the malignant process. I am surprised to hear his suggestion that connective tissue is developed. My experience, as I have said before, is that radium seems to gelatinize any connective tissue with which it may have come in contact.

Redasens says to avoid serious infection, he treats the cancer with a 1% solution of copper sulphate before applying the radium: he douches the vagina with a 1% solution. Of course, we realize that there are different forms of cancer which are less amenable to treatment by radium. But the papillary form of cancer is more demure.

Now, the last part of my paper I consider is by far the most important. We have here in our hands a means of curing some forms of cancer, a means of improving the condition of other forms of cancer, at any rate for a certain time, a means which, if not curative, at any rate will alleviate in some forms of cancer many of the most distressing symptoms, pain, bleeding and hæmorrhage. Associated with surgery it is the greatest ally surgeons have ever had in those dreadful cases. The price of radium limits its purchase to a fortunate few who are sufficiently wealthy to invest in its purchase. I do not really know how many of the public hospitals have a supply. I believe the Women's Hospital has purchased a certain amount, but not enough to make extensive observations and to help the indigent poor. We are entirely dependent upon the graciousness of a practitioner to lend his own private supply for hospital use. When radium is being taken to the public hospitals from time to time, there is always a risk of it being lost or stolen. It would be a great monetary loss to the owner. Had it not been for the gracious and kindly help of Dr. Herman Lawrence, I should have had no opportunity of making these investigations. He never failed me; he regulated the dosage and he indicated where best to apply the different parts of radium. We generally arrange for week-ends to apply radium, when he is not so likely to require his supply as he would during the other days. I cannot say enough about the kindness and generous help which Dr. Lawrence and with him his assistant, Dr. Colquhoun, have always given me.

But here comes my final point. It is not right that we should have to apply to medical men to help us at the general hospitals with their own private supply. There should be a central supply available for all the hospitals financed by the Government and under Government control. By applying to the radium bureau a trusted officer would convey the radium to the operator and collect it again when the application was completed. Or a radium bureau could be established by wealthy philanthropists and could be conducted and controlled in much the same manner. The cost to provide radium for each individual metropolitan hospital and country hospital would be very great, whereas the one set of radium, I am sure, at a less cost could be made quite effective.

Now, gentlemen, do not you think that it is quite time that we made our voices heard and made them carry far and wide to the Government and to the general public, urging what are our great requirements? I think we have quite enough evidence to show that, at any rate in some cases, we can effect a cure and, if not in all cases, at any rate we are knocking at the door and with a little more investigation may at last open that door. I want you all to consider these remarks seriously and to see if the Government cannot be forced to help us.

AN HISTORICAL ACCOUNT OF THE OCCURRENCE AND CAUSATION OF LEAD POISONING AMONG QUEENSLAND CHILDREN.

DRAWN UP FOR AND ENDORSED BY THE COUNCIL OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

WE propose to give, firstly, a brief account of the symptoms of the affections which we attribute to lead poisoning among Queensland children and our reasons for this diagnosis and, secondly, to discuss briefly the source of the poison and our reasons for believing that this is fully ascertained and easily removable by legislation.

The first cases were recorded in 1892;[1] they were ten in number and occurred among children admitted into the Hospital for Sick Children, Brisbane, in 1891 and 1892. Reference is made to another case, not included in the report, which was admitted early in 1890. Naturally the cases which first attracted attention were those which showed peripheral paralyses with degenerative electrical reactions and wasting of muscles. In all ten cases three groups of muscles were affected:

(1) Extensors of the fingers in all cases, the extensors of the wrists in one only. Consequently, with the fingers extended there was wrist drop, but with the fingers clenched the wrist, except in that one case, could be extended. The *supinator longus* was never paresed (compare Gowers's "Diseases of Nervous System," 1888, article on lead poisoning, page 871).

