

that it is impossible now to say cardiac mechanism to normal

of nodal extrasystoles added to very short *P-R* intervals (0.10 sec. of the two necessary requirements arising in Tawara's node,³ ventricle should contract simultaneously should be distinctly shortened (0.17 second). The other requirement, should be of altered in the second beat of each pair nervous condition of the patient me to obtain records free from literature a rhythm made up of one of each of these records has spite of the fact that Lewis and arising at an appreciable distance ity or inversion of the *P* wave. A esbyterian Hospital of New York⁴ cases of apparent nodal rhythm to a prolonged *P-R* interval equal diac cycle, so that *P* of one cycle

Whether true nodal rhythm can the form of *P* is a point that is well

he gradual lengthening of the *P-R* interval without change in the form of the *P* wave, short *P-R* interval of both cycles arising in Tawara's node, but to extrasystoles arising in the sinus node and ectopically. As the chief factor in the delay caused by the passage of the impulse through the bundle,⁵ one must assume that the delay was due to positive dromotropic influences that these influences disappeared later, as did the bathmotropic influences causing extrasystoles. About the possible conductivity, however, there is practically no clinical or experimental.

Arrhythmia is described in which a delay in different parts of Tawara's node or in the conduction time (*P-R* interval) with a prolonged *P-R* interval (pulsus bigeminus).

Electrocardiography, p. 50.

from Dr. Strong.

Ann. Phys., 1909-1910, cxxxi, 572.

BLACKFAN: LEAD POISONING IN CHILDREN

Both of these disorders of the cardiac mechanism were relieved by a pelvic operation (anterior and posterior colporrhaphy), at least to the extent that they disappeared during the period of observation.

My thanks are due to Dr. C. C. Norris for permission to study this case.

LEAD POISONING IN CHILDREN WITH ESPECIAL REFERENCE TO LEAD AS A CAUSE OF CONVULSIONS.¹

By KENNETH D. BLACKFAN, M.D.,

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We are indebted to the Australian writers Gibson, Love, Turner, Breinl and Young and others for much of the recent literature regarding lead poisoning in children. They have had an unusually good opportunity to study this condition. Thus, in the Brisbane Children's Hospital, 76 cases were seen in five years and Gibson himself observed 24 cases in six years.

I shall not attempt to give a complete review of the literature on the subject, as the chief object of this communication is to draw attention to lead as a cause of convulsions in children and to report 4 cases seen in Dr. Howland's clinic at the Harriet Lane Home, the Johns Hopkins Hospital. For the more important articles, the reader is referred to those from which I have freely quoted.²

The symptoms of lead poisoning vary according to the susceptibility of the child and to the duration of the infection. The degree of susceptibility is variable. Breinl and Young refer to many instances in which only one child in a family was affected, although the other children were exposed to the same influences. In other instances, one child reacted with pronounced symptoms while the only evidence of infection in the others was a blue line on the gums.

Gibson³ considers the ingestion of lead as the most likely source of infection, although he appreciates that it may occur from the inhalation of dust containing lead. He points out that children

¹ From the Harriet Lane Home, Johns Hopkins Hospital, and the Department of Pediatrics, Johns Hopkins University.

² Gibson, Love, Hardie, P. Bancroft, Jeffris Turner: Notes on Lead Poisoning as Observed in Children in Brisbane, Intercolonial Medical Congress of Australasia, Sydney, 1892, p. 76. Gibson, Lockhart: Ocular Neuritis Simulating Basal Meningitis Plumbism, Australasian Med. Gaz., October 20, 1897, p. 479. A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Among Queensland Children, Australasian Med. Gaz., 1904, xxiii, 149. The Importance of Lumbar Puncture in the Plumbic Ocular Neuritis of Children, Tr. Australasian Med. Congress, 1911, ii, 750. Breinl and Young: The Occurrence of Lead Poisoning Among North Queensland Children, Ann. Trop. Med. and Parasitol., 1914-15, viii, 475. Turner, Jeffris: Lead Poisoning Among Queensland Children, Australasian Med. Gaz., 1897, October 20, p. 475.

³ A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Among Queensland Children, Australasian Med. Gaz., 1904, xxiii, 149.

who bite their fingernails and suck their fingers are much more frequently affected, and believes that their hands became contaminated with dried paint from porch railings and houses. That poisoning may occur through inhalation has been shown by the experiments of Goadby and Goodbody⁴ and by Legge and Goadby.⁵ They conclude that the danger from inhalation is far greater than from the ingestion of lead. While this, no doubt, is true among workers in lead, the swallowing of lead in some form is probably the more common source of the poisoning in children.

