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THE OCCURRENCE OF LEAD POISONING AMONGST NORTH QUEENSLAND CHILDREN

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TWO CHARTS

INTRODUCTION

In 1892, Lockhart Gibson, Wilton Love, Jeffris Turner and others drew attention to a disease occurring amongst children living in the neighbourhood of Brisbane, which they attributed to lead poisoning.

The symptoms of the disease in the ten children mentioned was a paralysis of certain groups of muscles, namely, the extensors of the fingers, the tibialis anticus and the long extensors of the toes and the muscles which form the bulk of the thenar prominence, the abductor opponens and flexor pollicis brevis.

The electrical examination in three cases revealed a well-marked reaction of degeneration in the paralysed muscles. Besides the paralysis, an anaemia of varying degree was observed, the characteristic blue line in the gum was only rarely seen, and when present occurred opposite a few isolated teeth only. Pains in the limbs and spasmodic contractions of the calf muscles were most uncommon. None of the cases complained of colic, but four cases gave a clear history of gastrointestinal seizures and constipation. The diagnosis was based on the typical distribution of the palsy and the presence, in two cases, of distinct traces of lead in the urine after the commencement of the administration of potassium iodide. The question of the source of the poisoning was left in abeyance.

On the same occasion Jeffris Turner (1892) recorded four cases which lead him to the diagnosis of a localised meningitis. The children complained at first of headache and vomiting, and shortly afterwards an internal squint developed accompanied by a double optic neuritis, the outlines of the disc being totally obscured. The diagnosis of a slow-growing tubercle could be excluded, since pyrexia, convulsions and delirium were absent, and three of the four cases completely recovered whilst the fourth case improved slightly. The history and the course of the disease excluded syphilis, and localised basal meningitis of unknown origin was suspected.

Later, in 1897, Jeffris Turner divided the cases of lead poisoning in children into four groups, namely:

1. Paralytic cases showing symmetrical wrist and foot drop, spasms of the calf muscles, and as a secondary lesion a persistent talipes equinus.
2. Cases characterised by pains in the abdomen and limbs, concomitant with habitual constipation and an occasional blue line on the gums, the children becoming at the same time irritable and neurotic.
3. Eclamptic cases suffering from severe and persistent convulsions, which often end fatally.
4. Children suffering from ocular neuritis, a neuritis involving the optic and at the same time the oculomotor nerves.

Lead was found in the urine sometimes in minute traces, sometimes in appreciable quantities. According to Turner, seventy-six cases, fifty-five girls and twenty-one boys, were treated in the Brisbane Children's Hospital during six years, their ages varying between 6 and 12 years. As a number of the children who left the hospital apparently cured were re-admitted after an interval of a few months, the conclusion that the source of the poisoning was in the respective homes seemed beyond doubt. For drinking purposes, rain-water collected from the roofs in tanks had been used. The presence of lead in three different samples of tank water from houses where cases had occurred, examined after several months of dry weather, convinced the author that lead contained in the tank water was the source of the poisoning.

In the same year Lockhart Gibson (1897) published the histories

of a few cases suffering from double optic neuritis and simultaneously from paralysis of one external rectus. One of the children showed at the same time marked wrist drop and a tendency to ankle drop, another the blue line on the gums and traces of lead in the urine, and a less severe case distinct traces of lead in the urine, of which two specimens were examined. Lead poisoning was therefore thought to be the cause of the eye symptoms. The number of cases (twenty-four since 1891), the discovery of lead in the urine, the occurrence of colic, lead palsy, occurring simultaneously with the eye symptoms, the uniform absence of fever, the fact that no case which exhibited the symptoms attributed to ocular neuritis ended fatally, seemed to prove beyond doubt that lead was the cause of the disease in question, and tank water was considered as the source from which the lead was obtained. The fact that as a rule only one child of the household was attacked was attributed either to an increased sensitiveness of particular nerves to the lead, circulating in their capillary network, or to a deficient excretion of lead.

Three cases suffering from similar clinical symptoms, which ended fatally, were described by Green (1897). The pulse of these cases was rapid, the temperature above 100, rising to 105 before death. The post-mortem held on one showed an interstitial nephritis and a congestion of the cortex of the brain.

In 1899, Jeffris Turner amplified his observations. Lead, according to him, appeared to be only occasionally present in the urine, and then as a rule in extremely minute quantities. The characteristic sign of lead poisoning, the blue line on the gums, commenced as minute blackish dots opposite certain teeth only. He drew attention to the intense mental irritability and to the rapid wasting observed in severe cases, and to the common occurrence of pains in the limbs as well as constipation.

In 1904, Lockhart Gibson reported four cases of lead poisoning in children, with ocular neuritis. In two of them lead was found in the urine, 0·4 mg. and 0·32 mg. per litre urine respectively. Further observations convinced him that tank water could be practically excluded, and the lead paint of the walls and verandah railings was substituted as the source of infection.

In his conclusions he pointed out that the majority probably

all, of the children affected with lead poisoning had lived in houses in which the rooms, or at least the verandah railings, had been painted with lead paint.

Two conditions of painted surfaces would be more than usually liable to induce poisoning, viz.:

(*a*) Freshly painted or at least sticky surfaces.

(*b*) Painted surfaces which either by exposure to the sun and air, or for other reasons, had lost their oil and the paint had become a dry powder which was easily detached when rubbed, and intermingled with the dust of the room.

