remained constant. Interruption lasted between 5 and 72 months on 6 occasions in six patients. In three cases serum-creatinine remained stable whereas in three others it rose slightly but progressively (from 0·09 to 0·17 mmol/l over 6 months, from 0·09 to 0·18 mmol/l over 29 months, and from 0·11 to 0·44 mmol/l over 72 months). No biopsy was done and we do not know if this rise was due to chronic rejection. We can thus confirm that azathioprine withdrawal does not lead to acute irreversible rejection. The possibility that chronic rejection might ensue cannot be excluded, however, because serum-creatinine concentrations rose in three out of six patients after long-term withdrawal. This observation is in keeping with the data of Sheriff et al. who noted a rise of more than 0·05 mmol/l in two out of the fifteen patients in whom azathioprine was stopped 24 months after transplantation. Only a prospective, controlled study will demonstrate whether or not this frequency of decreased renal function is linked with azathioprine interruption. To throw light on this question we have reviewed serum-creatinine rises at between 24 and 48 months in fifty patients with a cadaver graft who had had azathioprine continuously. In six (12%) the serum-creatinine rose by more than 0·05 mmol/l, a frequency comparable with that recorded by Sheriff et al. (two out of fifteen, or 13%). Thus azathioprine withdrawal after 24 months does not seem to lead to a higher frequency of progressive deterioration of graft function.

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ANTIBIOTICS FOR LEGIONNAIRES' DISEASE

SIR,—The description of the results of limited trials of antibiotic therapy in the guineapig model of legionnaires' disease by Dr Fraser and his colleagues (Jan. 28, p. 175) will help the clinician to interpret in-vitro sensitivity data for this organism.[1] However, in view of the exquisite sensitivity of the guineapig to penicillin G, in which doses as small as 15 mg/kg may be fatal,[2] it seems premature to conclude that penicillin G is ineffective in vivo against the causative organism. Have antibiotic trials been done in animals in which penicillin is not a toxic antibiotic?

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DETECTING THE DISLOCATED HIP

SIR,—I agree with you that screening for hip dislocation in the neonatal department alone "is inadequate protection against future disability".[3] However, this "inadequate protection" should not be due to failure of diagnosis but only to treatment which is still not always successful even when started at an early age by experienced staff. Close collaboration between pædiatric and orthopædic staff well trained in hip examination may eliminate late diagnosis, provided that tests are repeated, at home, during the first month of life. This approach might overcome the three main reasons of failure you listed and would provide the opportunity to encourage simple preventive methods which should be applied to all children. Parents should, from the birth of their child, ensure that her limbs are always free for all movements, that the baby is carried astride when borne on the arms, and that she is never put under pressure to stand or walk.

In my experience a consistently negative click (jerk) test since birth accompanied by normal abduction of the thighs reflects a normal hip or a simple dysplasia which will recover spontaneously. On the other hand, a negative clinical test with very limited abduction makes a congenital pelvic deformity or severe dislocation very likely, and suggests the need for an X-ray (which, in these cases, is perfectly reliable even at birth).

This policy has been practised in Ferrara for more than forty years. Here late diagnosis has almost disappeared, avascular necrosis is very rare, and disability has diminished as treatment skills have improved. We have clinical, radiological and/or pathological data on more than eight thousand cases of hip dysplasia mostly detected in children in three first months of life, with high rates in the newborn, in premature babies, and in stillbirths.

Your view—based largely on neonatal testing done before discharge from hospital[4,5]—is too pessimistic.

1. Aldrich, F. D., Holmes J. H. *Arachs envir Hlth*, 1969, **19**, 129.
2. Poisonindex, National Center for Poison Information, Rocky Mountain Poison Center, University of Colorado Medical Center, Denver, Colorado.
3. Sandifer, S. H. Personal communication.

1. *Morbid. Mortal wkly Rep.* 1977, **26**, 152.
2. Newton, W. L., Steinman, H. G., Brandriss, M. W. *J. Bact.* 1964, **88**, 537.
3. *Lancet*, 1977, II, 909
4. Jones, D *J. Bone Jt Surg* 1977, **59B**, 318.
5. Bjerkreim, I *Acta orthopæd. scand suppl.* 157.