(2) The small muscles of the hand were wasted and paresed, the *adductor opponens* and *flexor brevis pollicis* always, sometimes the adductor, hypothenar muscles and interossei. These muscles appeared to suffer from what Gowers terms the "primary atrophic" form of paralysis (*ibid.*, page 873).

(3) Paralysis, usually with wasting of the *tibialis anticus* and *extensor longus digitorum pedis*, was present in all cases. In one there was some affection of the peronei.

The paralyses were always of gradual onset and their duration varied from six weeks to several months before admission. In two cases there was a history over several years, the first attack having been recovered from and succeeded by a second after long intervals.

In nine cases the leg muscles were affected to a greater extent than those of the arm. This puzzled us, until we found that J. J. Putnam (in Kearing's "Cyclopædia of Diseases of Children") had recorded this as characteristic of lead palsy in childhood.

The electrical reactions were tested in three cases. In all faradic irritability was lessened or abolished.

Sometimes galvanic irritability was slightly increased, the anodal closure disproportionately, so as to equal the kathodal closure, or the irritability to kathode was diminished, so as to be about equal to that of the anode, or again the kathodal irritability was diminished, so as to be distinctly less than the anodal, which was normal or less than normal.

These extensor paralyses below knee and elbow were considered attributable to peripheral neuritis. Such possible causes as beri-beri, alcoholism, arsenic poisoning were thought of, but no evidence could be found pointing that way. The symptoms associated with the paralyses were consistent with plumbism. They were anæmia more or less marked, pain in the affected limbs, colicky attacks and the presence of a blue line on the gums in some of the cases. In two cases the urine, after the administration of a few doses of iodide of potash, was analysed by two independent observers and found to contain small but distinct traces of lead.

The subject was reopened in 1897.[3] So far as the paralytic cases were concerned, former observations were confirmed. It was noted in accordance with American experience of plumbism in children, that in every case foot-drop appeared before and disappeared later than wrist-drop. Paralysis of the peronei had been occasionally observed, also spasm of the calf muscles and secondary *talipes equinus*. In two cases there had been paralysis of the diaphragm. It was found possible to recognize a prodromal stage of poisoning before paralysis appeared, characterized by attacks of colic, often with vomiting, relieved by pressure and usually accompanied by constipation, often also with pain and tenderness or severe cramps in the leg muscles. Sometimes a blue line would be observed in these early cases. Severe attacks of convulsions, sometimes fatal, had been observed in many of the paralytic cases. New ground was broken by the recognition of a group of cases with ocular symptoms. In 1892 four cases were described[2] as a definite clinical group characterized by symptoms of cerebral irritation—headache, vomiting—with intense optic neuritis and paralysis of external recti. Three cases recovered completely, the fourth was removed from observation. It was suggested that the cause might be a localized basic meningitis. In 1897 twenty-four cases were reported[3] in children between the ages of four and eight. These cases were remarkable for the excessive swelling of the optic disc and paralysis of one or both external recti in every instance. In two there was paralysis of all the external ocular muscles and in one of these ptosis. In the more severe cases the head was slightly retracted and the neck muscles stiff. All the patients recovered, but seven became blind; in two vision was impaired, in the remainder it appeared normal. Only one patient had had a previous attack of wrist-drop and foot-drop. Some of the patients had suffered from colic. In several a blue line was observed on the gums. In several lead was found in the urine. The conclusion arrived at was that these cases were due to plumbism affecting the brain and ocular nerves. In some cases of plumbism albuminuria was observed and in one fatal case there appeared to be actual nephritis.[4] Most of the cases observed were in Brisbane children; but cases had occurred in Charters Towers, Gympie and Southport.

In 1899 cases were recorded also from Maryborough, Ipswich and Toowoomba and two cases were exhibited which had originated in New South Wales.[6] In a paper published in this year[7] no important facts are added, but the "blue line" is discussed. This is rarely conspicuous in children. It consists, when observed by a lens, of confluent dots inside the gum opposite, perhaps, one or two teeth only. Its absence does not negative the diagnosis of plumbism. Rarely it may occur without symptoms, perhaps in a family in which others have characteristic symptoms. When necrosis and suppuration are present in or around the teeth there may be extensive blackish discolouration, not only in the gums, but also in the mucous membrane of the inner surface of the lips.