Dust, of course, is capable of being both swallowed and inhaled. The greatest danger, in his opinion, was the adhesion of the paint either by nature of its stickiness or of its powdery character to the fingers and nails, whence it is conveyed to the mouths of children, especially in those who bite their nails, suck their fingers, or eat with unwashed hands.

A further danger, in his opinion, worthy of consideration, was the absorption of the soluble lead salts contained in paint by the skin of the children's hands and feet.

He further quoted the case of two children who developed typical signs of lead poisoning shortly after having moved into a new house recently painted, and these two children were, according to the mother's statement, the only members of the family addicted to biting their nails.

An inquiry was therefore begun amongst the parents of undoubted lead cases—twelve in number—and their answers, ten in number, elicited the information that eight of the children were in the habit of biting their nails or sucking their fingers.

It was found by consulting the records of the Children's Hospital in Brisbane that from 1898 to 1903 eighty-five cases of lead poisoning had been treated as in-patients in the hospital, and of these forty-two were admitted during the months of December, January and February—the hottest months; twenty-eight were admitted during October, November, March and April; and sixteen only were admitted during the five cooler months.

A further contribution to this question was Dr. Jeffris Turner's Presidential Address on 'Lead Poisoning in Childhood,' which sums up the question up to the year 1909. Clinically he

distinguishes cases with gastro-intestinal symptoms, with paralysis of the muscles of the limbs, with diaphragmatic and cardiac paralysis, with remote effects as anaemia, albuminuria, with eclampsia, acute optic neuritis associated with oculomotor paresis, and finally with chronic optic neuritis and atrophy without paresis of the sixth nerve.

Lockhart Gibson (1913) recommends the lumbar puncture, in all cases of lead poisoning where eye symptoms are prominent, as the best mode of treatment, since in his experience there was, in the majority of cases, a distinct increased tension of the cerebro-spinal fluid, and lumbar puncture seemed to relieve the paralytic symptoms considerably.

OBSERVATIONS IN TOWNSVILLE

Opportunity offered itself in Townsville to observe cases resembling those described by Jeffris Turner, Lockhart Gibson, and others in children admitted to the hospital.

Unfortunately during the first three years no chemical analysis of the urine for lead could be carried out. As the characteristic signs of lead poisoning, the blue line on the gums and drop-wrist, were only rarely met with, and as in the first cases basophilic granulations in the erythrocytes, which are generally considered as characteristic of lead poisoning, were absent from the blood, considerable doubt was felt about lead being the etiology of the cases in question. The chemical analyses of the cases previously reported in the literature seemed incomplete, and moreover, only in a few of the cases any reference at all was made to the presence of lead in the urine. Besides, no urine of normal children had been examined for the presence of lead, and in no case had any analysis of the faeces been made.

The clinical symptoms alone were barely sufficient to warrant the diagnosis of lead poisoning, especially as our knowledge of the clinical picture of lead poisoning in children is very incomplete.

Within the last year, however, the urine and faeces of a number of children clinically suspected as suffering from lead poisoning have been examined chemically, and the results have proved beyond doubt that lead poisoning is not of uncommon occurrence among children. During 1913 the excreta of twenty-two

children were examined for the presence of lead, and in eighteen of these lead was found in varying quantities.

The symptoms observed corresponded, on the whole, to those previously described.

The cases may be classed according to the severity of the symptoms. The very early cases have no definite symptoms. Parents complain of a change in the character of the children within the last few weeks; whilst previously they had been normal and well-behaved, lately they had become peevish and fretful, were very restless at night, and had lost their appetite completely. As a rule only careful cross-questioning elicited the information that the child had been complaining of slight pains in the epigastrium and pains in the legs. The breath was often foul, and now and again ulceration was noticed in the mouth. According to the parents' statements, the children were flushed in their faces and appeared feverish. All the symptoms referred to are very indefinite and may be considered as the commencement of any infectious disease.

Even in these early cases lead can be found in the urine often in appreciable quantities, and without the chemical analysis the correct diagnosis would be very difficult.

In cases of slightly longer duration the pains in the abdomen, intermittent at first, have become more continuous. Constipation is the rule, but in rare cases a history of intermittent diarrhoea is given. Vomiting is only rarely observed. The pains in the calf of the legs become more marked, the calf muscles are often so painful that the little patients cannot endure even the weight of the bed-clothes, and at this stage the gait of the child becomes affected.

The Burtonian line was observed only in a small percentage of the children. This 'blue-line,' due to the interaction of lead salts which have gained access to the mouth, and the sulphuretted hydrogen produced by the decomposition and putrefaction of food, is at present regarded 'of no more value than that the person showing such pigmented gums has been exposed to lead absorption.' (Legge and Goadby, 1912.)

A continuous line is not often found in children, but the characteristic minute black spots occur around teeth which are coated with a deposit of tartar, requiring the use of a hand-lens for their diagnosis.

Many of the cases showed foot- and wrist-drop. As pointed out by other observers, certain nerves of the legs are as a rule affected first, thus differing from the clinical symptoms of lead poisoning in adults, where drop-foot is but rarely present.

The extensors of the toes and the peronei muscles are most frequently involved. The patients have a characteristic gait, they walk on the outside of their feet, the gait is high-stepping, the toes are dragged along the ground, and at each step the legs are swung sideways before again being put down to the ground.

