of C.S.F. with the isotope solution may be expected to cause less disturbance to the C.S.F. system than the injection of air.

(4) Finally, air injected during the A.E.G. is reabsorbed very slowly in communicating hydrocephalus, and its presence may delay curative surgical treatment because of the risk of air embolism. There is no such risk after isotope encephalography.

It must be recognised that isotope encephalography is usually complementary to A.E.G. and does not supplant it in the investigation of progressive dementia. The A.E.G. may show more clearly than isotope encephalography, focal blockage in the basal cisterns and dilatation of cisterns caudal to the blockage (Crunquist 1967). However, if (for example, after subarachnoid hæmorrhage or head injury or meningitis) obstruction due to a tumour can be excluded, from the history, isotope encephalography may become the investigation of choice in a patient with suspected communicating hydrocephalus.

The relation between ventricular size and C.S.F. pressure in communicating hydrocephalus has attracted considerable interest lately. An early case of communicating hydrocephalus after subarachnoid hæmorrhage with normal C.S.F. pressure was described by Foltz and Ward (1956). Adams et al. (1965) have described further cases and have introduced the term "normal pressure hydrocephalus". This term is perhaps misleading because it is likely that the C.S.F. pressure is raised at some stage of the disease. Analogies with a simple physical system have been discussed by Adams et al. (1965) and these workers have pointed out that if the volume of such a system increases then the force on the wall may increase even if the actual pressure within the system remains the same. The analogy has, however, only indirect application because the resistance of the ventricular wall is unlikely to be uniform. Whatever the theory, the fact of the matter is that patients with communicating hydrocephalus and "normal cerebrospinal-fluid pressure" improve if the pressure is lowered.

What is the cause of communicating hydrocephalus in patients in whom there is no history of subarachnoid hæmorrhage, head injury, or meningitis? These patients could not be distinguished, on clinical grounds or on investigation, from the patients with an obvious cause, though in one patient the C.S.F. protein was slightly raised. It seems probable that there is a similar obstruction to C.S.F. flow in both groups of patients, though the pathological cause may be different. Further work is needed to establish whether there is a chronic arachnoiditis or possibly late decompensation of long-standing communicating hydrocephalus in those patients without any obvious preceding cause. Our present ignorance about the ætiology in such cases need not halt the search for patients with communicating hydrocephalus who can be helped by ventriculoatrial drainage.

We thank the physicians and surgeons of the National Hospitals who permitted us to study patients under their care and Dr. James [illegible] for his help and encouragement.

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FÆCAL EXCRETION OF LEAD BY CHILDREN

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Summary Fæcal lead is a sensitive index of ingested lead. Normal values for children aged 2 years have been determined and contrasted with the findings in three cases of lead poisoning. Fæcal lead determinations have a place in population screening programmes and in the interpretation of blood-lead values in children.

Introduction

THE detection of children abnormally exposed to lead has previously depended upon a history of pica, elevated blood and urine lead levels, and evidence of disturbed porphyrin metabolism. None of these methods has proved to be satisfactory in the detection of exposed as opposed to poisoned children (Barltrop 1966, 1967). Although emphasis has been placed on the determination of blood-lead (Moncrieff et al. 1964, Gordon et al. 1967), the significance of moderately elevated values may be doubtful since they may indicate release of bound skeletal lead rather than recent lead ingestion (Byers and Maloof 1954).

The administration of lead compounds to human adults (Kehoe 1961) and to animals (Blaxter 1950) is accompanied by increased fæcal levels and at least 90% of the ingested lead is unabsorbed. Children with lead poisoning also have a raised fæcal lead (Chisolm and Harrison 1956) but little advantage has been taken of this either in the detection of children abnormally exposed to lead or in the interpretation of elevated blood-levels.

Preliminary animal work confirmed that fæcal lead was a sensitive index of the ingestion of lead compounds compared with blood and urine measurements. The findings were applied to healthy and lead-poisoned children.