In 1904[9] in a paper dealing chiefly with the ætiology, an account of several fresh cases of ocular neuritis is given. The great swelling of the ocular discs is emphasized, the presence of ocular paralyses affecting always the external rectus, with but one exception, and the mildness of the constitutional symptoms in many of the cases. Of four cases in which the urine was analysed for lead, one gave 0.4 milligramme per litre, one 0.32 milligramme; in the other two no lead was detected.

In 1905[9] two hundred cases of plumbism in all had been recorded at the Brisbane Hospital for Sick Children, including fifty-four cases of ocular neuritis. In only two or three of these cases did paralysis of limb muscles occur at the same time as ocular symptoms, though in several others both groups of symptoms occurred in succession in the same patients. The resemblance of the ocular symptoms to those of cerebral tumour is pointed out and also to those of basal meningitis; but, unlike these conditions, ocular plumbism does not cause death.

The process used in the Government Laboratory for testing urine for lead is given. The possibility of traces of lead existing in the reagents used had been eliminated by careful controls.

In 1908[10] and 1909[11] the whole subject was again reviewed. Two hundred and sixty-two cases had occurred in the Brisbane Children's Hospital and some twenty children were being admitted every year, while every medical man practising among children in that town should see several cases annually. The symptomatology is given in detail, but little of it is new.

In 1911[12] the importance of lumbar puncture as a remedy in plumbic ocular neuritis was demonstrated. Increased tension of cerebro-spinal fluid is always present. Out of nine children so treated, all recovered good sight except one, who had seen badly two weeks before treatment was commenced. The condition most liable to be confused with plumbic ocular neuritis is gummatous meningitis.

In 1914[13] the Report of the Institute of Tropical Medicine gives an account of lead poisoning in children in Townsville. At first the cases were regarded with some scepticism, but this was completely removed by chemical analysis of the excreta.

Basophilic granulations in a small number of erythrocytes were found in the majority of cases, sometimes only after a prolonged search. A blue line was observed in a small percentage of the cases. The symptoms observed were malaise, vomiting, pains in the calf muscles, foot- and wrist-drop, paralysis and wasting of the small muscles of the hand, mental irritability and in one case dimness of vision. Severe convulsions occurred in some. Anæmia was only marked in cases of long standing. Both fæces and urine were examined for lead and a tabular statement of the quantities obtained in repeated examinations in sixteen cases is given. An attempt to find lead in the urine of healthy children living under the same conditions completely failed. It may be stated that for several years the basophilic reaction of the red cells has been used as a routine test of plumbism in the Brisbane Children's Hospital and has been found to be a valuable aid in diagnosis.

In 1917[14] the diagnosis of the ocular neuritis due to plumbism was again discussed, its difficulty, especially to one unaccustomed to these cases, being fully recognized and the importance of immediate treatment, especially by lumbar puncture, clearly set forth.

In 1919[15] the deionizing treatment recommended by Oliver was instituted at the Children's Hospital, Brisbane. In one case, in which active symptoms had disappeared, after deionization, the Government Analyst found just under 0.6 milligramme of lead on the negative electrode. Since then the same official has found lead under similar circumstances in another case. (Owing to the absence of controls, these analyses are not absolutely diagnostic as are the urine analyses.)

There is a strong impression among some Brisbane practitioners that the prevalence of chronic nephritis in young adults in this city is partly a late result of the absorption of lead during childhood. To prove this would be very difficult and in the absence of proof it can only be regarded as a probable opinion.