Drop-wrist due to a paralysis of the extensor communis digitorum is not rare. In two advanced cases the Aran-Duchenne type of paralysis was present, in which the muscles of the thenar and hypothenar eminences and the interossei showed marked atrophy. In the majority of cases with nervous symptoms a slight atrophy of the muscles in question was noticeable.

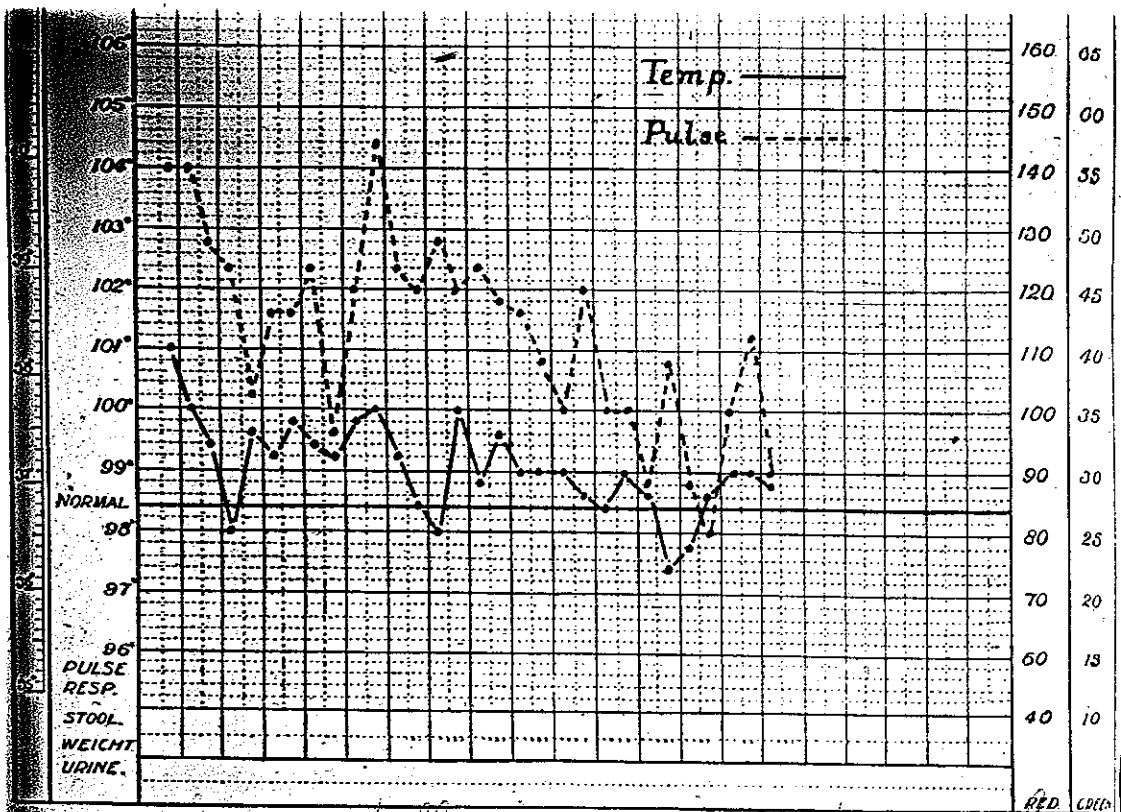
The eye-symptoms described by Lockhart Gibson were only present in one out of the twenty-two cases examined. This child complained of dimness of vision, but an examination of the fundus was impossible on account of the restlessness of the child.

Brain symptoms are common, ranging from obstreperousness and fretfulness to a state resembling mania. As already remarked, the parents invariably complain of a marked change in the character of the previously well-behaved children. In two extreme cases the children had become quite unmanageable since their illness. They screamed day and night, and kicked at everybody who came near them. Shortly after their removal from home, and after a vigorous treatment with potassium iodide, these children again became quite normal.

Not rarely convulsions are the outcome of chronic lead poisoning in children, and these do not differ in any way from convulsions due to other causes, except in their severity. In one case the convulsions, after admission to the hospital, continued for three-quarters of an hour, and only abated after the administration of large doses of chloral hydrate and bromides.

