93 brand A, all of 77 brand B, 6 of 10 brand C, and 1 of 3 brand F powders), and, while no precise conclusion can be drawn, these findings do suggest that the proportion of Sheffield infants to-day receiving mercury powders is nearer to one-tenth than the one-quarter surmised for the 1946-8 period.

As regards medical prescribing of mercury no data are available by which to compare the position in 1946-8 with that to-day, but there can be little doubt that it also is much reduced. Thus the chairman and secretary of the Sheffield Pharmaceutical Committees recall that seven years ago they would expect to see three or four prescriptions for calomel or grey powder each week, whereas it is now many months since either of them saw the last. Their colleagues speak in similar terms, and almost all the family doctors with whom the matter has been discussed say that they have stopped prescribing mercury for infants.

Incidence of Pink Disease in Sheffield, 1947-55

It is not possible to learn accurately the incidence of pink disease in a community, as the condition is not always recognized and is not notifiable. The study of incidence trends must therefore be based on indirect evidence. In this respect mortality rates have in Sheffield been too small to permit conclusions, and the best index of incidence trend has been the number of cases treated at hospital each year. All these patients are not referred to hospital, but discussions with family doctors suggest that the majority at any rate of definite examples of the disease are in fact sent to hospital, and column 2 of the Table shows the numbers attending the various paediatric units in the city each year for nine consecutive years.

Patients with Pink Disease Treated at the Sheffield Children's and City General Hospitals during 1947-55. Figures in Parentheses Represent Fatalities

Year	Total No.	No. Living Outside City Boundary	No. with Untraced Addresses	No. Living Within City Boundary
1947	65 (4)	30 (1)	3 (1)	32 (2)
1948	69 (3)	25 (1)	15 (1)	29 (1)
1949	57 (0)	26 (0)	0 (0)	31 (0)
1950	64 (2)	27 (1)	0 (0)	37 (1)
1951	55 (4)	19 (2)	1 (0)	36 (2)
1952	45 (2)	19 (0)	0 (0)	26 (2)
1953	36 (1)	15 (1)	1 (0)	21 (0)
1954	20 (1)	3 (1)	0 (0)	17 (0)
1955	15 (1)	9 (1)	0 (0)	6 (0)

The down trend in incidence during the last three or four years will at once be noted, but is to be regarded with reserve as five new paediatric clinics were established in 1948 in near-by towns, and by 1952 or so these may have begun to deal with many patients who might formerly have been referred to Sheffield hospitals. To eliminate this factor it is necessary to exclude from present consideration all patients except those living within the city boundary; the numbers of the latter are shown in column 5 of the Table. The numbers of patients shown each year in column 5 are much smaller than those in column 2, but again a down trend in incidence during the last three or four years is shown. The 1955 figure of six patients a year is the lowest encountered in nine consecutive years and amounts to less than a quarter of the average incidence for these nine years. The recent down trend is believed to indicate a true decline in the incidence of the disease in the city.

Discussion

There is evidence, therefore, of less pink disease and less mercury ingestion among infants to-day than in 1948. There is also evidence that the decline in the incidence of the disease has taken place mainly during the last two or three years of the intervening period, and if it could be shown that the decline in mercury ingestion also took place mainly during the last two or three years then the case for the mercury hypothesis would be so much the stronger. There is no direct evidence on this point, but the circumstantial evidence is suggestive enough to be worth recording. Thus,

so far as teething powders are concerned, three of the most effective measures securing a reduction in mercury ingestion have been: (a) the action of the manufacturers of the two most popular brands (A and B) of powder in Sheffield in removing the metal from their products; (b) the recall by one of these firms of practically all old mercury-containing stocks from the retailers and a similar though less complete recall by the other; and (c) the action of the medical officer of health in arranging for members of his staff to visit retailers and to draw their attention to the possible dangers of selling mercury medicaments for babies. It cannot be ignored that these three measures were initiated at approximately the same time that the down trend in the incidence of the disease was first observed.