Methods

Children of both sexes aged 24–35 months were investigated, comprising four groups:

Group I.—Consecutive hospital inpatients admitted during the study period with illnesses other than lead poisoning.

Group II.—Healthy children of unselected mothers attending a local infant-welfare clinic. No history of pica or mouthing for painted materials for 7 days preceding interview.

Group III.—As group II but with a positive history of pica or mouthing for painted materials during the 7 days preceding interview.

Group IV.—Hospital inpatients admitted for treatment of lead poisoning.

The first stool passed by each child after admission to hospital or the clinic interview was collected directly into a polyethylene bag, urine was separated in the laboratory and discarded. All fæcal specimens were homogenised with distilled deionised water in an 'Ato-Mix' blender and a weighed sample was obtained. Serial fæcal collections were made from two of the cases of lead poisoning and also from an ambulant child with mild cerebral palsy on a normal ward diet. Venous blood specimens were obtained from children with lead poisoning with disposable syringes and needles and collected in heparinised polystyrene tubes.

Lead was analysed after preliminary wet-digestion using a semiautomated alkaline dithizone method (Browett and Moss

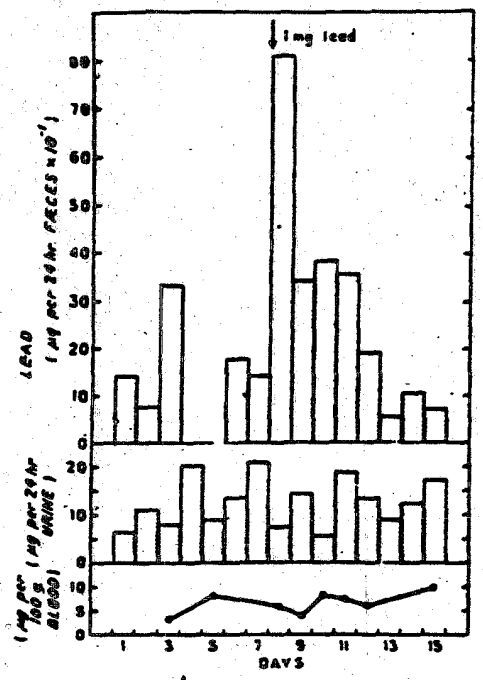

Fig. 1—Blood, urine, and faecal lead in a 3·0 kg. corgi pup. Oral lead 1·0 mg. given on day 8.

1965). This method had a lower limit of detection of 0·13 μg per aliquot. For repeated determinations of a standard containing 45·2 μg. per 100 g. the coefficient of variation was 4·2%. The mean recovery of lead compounds added to a faecal sample in each batch was 105%.

The animals investigated were pedigree corgi bitches maintained throughout on a standard commercial diet in nylon-dipped metabolism cages. Lead was administered to the animals orally as the acetate in 5·0 ml. aqueous solution.

Case-reports

Case 1

A 2-year-old boy who had licked paint from his cot and window sills for 6 months. For 6 weeks before admission he had been restless, irritable, reluctant to sleep, and was anorexic for ordinary foods. X-ray examination showed opaque material in the gut and dense metaphyseal lines at the wrist and knee. Haemoglobin 9·8 g. per 100 ml., scanty basophilic stippled cells seen in the peripheral blood, blood-lead 84·8 μg. per 100 g., faecal lead 1·9 mg. per specimen.

Case 2

A female child aged 2 years 2 months with a history of pica for flaking paint for 5 months. For 2 weeks before admission she had been miserable, anorexic, and vomited four times. X-ray examination showed dense metaphyseal lines at the wrist but no opaque material in the gut. Urine coproporphyrin 0·1 μg. per ml., haemoglobin 9·2 g. per 100 ml., no basophilic stippled cells seen in the peripheral blood, blood-lead 68·3 μg. per 100 g., faecal lead 1·3 mg. per specimen.