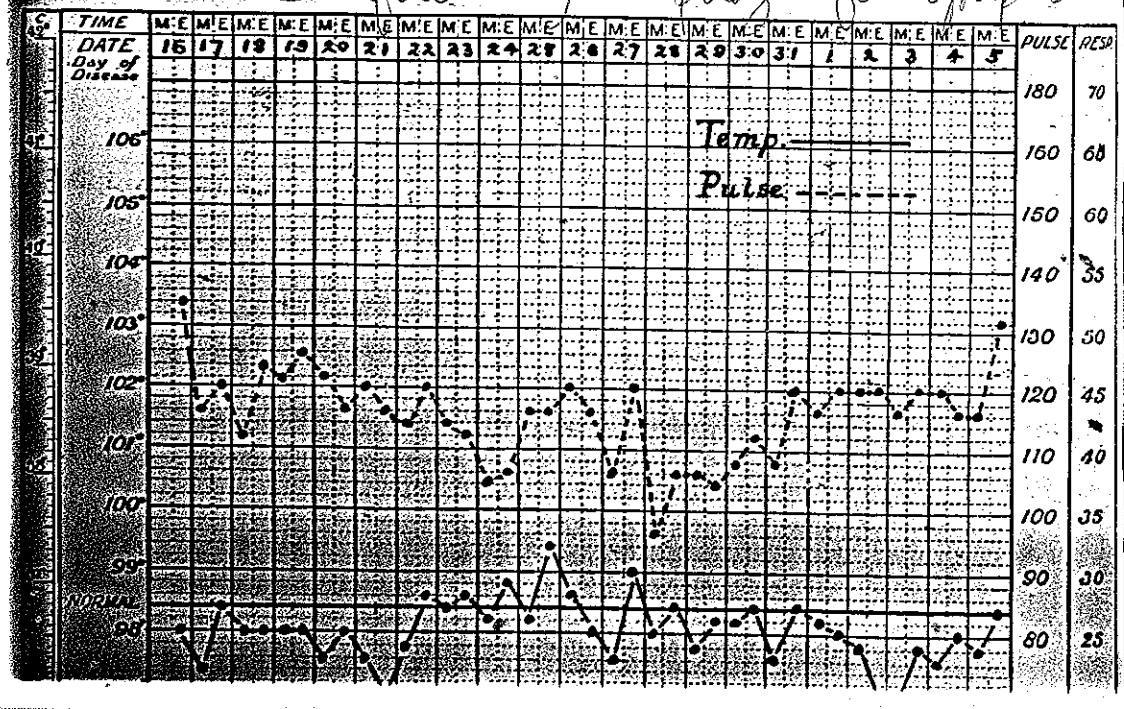
Albumen was not found in the urine of any of the cases.

The body temperature is often normal throughout, sometimes irregular, but very rarely rising above 101° F. (see Chart 1). The pulse-rate is invariably high in spite of normal temperature (comp. Chart 2). Respiration is normal.



See following pages for graphs

CHART I.



Anaemia is only marked in cases of long standing. Blood counts in early cases reveal normal conditions; in chronic cases the number of red cells may be diminished; the lowest count gave 3,000,000 red blood corpuscles with a haemoglobin of 9·34 grams in 100 c.cm. blood.

Basophilic granulations in a small number of erythrocytes were found in the majority of cases, sometimes only after a prolonged search.

COMPARISON OF THE SYMPTOMS OF LEAD POISONING IN CHILDREN AND ADULTS

The clinical symptoms of lead poisoning in children differ in many ways from plumbism in adults.

The occurrence of the blue line and colic is common to all ages. Whereas in adults the palsy affects the upper limbs most frequently, and the lower limbs only in a proportion of 13 to 100 (Tanquerel, 1913), in children the first symptoms of paralysis appear in the lower limbs, the arms becoming affected later. Cerebral symptoms resembling a mild form of mania are not at all uncommon in any age.

The pulse in lead poisoning in adults, according to Legge and Goadby (1912), may be increased during the early stages only. In children a quick and small pulse is the rule.

The occurrence of eye symptoms such as optic neuritis is an exception in adults (Legge and Goadby), whereas in children eye symptoms in the form of oculomotor neuritis are common (Lockhart Gibson).

The mucous membrane of the mouth and gums in children is often inflamed, and shows inclination to formation of sores.

In the same way as adults, children are prone to relapses in cases where they have returned to the locality where they first became affected, and many such children are readmitted to the hospital at irregular intervals.

PROGNOSIS

The prognosis of lead poisoning in children in the early stages is, on the whole, good. The colic becomes less violent and the pains in the legs disappear completely. As in adults, the complain