At our request we have received a report from the Brisbane Children's Hospital, showing that there are at the present time twelve children in the wards suffering from lead poisoning. One of these has ocular plumbism, the other eleven paralyses of limb muscles; of the latter, one has had papilloedema recently. Colic is noted in three children; leg pains in two. Ten children have foot-drop; three have wrist-drop. Ten of the children are said to be nail-biters; two are not. A blue line on the gums is present in four; in one it is doubtfully present. All have basophilia of the red cells. In four the red cell count is below 3,000,000. Urinary casts have been found in seven, albuminuria in two.

So far nothing has been said as to the source of the poison. We are anxious to establish the fact of lead poisoning among Queensland children, apart from any theory which may be held as to its causation. Certainly when considering any single case the fact, if known, that the child has been exposed to the ingestion of poison, may be a factor of some weight in determining our diagnosis and treatment. But even here in a well-marked case showing the symptoms and stigmata of plumbism, such knowledge is unnecessary for diagnosis, though of the utmost importance for prevention and cure. Still more strongly does this hold of a large number of cases occurring consistently without interruption in one locality over a period of thirty years. These cases show symptoms which are recognized all over the world as characteristic of plumbism. Indeed few, if any, of such symptoms have been absent. And if we have enlarged the known list of symptoms somewhat, this is not surprising, as no other locality has furnished such a number of cases in childhood spread over an equal period of years. The characteristic stigma on the gums has been present in a large number of these cases. Repeatedly the urine has been tested by our Government Analyst and by other skilled chemists and has been found to contain minute but indubitable traces of the metal time after time. We maintain that these facts should bring conviction to every competent and unprejudiced mind. Unanimity in our profession is rare and we cannot say whether it exists on this subject in Brisbane; but the diagnosis of plumbism, at first confined to a few, has spread until it has become the settled conviction of all medical men connected with the Children's Hospital, of all specially interested in children's diseases, of many excellent general practitioners and of at least the great majority of members of this Branch.

Needless to say, the source of the poison has exercised the minds of some of the profession here for the last thirty years. What we believe to be the true solution was slow in discovery; it was first suggested by one of our members, lately alas deceased, in course of a discussion in 1899,[7] but attracted little attention, as suspicion was then directed in another direction and, being unsupported by detailed evidence, it was forgotten until revived in 1904.[8] Received then with mingled acceptance and doubt it has stood the test of eighteen years' repeated investigation and use for prophylaxis and cure, for the cure of plumbism consists primarily in the prevention of the further ingestion of lead.

Any hypothesis as to the source of the poison had to explain (*i.*) why cases arose every year among children, while no case (apart from those traceable to industrial occupation) was ever discovered in an adult, (*ii.*) why it occurred in Brisbane and other towns in Queensland from the extreme north to the extreme south, but not in rural areas, (*iii.*) why it should be comparatively common in Queensland, but rare or unknown as a malady of childhood in the southern States, (*iv.*) why, though often affecting several in a family, it often also picked out one child, the others remaining immune. It had also to be a *vera causa*, that is to say, the ingestion of lead in sufficient quantity over a sufficient period had to be proved. We claim that all these conditions have been fulfilled.

At the outset in 1892[11] suspicion was directed to the metal wrappings of sweetmeats and cigarettes, some of which were found on analysis to contain lead in large proportion. This hypothesis was found

to be inapplicable to so many cases that it was soon abandoned.

Of all sources of plumbism, apart from the industrial forms, perhaps the most important has been the contamination of drinking water. Now in Queensland towns much of the drinking water is collected from galvanized iron roofs which are fastened together by lead-capped nails. This water is stored in tanks, usually made of galvanized iron, the iron of some of the cheaper kinds being coated with an alloy containing lead instead of with pure zinc.[3] Tanks were sometimes repaired by solder containing lead and the connexions were sometimes made with lead piping. Suspicion of tank water was natural and was supported by the Government Analyst's report of the presence of lead in three samples of tank water. The hypothesis admirably explained the occurrence of plumbism in Queensland towns, but failed to explain why it did not affect adults. We were compelled to assume a greater susceptibility in childhood, but as year after year failed to bring forth a single adult case, this assumption became overstrained. It was also weakened by the failure of the Government Analyst to find lead in repeated samples of tank water submitted to him after the three positive analyses. Finally the hypothesis was destroyed by the Analyst informing us that water stored in galvanized iron tanks always contained zinc, that in the presence of dissolved zinc salts lead in solution is an impossibility, and that his previous positive results may have been due to impurity in the reagents used. We were grateful to him for his candour, our faith in the accuracy of his analyses (which are universally acknowledged) was, if anything, increased and we were left in doubt as to the ætiology of our cases.