Conclusions

Mercury ingestion among infants and the incidence of pink disease have declined in Sheffield during the past nine years.

Yearly figures show that the down trend in the incidence of the disease has occurred only during the last three or four years, and there is circumstantial evidence that this was the period when the decline in the ingestion of mercury was mainly taking place.

These associations support the contention that the ingestion of mercury may be an important factor in the aetiology of the disease.

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POTASSIUM-LOSING PYELONEPHRITIS

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Two definite cases of potassium-losing pyelonephritis have been recorded (Brown *et al.*, 1944; Davidsen *et al.*, 1951), and one possible case (Sayers *et al.*, 1955). Milne *et al.* (1952) describe hypokalaemia developing in a case of hyperchloraemic renal acidosis with osteomalacia. Conn (1955), who describes renal potassium loss in a case of primary aldosteronism, is of the opinion that the cases described by Earle *et al.* (1951), Evans and Milne (1954), and Wyngaarden *et al.* (1954) suffered from primary aldosteronism. A further case report of another patient suffering from potassium-losing pyelonephritis is presented.

Case Report

A 51-year-old woman was admitted urgently on April 10, 1955, with a history of eight days' bilateral renal pain, polydipsia, polyuria, pyrexia, and frequent vomiting. Forty-eight hours before admission a marked generalized weakness developed without drowsiness, and for twenty-four hours she

was unable to raise her head from the pillow. She gave a past history of repeated attacks of infective parotitis since 1948.

Examination showed an extremely weak but normally orientated thin woman: weight, 44.5 kg.; blood pressure, 84/64; pulse, 80 a minute, regular; temperature, 98° F. (36.7° C.). Bilateral renal tenderness, especially marked on the left side, was present. Gross weakness of the limbs was evident; slight voluntary movement was possible in the arms, but the legs were completely flaccid. All tendon reflexes were present and equal; the cranial nerves were normal.

Clinically she was suffering from acute pyelonephritis; the urine was loaded with albumin and pus cells, and *Bact. coli* was later isolated. The white blood cell count was 60,800 per c.mm. (polymorphs 93%, lymphocytes 7%); blood urea, 160 mg. per 100 ml. A satisfactory response to streptomycin occurred, renal pain ceased, the urine cleared, and the blood urea fell to 43 mg. per 100 ml. in twelve days. The isolated *Bact. coli* was insensitive *in vitro* to the antibiotic used.

On admission a plasma electrolyte examination showed: sodium 135, potassium 1.2, chloride 106.3, and alkali reserve 8.9 mEq/litre. Even with this excessively low level of plasma potassium she was then excreting 13.9 mEq/l. in the urine. The hyponatraemic acidosis was corrected by 15 g. of sodium bicarbonate orally, and the plasma potassium level rose to 3.8 mEq/l. in 48 hours after ingestion of 20 g. of potassium (512 mEq) given as potassium chloride and potassium citrate. Consequent upon this improvement, the paralysis resolved and the patient was maintained on a normal diet to which 1.5 g. of potassium was added daily.

A recent but milder attack of acute pyelonephritis occurred 30 days after admission; it was accompanied by a milder hypokalaemia ($K=2.9$ mEq/l.) which resolved in three days as the infection cleared. No added potassium was given other than the maintenance dose.

The following investigations were carried out subsequently: abdominal x-ray examination revealed bilateral diffuse nephrocalcinosis; intravenous pyelogram showed normal excretion time, but not all the calices were visualized; urine concentration test, deprivation of fluids for 14 hours on two occasions resulted in a urine specific gravity of 1006 (unfortunately, longer deprivation of fluids was not attempted); urine dilution test, a marked response with a large diuresis; 9 g. of ammonium chloride failed to acidify a constant urinary pH of 7 and did not increase ammonia excretion. Van Slyke standard blood urea clearance tests were 17 ml., 16 ml., and 11 ml. a minute respectively, the blood urea being 29 mg. per 100 ml.; serum calcium, 8.6 mg. per 100 ml.; plasma inorganic phosphorus, 2.7 mg. per 100 ml.; serum alkaline phosphatase, 10 units per 100 ml. (King-Armstrong).