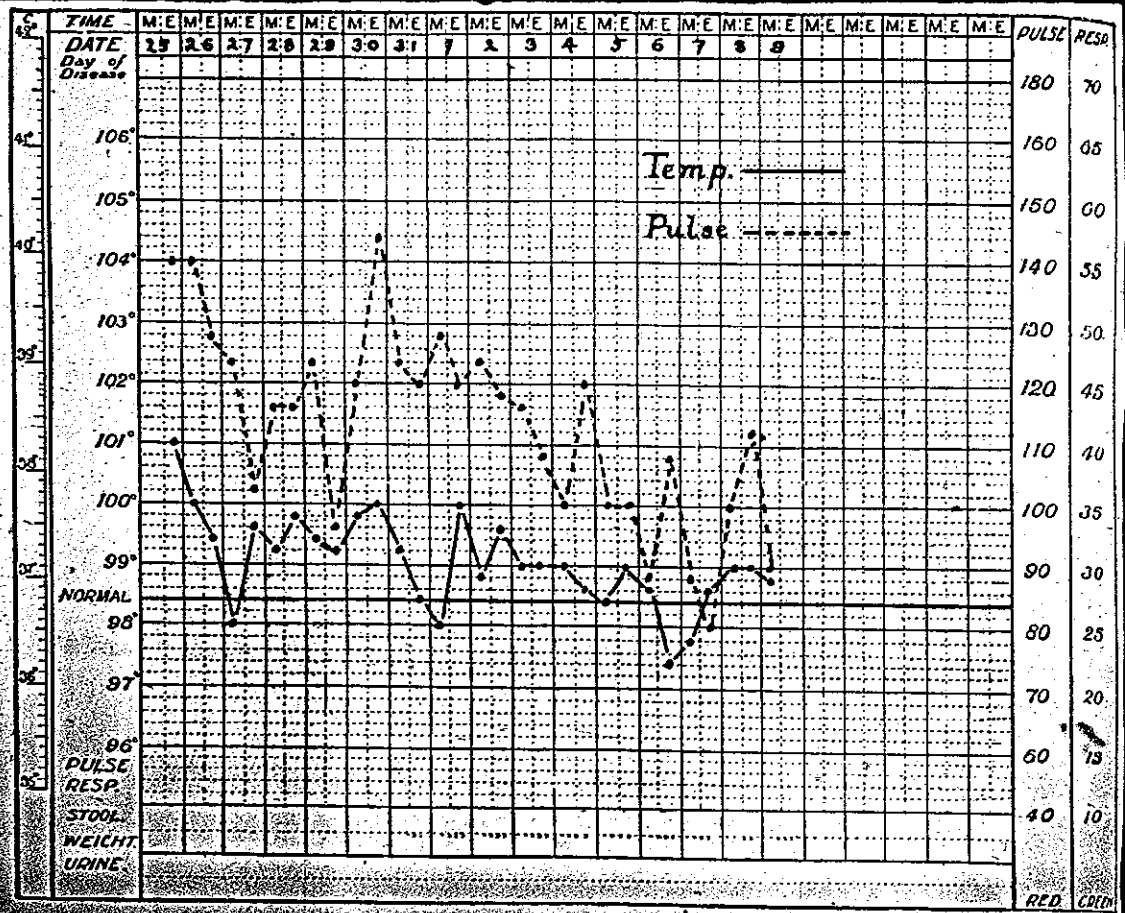
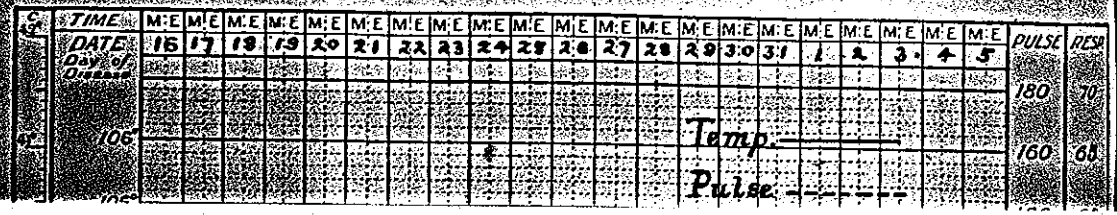


CHART I.



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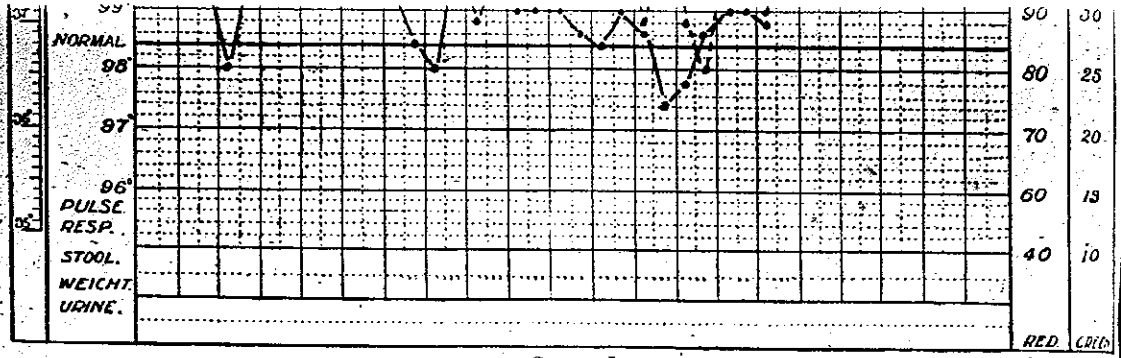