Case 3

Male child aged 2 years 3 months with a 4-month history of pica for flaking paint. For 3 weeks before admission he was unwell, irritable, and vomited each morning. Pica ceased 5 days before admission. X-ray examination showed dense metaphyseal lines at the wrist. Haemoglobin 9·7 g. per 100 ml., a basophilic stippled cells seen in the peripheral blood, urine coproporphyrin 0·2 μg. per ml., blood-lead 92·0 μg. per 100 g., faecal lead (1st specimen) 0·17 mg., (3rd specimen) 0·57 mg. per specimen.

Results

Preliminary animal studies with a constant oral daily dose confirmed that the faecal lead content increased as 91·4% of the administered dose within 8 days. The sensitivity of the faecal lead as an index of ingestion was determined by reducing the oral dose to a single 1·0 mg. administration after a control period. This caused a significant rise in the 24-hour faecal lead although the dose was too small to influence the blood or urine lead concentrations (fig. 1).

The mean faecal lead of single stool specimens from nineteen healthy children, irrespective of pica (groups II and III) was 123 μg. per stool with an upper limit of normal (mean + 2 S.D.) of 183 μg. Values for six hospital in-patients (group I) fell within these limits. There was no significant difference between the means of groups I, II and III, however at least one child with pica had a faecal lead content (468 μg.) which suggested recent ingestion.

The three lead-poisoned children (group IV) had faecal lead levels which exceeded those of normal children by an amount that made statistical comparison unnecessary (fig. 2). For cases 1 and 2 the values recorded were for the first stool after admission. Case 3, whose pica had ceased 5 days before admission, had an initial faecal lead within normal limits and the peak faecal lead excretion occurred on the 3rd day after admission. Serial specimens from case 2 showed that in hospital, normal values were regained within 6 days, although the blood-lead showed little alteration. Since day-to-day variation in faecal lead might be normal, serial collections were made from a child with mild cerebral palsy in the ward on a normal diet.

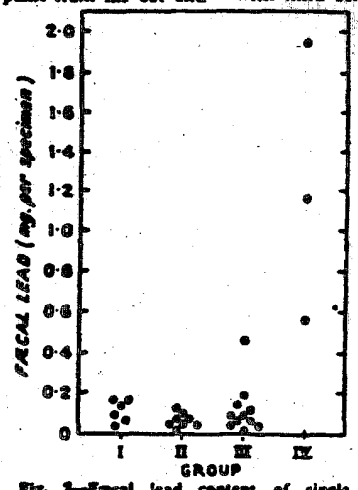

Fig. 2—Faecal lead content of single specimens from groups I-IV.

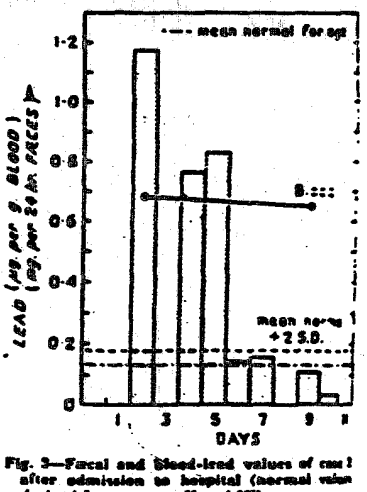

Fig. 3—Faecal and blood-lead values of case 1 after admission to hospital (normal values derived from groups II and III).

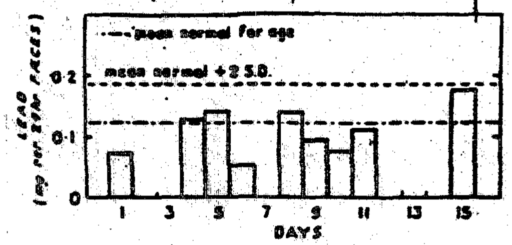

Fig. 4—Serial fæcal-lead determinations for an ambulant inpatient (normal values derived from groups II and III).

During a 2-week period the fæcal lead content fluctuated within, but did not exceed, normal limits (fig. 4).