The patient returned to her sedentary occupation on discharge on June 10 and remained well, apart from one mild attack of parotitis, which was accompanied by some left renal pain. This attack subsided within 24 hours after sulphadimidine administration.

Further plasma potassium estimations performed when she was an out-patient were around 3 mEq/l.; the potassium intake was therefore raised to 3 g. daily in addition to her normal diet, and at the time of writing her plasma potassium levels were about 3.3 mEq/l. At no time has she suffered from prolonged diarrhoea.

Discussion

Nephrocalcinosis in this case would probably result from long-standing hyperchloraemic acidosis of renal origin, and the low normal serum calcium and normal plasma inorganic phosphorus concentrations support this. Two attacks of acute pyelonephritis were observed in hospital; subclinical attacks of pyelonephritis presumably occurred before her admission. It is possible that they may have originated from the parotitis; this may be further supported by the clinical course of the infection, which flared up after discharge.

Gross renal impairment is readily evident from the results of the renal function tests; the patient was rapidly approaching isosthenuria, although she could still respond by marked diuresis to a water load.

We believe that the potassium loss in this case is renal in origin. Primary aldosteronism, as described by Conn (1955), presents with a low plasma potassium, but also with a normal or raised plasma sodium, a raised plasma bicarbonate, and a raised plasma pH—that is, as a metabolic alkalosis with hypokalaemia—and also attacks of tetany. The case of Evans and Milne (1954) resembles this closely, and Cope and Milne (1955) agree with Conn (1955) that that case was probably one of primary aldosteronism, particularly as the urine contained an abnormally high concentration of electrocortin and the plasma hydroxycorticoids were raised.

Milne *et al.* (1952) described a case of Fanconi's syndrome with metabolic acidosis, hypokalaemia, amino-aciduria, and pseudofractures, and also a case of renal hyperchloraemic acidosis. This latter case resembles ours in that nephrocalcinosis was present, the urine was neutral, the plasma bicarbonate was low, and at the times of attacks of paralysis it was assumed that the plasma potassium was low (although in the intervals between attacks it was normal). The differences between the second case of Milne *et al.* (1952) and our present case are that in the latter there were no pseudofractures, the alkaline plasma phosphatase was normal, and hence there was no evidence of osteomalacia; also, on admission our case had a low plasma sodium concentration as well as hypokalaemia.

Although the patient responded rapidly to oral potassium when the acute pyelonephritis was controlled, she again developed hypokalaemia while taking additional potassium in hospital, when she developed a further attack of acute pyelonephritis. The rapid response in the plasma potassium level to fairly small doses of potassium again favours a renal lesion rather than primary aldosteronism.

No electrolyte balance studies were done, but when she was an out-patient on a high potassium intake repeated plasma estimations showed hypokalaemia. Thus we postulate that only a renal lesion could explain the clinical and laboratory findings in this case. This lesion may either have been present before the attacks of pyelonephritis or could possibly have developed as a result of it.

We are grateful to Dr. C. E. Kellett for permission to publish this case report.

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The National Association for Mental Health has issued a booklet entitled *Teaching the Mentally Handicapped Child to Speak*. The author, Adelaide Trainor, director of Speech and Drama, Northern School of Music, Manchester, explains that the purpose of the book "is not so much to offer instruction in the formation of speech sounds, etc., as to suggest a possible line of approach to the all-important subject of speech for the mentally handicapped." Advice is given on how to approach the silent child, the mongol child, and the child who is able to speak though indistinctly. At the end of the book are many rhymes to assist breathing, and for vowel or consonant practice, etc. This booklet, price 2s., is obtainable from Publications Department, N.A.M.H., 39, Queen Anne Street, London, W.1.