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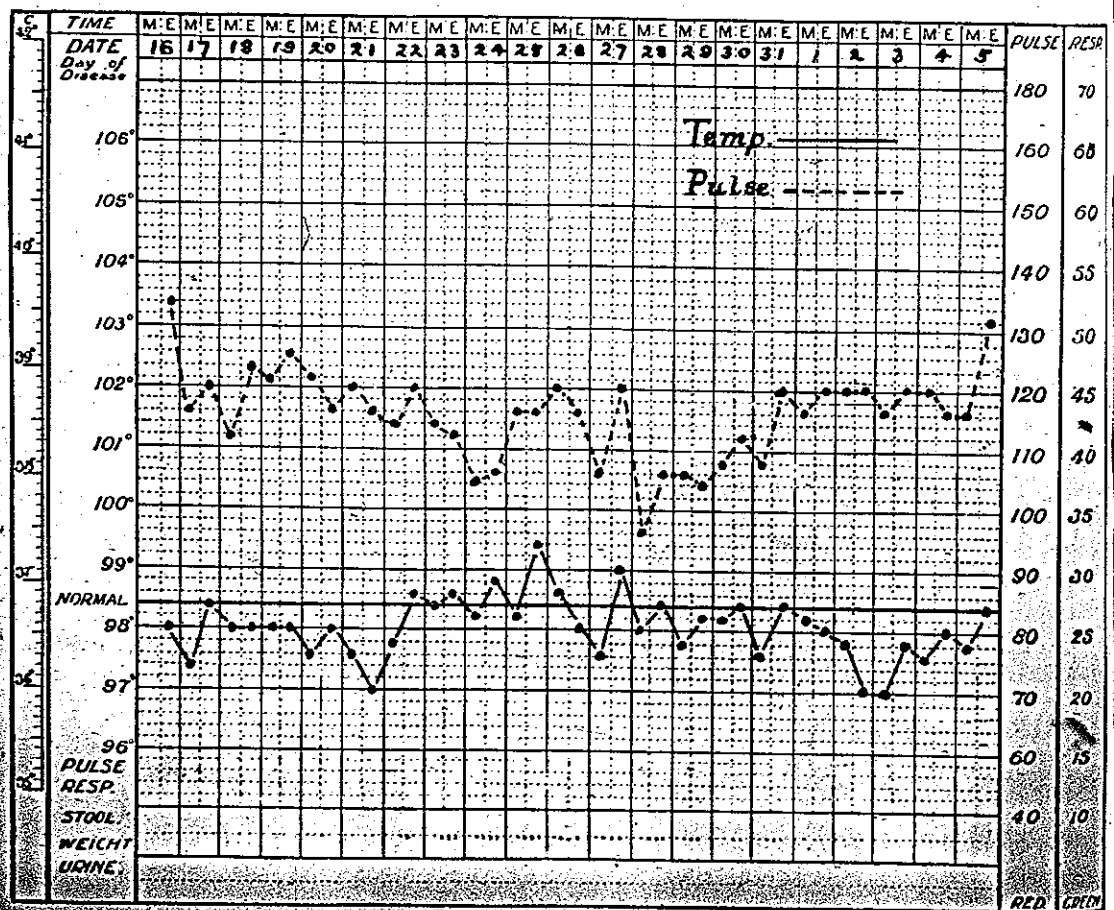

CHART II.

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PROGNOSIS

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becomes more serious with the second and third attack. Whereas children treated in the early stages recover the use of their limbs completely in the course of a few weeks, in the latter stages it requires months before they are able to walk. In many instances the patients remain crippled for life, in spite of careful treatment.

The slight mental disturbances so marked in the early cases abate with the general improvement, and even the severe cerebral symptoms, such as convulsions and mania, disappear after treatment for a comparatively short time.

CHEMICAL EXAMINATION OF THE EXCRETA

The urine and faeces of cases clinically suspected of lead poisoning were examined chemically. Although lead is often present in the urine of cases which have been exposed to lead absorption, especially after the administration of potassium iodide, the results of numerous investigations have shown that this is not invariably the case, and that even when lead is present it only occurs in small amounts.

Thus Legge and Goadby (1912) state that 'the presence of lead in the urine is not so common nor so definite a symptom as might be supposed,' whilst Jaksch (1901) goes so far as to assert that no lead at all is excreted in the urine of patients suffering from chronic lead poisoning, but only in the acute stages. Dixon Mann (1908) described a similar case in which during treatment with potassium iodide the excreta were analysed separately, with the result that much larger quantities of lead were found in the faeces than in the urine. When lead salts were administered to the same patient, the bulk of the lead appeared in the faeces. These observations led him to the conclusion that lead salts are mainly excreted into the intestine and eliminated with the faeces. Experiments carried out by Legge and Goadby and other investigators confirmed this view.

It appeared therefore desirable to examine the faeces as well as the urine, and the results obtained justified this proceeding. In every case a larger quantity of lead was found in the faeces, and lead was often present in these when no trace could be detected in the urine.

For the chemical analysis the material was treated in the

following manner: The organic matter in the urine and faeces was oxidised by the well-known method of Fresenius and Babo with hydrochloric acid and potassium chlorate. The resulting liquid was evaporated to dryness, and the main excess of hydrochloric acid removed by repeatedly adding distilled water and evaporating down. This was then almost neutralised with sodium bicarbonate, filtered hot, and the faintly acid solution saturated with sulphuretted hydrogen. Any lead sulphide was filtered off, washed with sulphuretted hydrogen water, and dissolved by repeatedly evaporating to dryness; the residue was taken up in water, and the lead estimated colorimetrically. The brown colour given on addition of sulphuretted hydrogen water in the presence of potassium hydrate was compared with that given under similar conditions by a standard solution of lead nitrate. When the quantity of lead present exceeded one milligramme, it was re-dissolved in nitric acid and estimated gravimetrically as lead sulphate.

In every case confirmatory qualitative tests for lead were made on one sample of the solution in nitric acid obtained as above. The tests employed were the yellow precipitate with potassium chromate, white precipitate with sulphuric acid insoluble in cold nitric acid and soluble in ammoniacal ammonium tartrate, and the formation of microscopical violet-black cubical crystals of copper lead nitrate when the solution was mixed with copper acetate and potassium nitrate and allowed to evaporate.

The residue of fat and unoxidised matter which was filtered off after the oxidation with hydrochloric acid and potassium chlorate would contain any lead sulphate which might be present in the faeces or urine. It was therefore treated with hot ammoniacal ammonium tartrate, and the filtered liquid evaporated to a small bulk, acidified with hydrochloric acid, and saturated with sulphuretted hydrogen. In no case did this residue contain more than a trace of lead.