Discussion

The rapid return to normal of the fæcal lead content of children who had ingested lead compounds, after admission to hospital and the almost quantitative transient fæcal elevation after single-dose adminstration of lead to a dog, both support the suggestion that the fæcal lead is indicative of ingested lead. Since lead can be detected in the fæces after ingestion without concomitant increase in the blood and urine levels, we suggest that blood and urine levels be abandoned as methods for the detection of exposed children.

The mean normal value of 130 μg. lead per specimen is similar to that of 132 μg. described by Chisolm and Harrison (1956) for children aged 12–35 months. However, the fæcal lead observed in cases 1–3 (0·57–1·9 mg.) are considerably lower than those reported by them (5·0–1040 mg.). The reason for this discrepancy is not clear although many of Chisolm's cases were severe and had frank encephalopathy.

The total lead ingested by children who become poisoned is relevant to legislation on paint and surface coating. If the first stool passed on admission to hospital represents the daily lead intake before admission, then cases 1 and 2 ingested 160–310 mg. of lead during 5–6 months. These figures compare with the whole-body lead of 220 mg. of a child who had died after pica for lead paint described by Aub et al. (1926). Deductions of this nature may be erroneous, since anorexia, vomiting, and constipation are well-recognised features of lead poisoning in children and were present in the cases reported here. The experimental ingestion of 3·0 mg. of lead daily by an adult resulted in unacceptably high blood and tissue lead concentrations after 4 months (Kehoe 1961).

This technique may have advantages for the screening of large numbers of young children, since the specimens are easily obtained and transported. The demonstration that at least one otherwise healthy child had ingested an abnormal amount of lead supports this, and indicates that preventive measures might be applied before toxic amounts of lead have been absorbed.

Conversely, after treatment for lead poisoning, some children may be found to have elevated blood-lead concentrations for several weeks. In such circumstances it is important to discover whether or not there has been a recurrence of pica for lead-containing materials. The lack of association between the blood and fæcal lead contents allows this distinction to be made and provides objective verification of the pica history.

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Requests for reprints should be addressed to D. B.

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Preliminary Communications

PLASMA-ZINC CONCENTRATIONS IN PATIENTS WITH PSORIASIS, OTHER DERMATOSES, AND VENOUS LEG ULCERATION

Summary Significantly lowered plasma-zinc concentrations were found in patients with psoriasis, other dermatoses, and venous leg ulceration. The significance of this finding requires further investigation.

INTRODUCTION

ZINC is an essential dietary constituent; it is a component of several metalloenzymes and behaves as a co-factor for many other enzymes. Although in man low plasma or serum zinc concentrations have been associated with cirrhosis of the liver [1] and the postoperative period,[2] and reduced amounts of zinc have been found in the hair of patients with atherosclerosis,[3] systematic studies of zinc metabolism in human skin disease have not been reported. We have, therefore, determined plasma-zinc concentrations in a group of patients with psoriasis, a group with ichthyosis, and a group with miscellaneous skin conditions. Because of the recent observation by Pories et al.[4] of accelerated wound healing after zinc administration, we also studied a small group of patients with chronic venous ulceration of the leg.

SUBJECTS AND METHODS

The subjects studied were aged 15–64 years, and were in five groups: (1) 41 healthy subjects (including 17 females) with no past history of skin disease; (2) 20 patients (including 9 females) with psoriasis involving 10% or more of the body-surface; (3) 13 patients (including 7 females) with miscellaneous skin conditions in which neither dyskeratosis nor chronic ulceration was a feature; (4) 10 patients (including 2 females) with ichthyosis; (5) 9 patients (including 8 females) with chronic venous leg ulceration.

Venous blood samples, collected using disposable plastic syringes and stainless-steel needles, were each transferred to a polystyrene specimen tube and mixed with 1 drop of heparin (heparin injection *B.P.*, 5000 units per ml., Weddell Pharmaceuticals Ltd.).* Within 2 hours of collection, the specimens were centrifuged at 3000 r.p.m. (M.S.E. "Minor" centrifuge) for 10 minutes; the plasma was separated, and stored at −20 C in polystyrene tubes.

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