In 1904[4] evidence was brought forward that the plumbism of children was caused by lead paint. It was pointed out that lead paint was dangerous (i.) when it was fresh and sticky, (ii.) when by exposure to sun and air the paint was loose and powdery and easily came off on the hands and was present in the form of dust. The first condition, stickiness, was of short duration and likely to be guarded against. The second, powderiness, was persistent until the surfaces were repainted and was especially common on verandah railings, fences and other outside surfaces exposed to sun and air. Two pieces of calico each a foot square were rubbed on some verandah railings. On analysis the first gave 0.3 gramme of lead, equivalent to 0.375 gramme of "white lead." Eighteen square inches from the centre of the other cloth were soaked in distilled water for twenty-four hours and practically the whole of the lead went into solution. No attempt was made to keep carbonic acid out of the distilled water, as it is present in all natural waters. In the case of one child suffering from ocular plumbism and passing 0.4 milligramme of lead per litre of urine, the child's brother showed a blue line on the gums. The parents had moved into a freshly painted house on December 5 and the child was brought to the Children's Hospital on January 7. The paint was still sticky. The mother said: "Those are the only two of my children who bite their nails." The evidence is not conclusive that the poison was derived from this house and not from powdery paint at their previous residence.

A visit was made to the house of a child suffering from ocular plumbism and passing 0.32 milligramme of lead per litre of urine. The child was two years of age and must have acquired the poison at home. The paint inside the house was in good condition, polished and not detachable on light friction. The verandah floors, as well as railings, were painted. Here the paint was dried and powdery. Pieces of calico cloth, 22.5 cm. × 10 cm., were used for rubbing the ceilings, walls of the living room, painted floor margin and verandah floors. The house had been cleaned and vacated and there was not much dust in the room. Samples of dust were carefully brushed off ledges into watch-glasses with a camel's hair brush to avoid dislodging the paint. From the cloth rubbed on the ceiling, where the paint was glossy and showed no signs of wear, no lead was obtained. From the cloths rubbed on the walls where the painted surface showed signs of wear 0.2 milligramme of lead carbonate was obtained. A similar quantity of lead carbonate was obtained from the cloth rubbed on the painted floor margin. From the cloth rubbed on the verandah floor twelve milligrammes of lead were obtained.

Dust.	Total Weight. Grammes.	Lead Carbonate Found. Milligrammes.
From top of moulding above door	0.2	1.5
Top of fanlight facing side verandah	0.25	Nil
Top of floor facing front verandah	0.25	0.2
Top piece of verandah blind	0.32	Nil
Top of skirting near door and side verandah	0.2	Nil

It was a hot day and it was found that the paint came off more noticeably on hands moist with sweat than on calico cloths. This child spent much of the day on the verandah floor and was fond of grasping the verandah rails.

Another house visited had white painted railings and a white painted gate from which the paint was readily detachable as powder. The paint had been rubbed off railings just at the height the children's hands would come in contact with it. One of the affected children in this household sucked her thumb. She had had repeated attacks of lead poisoning, occurring always in the summer months.

In summer children spend much time on the verandah and have hands moist and sweaty, so that the powdery paint adheres easily to them. On inquiry it was found that of eighty-five cases of lead poisoning in the Children's Hospital, forty-two had been admitted during the three hottest months, December to February, twenty-eight during the four months October, November, March, April and only sixteen during the five cooler months.