The chemicals used in the analysis were carefully tested for lead, and only those free from lead were employed. Blank experiments were also carried out with normal children's urine in the vessels actually employed in the operations, and these yielded negative results throughout.

The quantities of lead obtained are given in Tables 1-3, the cases being classed according to the severity of the symptoms. In some instances the total excretion of urine and faeces during several definite periods were analysed separately. In others, however, urine was collected until a quantity sufficient for analysis (at least 1000 c.c.) had been obtained, and since the quantities of lead found do not represent the excretion over any definite period, the amount of lead per 1000 c.c. of urine is given.

TABLE 1.—Early cases in which the only symptoms were pains in the legs and epigastrium.

Particulars of Case.	Date.	Material Examined.	Lead Found.
1. G.M.—Girl, 12 years old... Potassium iodide commenced 29.2.13	25.6.13	1 day's urine	0·23 mgm.
	28.6.13	3 days' urine	0·70 ,,
	1.7.13	3 days' urine and faeces	3·2 ,,
	Dec., 1913	Urine	None.
2. R.D.—Boy, 13 years ... Before potassium iodide	29.7.13	2 days' faeces	6·8 mgm.
	30.7.13	1 day's urine	0·6 ,,
	1.8.13	1 day's faeces	0·2 ,,
	14.8.13	1 day's urine	None.
		1 day's faeces	Trace.
3. B.Y.—Girl, 4 years ... No potassium iodide ...	2.7.13	Urine.	0·32 mgm. per litre.
		1 day's urine.	0·43 ,,
4. J.H.—Boy, 10 years ...	21.7.13	Urine.	0·18 mgm. per litre.
		Faeces.	0·32 mgm.
5. H.H.—Girl, 9 years ...	16.8.13	Two stools.	1·7 mgm.
6. D.S.—Girl, 9 years ...	18.10.13	Urine.	0·32 mgm. per litre.
7. H.H.—Boy, 9 years ...	5.11.13	Urine.	0·24 mgm. per litre.
	6.11.13	Two stools.	2·4 mgm.
8. E.W.—Boy, 6 years ...	20.10.13	Urine.	0·09 mgm. per litre.

TABLE 2.—Cases with paralytic symptoms. (Drop wrist, drop foot.)

Particulars of Case	Date	Material Examined	Lead Found
9. D.O'B.—Girl, 4 years ... Receiving potassium iodide.	15.2.13	Urine.	0·37 mgm. per litre.
	28.2.13	Urine.	0·65 ,,
	2.3.13	2 days' urine	0·40 mgm.
	4.3.13	2 days' urine.	0·30 ,,
	6.3.13	2 days' urine.	0·35 ,,
10. E.B.—Girl, 6 years ... Treatment with potassium iodide commenced on 28.2.13 (Compare Chart 2.)	27.2.13	1 day's urine.	0·48 mgm.
		1 day's faeces.	1·27 ,,
	1.3.13	2 days' urine and faeces	3·2 ,,
	2.3.13	1 day's urine	None
		1 day's faeces	0·50 mgm.
	3.3.13	1 day's urine and faeces	1·3 ,,
	4.3.13	1 day's urine and faeces	0·50 ,,
	5.3.13	1 day's urine and faeces	0·42 ,,
	6.3.13	1 day's urine and faeces	1·9 ,,
	8.3.13	2 days' urine and faeces	0·22 ,,
	10.3.13	2 days' urine and faeces	1·7 ,,
11. M.R.—Girl, 3 years ... No potassium iodide	20.1.14	Urine	0·34 mgm. per litre.
	21.1.14	Two stools	1·2

TABLE 3.—Cases showing pronounced cerebral symptoms.

Particulars of Case	Date	Material Examined	Lead Found
12. E.McC.—Girl, 3 years ... Receiving potassium iodide	7.6.13	1 day's urine and faeces	1·9 mgm.
	10.6.13	3 days' urine and faeces	4·6 ,,
	12.6.13	2 days' urine and faeces	1·2 ,,
13. T.E.—Boy, 14 years ... ... No potassium iodide	9.1.14	Urine	0·49 mgm. per litre.
14. R.E.—Boy, 6 years ... No potassium iodide	28.12.13	Urine	Trace only in litre.
	30.12.13	Urine	0·45 mgm. per litre.
15. M.—Girl, 6 years ... No potassium iodide	23.8.13	One stool	0·4 mgm.
	25.8.13	1 day's urine and faeces	0·30 ,,
16. L.H.—Boy, 4 years ... No potassium iodide	7.12.13	Sample urine	

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	Dec., 1913	Urine	None.
2. R.D.—Boy, 13 years ... Before potassium iodide	29.7.13	2 days' faeces	6·8 mgm.
	30.7.13	1 day's urine	0·6 ,,
	1.8.13	1 day's faeces	0·2 ,,
	14.8.13	1 day's urine	None.
		1 day's faeces	Trace.
3. B.Y.—Girl, 4 years ... No potassium iodide ...	2.7.13	Urine.	0·32 mgm. per litre.
		1 day's urine.	0·43 ,,
4. J.H.—Boy, 10 years ...	21.7.13	Urine.	0·18 mgm. per litre.
		Faeces.	0·32 mgm.
5. H.H.—Girl, 9 years ...	16.8.13	Two stools.	1·7 mgm.
6. D.S.—Girl, 9 years ...	18.10.13	Urine.	0·32 mgm. per litre.
7. H.H.—Boy, 9 years ...	5.11.13	Urine.	0·24 mgm. per litre.
	6.11.13	Two stools.	2·4 mgm.
8. P.W.—Boy, 6 years ...	29.10.13	Urine.	0·69 mgm. per litre.

TABLE 2.—Cases with paralytic symptoms. (Drop wrist, drop foot.)

Particulars of Case	Date	Material Examined	Lead Found
9. D.O'B.—Girl, 4 years ... Receiving potassium iodide.	15.2.13	Urine.	0·37 mgm. per litre.
	28.2.13	Urine.	0·65 ,,
	2.3.13	2 days' urine	0·40 mgm.
	4.3.13	2 days' urine.	0·30 ,,
	6.3.13	2 days' urine.	0·35 ,,
10. E.B.—Girl, 6 years ... Treatment with potassium iodide commenced on 28.2.13 (Compare Chart 2.)	27.2.13	1 day's urine.	0·48 mgm.
		1 day's faeces.	1·27 ,,
	1.3.13	2 days' urine and faeces	3·2 ,,
	2.3.13	1 day's urine	None
		1 day's faeces	0·50 mgm.
	3.3.13	1 day's urine and faeces	1·3 ,,
	4.3.13	1 day's urine and faeces	0·50 ,,
	5.3.13	1 day's urine and faeces	0·42 ,,
	6.3.13	1 day's urine and faeces	1·9 ,,
	8.3.13	2 days' urine and faeces	0·22 ,,
	10.3.13	2 days' urine and faeces	1·7 ,,
11. M.R.—Girl, 3 years ... No potassium iodide	20.1.14	Urine	0·34 mgm. per litre.
	21.1.14	Two stools	1·2

TABLE 3.—Cases showing pronounced cerebral symptoms.

Particulars of Case	Date	Material Examined	Lead Found
12. E.McC.—Girl, 3 years ... Receiving potassium iodide	7.6.13	1 day's urine and faeces	1·9 mgm.
	10.6.13	3 days' urine and faeces	4·6 ,,
	12.6.13	2 days' urine and faeces	1·2 ,,
13. T.E.—Boy, 14 years ... No potassium iodide	9.1.14	Urine	0·49 mgm. per litre.
14. B.E.—Boy, 6 years ... No potassium iodide	28.12.13	Urine	Trace only in litre.
	30.12.13	Urine	0·45 mgm. per litre.
15. M.—Girl, 6 years ... No potassium iodide	23.8.13	One stool	0·54 mgm.
	25.8.13	1 day's urine and faeces	0·30 ,,
16. L.H.—Boy, 4 years ... No potassium iodide	27.12.13	Sample of faeces and urine mixed	2·1 mgm.

The results of the chemical analysis show that the quantity of lead excreted is not always in direct proportion to the clinical symptoms of the case. It is thus not the amount of lead excreted, but the amount of lead retained in the body, which regulates the severity of the symptoms.

SUSCEPTIBILITY

Just as with adults, the individual susceptibility of children to lead poisoning shows great variation. It often happens that only one child out of a family of several develops symptoms of lead poisoning, although all have been exposed to the same degree of resorption.

Previous observers in Queensland have noted that where one child in a family is suffering from pronounced lead poisoning one or two of the others occasionally show only the blue line on the gum.

From this point of view in two instances chemical examination was made of urine of the brothers or sisters of the patient. In one case the urines of a baby and of the brother (approximately of the same age as the patient) living in the same house were analysed; the former was quite free from, and the latter showed traces of, lead. In the other case the urine of the brother was examined on two different occasions, and 0·6 mg. and a trace of lead respectively were detected.

In the light of these results the question arises as to whether lead in small quantities may be found normally in the urine of children living in houses coated with lead paint, clinical symptoms appearing only in those particularly susceptible to the poison. The complete failure to detect lead when the urine of a number of healthy children living under similar conditions was analysed, disproves this conception.

SOURCE OF THE LEAD

As to the source of the poison, Lockhart Gibson, quoted above, considered ingestion of paint the most likely one, although he recognised the possibility of dust containing lead entering the body by inhalation.

Experiments bearing on this question were carried out by Goadby and Goodbody (1909) and by Legge and Goadby (1912) which made it evident that the danger of lead inhaled as dust is many times greater than that of lead swallowed, and that the ratio of poisoning viâ the lungs to poisoning viâ the intestinal canal is 100 : 1.

A limited number of experiments in the same direction have been carried out on monkeys in this laboratory. Two monkeys were fed with white lead paint as used for house decoration. Varying small quantities were administered every few days in the form of pills, or were smeared over the tongue or mucous membrane of the cheeks for about four months. Neither of the animals was the worse for the treatment.

These experiments suggest the probability that the inhalation of dust containing lead may be the more common source of poisoning in children, although observations here coincide with those of Lockhart Gibson, namely, that a great number of the children affected were addicted to biting their finger-nails.

Further experiments are, however, required before a definite pronouncement as to the way by which the lead enters the body can be made.

It is striking that cases similar to those described above should not have been recorded from other parts of the tropics where lead paint is employed. Whether this be due to local conditions prevalent in Queensland only, to the difficulty of diagnosis, or to the lack of a definite clue associating such symptoms with lead, remains to be seen.

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