It was pointed out that powdery paint might be inhaled as dust and absorbed through the lungs, that possibly it might be absorbed through the skin, but it was thought that biting nails and sucking fingers was the chief method of absorption. The parents of ten children who suffered from lead poisoning, in answer to a question, replied in eight cases that these children bit their nails or sucked their fingers. Of five other children then under direct

observation in hospital, four bit their nails and one, aged two years, sucked his fingers.

In 1905[9] these facts were brought before the Intercolonial Medical Congress in Adelaide. The following motion was passed by the Section of Ophthalmology and reported to full Congress:

> That permanent blindness from lead poisoning among young children in Queensland has been recognized; that it is highly probable that paint used on verandah railings, etc., especially when exposed to weather influences, is the source of the poison and that this matter should be brought under the notice of the proper authorities.

Subsequently a confirmatory communication was received from the Queensland Commissioner of Health and published in the *Transactions*.

In 1908[11] special stress was laid on the danger of powdery paint on verandah railings and it was shown how the domestic architecture of Queensland towns favoured poisoning. The houses are almost always built of wood, raised on wooden piles and the verandahs railed in with painted railings. Young children pass their playing hours on these verandahs and clasp the railings with their moist, sweaty hands. In country districts, where the verandahs are low and unrailed or the railings unpainted, these cases of poisoning do not occur.

In 1917[14] experience had shown that recurrent attacks of lead poisoning could be prevented by warning the parents of the source of the poison and getting them to replace lead paint by zinc paint on verandah railings and outside surfaces within the reach of children. Among those who lived in rented houses and especially in the case of hospital patients, we could seldom get this done and patients cured in hospital frequently returned within a few months, often with a more severe attack. Here cure without prophylaxis was of no permanent value.

We had come to the conclusion that to prevent lead poisoning among Queensland children, legislation was necessary. Our effort in this direction culminated in a resolution familiar to readers, adopted at the Australasian Medical Congress held in Brisbane in 1920. A deputation from the Branch waited upon the Queensland Home Secretary in 1921 and from him obtained a promise that the forthcoming *Health Act* will contain a clause prohibiting the use of lead paint on verandah railings and outside surfaces within reach of children's fingers. That this proposed legislation should meet with determined opposition from powerful moneyed interests we fully expected. That that opposition should receive medical support, we certainly did not expect. In 1921 the subject of lead poisoning was introduced at an inquiry held by the New South Wales Board of Trade on the use of white lead in the painting industry. Evidence was given by Dr. S. A. Smith subversive of our conclusions. We propose to criticize Dr. Smith's evidence in a subsequent communication.

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Reports of Cases.

INTERMEDIATE PARALYSIS OF RESPIRATORY CENTRE.

BY F. J. T. SAWKINS, M.B., CH.M.,
Medical Officer, Kenmore, New South Wales.

SOME twenty-eight years ago I saw several hospital patients with intermediate central respiratory paralysis in whom the outstanding clinical features were sudden failure of respiration, while the heart continued to beat regularly for considerable periods, if artificial respiration were maintained. One patient recovered sufficiently to be discharged from hospital and was lost sight of. One other recovered temporarily, but—I write from memory—died in a subsequent attack. The condition in both these patients was, I believe, discovered by Dr. W. F. Litchfield; certainly the first one was.

Another patient was the subject of an historic case of brain hydatid recorded by the late Sir James Graham. This man had been the first or one of the first to recover after operation therefor. He had been re-admitted some years after the operation for a recurrence of cerebral symptoms and died of intermediate central respiratory paralysis. The *post mortem* examination disclosed several hydatid cysts in the region of the original cyst; one of these had burst into the lateral ventricle. These cases and some others were afterwards published by Dr. A. S. Vallack in *The Lancet*.

Considering the extraordinary results of surgical interference in conditions of internal hydrocephalus recently reported from America, I venture to record another case of