In the early cases a change in disposition is often the first symptom which is noticed. The child becomes fretful, peevish, and often very restless at night. The appetite becomes poor, the breath foul, and frequently hemorrhages occur from the gums. The child may complain of pain in the epigastrium and legs.

In the cases of longer duration the pains in the abdomen become continuous and more severe. Constipation is present, as a rule. Vomiting rarely occurs. The muscles are often so painful as not to permit of the weight of the bed-clothing. The gait of the patients is described as being characteristic. It is a waddling gait; they walk on the outside of the feet, the toes are dragged, and with each step the legs are swung sideways before the feet are put to the ground. Occasionally the parents' attention may be attracted by one or more of the many indefinite signs enumerated above, but not infrequently nothing abnormal is noticed until the development of cerebral disturbances. The cerebral manifestations will be referred to later.

Very few of the cases reported by Breinl and Young showed the characteristic wrist-drop so common in adults. This has been the experience of others, and generally the paralysis first affects the legs in children. A paralysis much like that of the Aran-Duchenne type occurring in muscular dystrophy has been reported in 2 cases. Paralysis of the cranial nerves is very common; the facial and the motor oculi nerves, either entire or in part, are those most frequently involved.

Gibson⁶ has reported a most interesting group of eye symptoms which he refers to as a "plumbic ocular neuritis." The neuritis usually occurs in children below the age of eight years. There may be an optic neuritis, with retinal hemorrhages, or an optic atrophy. There is an accompanying weakness of one or both external recti muscles. He has seen as many as 9 such cases in a year, but in the 22 cases reported by Breinl and Young only 1 patient was so affected.

⁴ Seventh International Congress of Applied Chemistry, 1909, Section VIIIa, 306.

⁵ Poisoning and Lead Absorption, London, 1912, p. 122.

⁶ Ocular Neuritis Simulating Basal Meningitis-Plumbism, Austral. Med. Gaz., October 20, 1897, p. 479. The Importance of Lumbar Puncture in the Plumbic Ocular Neuritis of Children, Tr. Austral. Med. Congress, 1911, ii, 750.

The temperature is, as a rule, elevated. In the fatal cases, disturbances, it may be high, 1

Very often the clinical symptoms of lead poisoning. diagnosis of lead poisoning. cases. However, when there evidences of the condition are usually the blue line on the gums and granulations are present in a small in the majority of the cases. characteristic, although there apparently is determined by the

Direct evidence of lead poisoning of lead in the urine or feces. Irritations of Breinl and Young are eight children with early signs of epigastrium and legs, but without lead line, and found appreciable. They found there was always a blue line in the urine, and that it was often not be demonstrated in the urine quantities of lead present in the urine have made this observation, and in the urine is not so common.

From the foregoing summary of cases seen in lead poisoning in children classification of the cases into different groups has to be based on the severity of the disease. For instance, one patient may present only irritability of the abdomen, whereas another may be convulsed while in apparent health attendant upon establishing a diagnosis. Jeffris Turner⁷ speaks of four main

1. Paralytic cases showing symptoms of spasm of the calf muscles, and at talipes equinus; (2) cases characterized by paralysis of the limbs, concomitant with habitual blue line on the gums, the child is irritable and neurotic; (3) children with a neuritis involving the optic, and motor nerves; (4) eclamptic cases with persistent convulsions which often end in death.

In the cases reported from Australia

⁷ Forensic Medicine and Toxicology, London, 1897, p. 475.

⁸ Lead Poisoning Among Queensland Children, 1897, p. 475.

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The temperature is, as a rule, normal, though it may be irregularly elevated. In the fatal cases, and especially those with cerebral disturbances, it may be high, reaching 105° or 106° before death.

Very often the clinical symptoms are not sufficient to warrant a diagnosis of lead poisoning. This is especially true in the early cases. However, when there are suggestive symptoms other evidences of the condition are usually to be found. Among these are the blue line on the gums and stippling of the red cells. Basophilic granulations are present in a small number of red blood corpuscles in the majority of the cases. The blood otherwise shows nothing characteristic, although there may be a secondary anemia. This apparently is determined by the duration of the poisoning.

Direct evidence of lead poisoning is obtained by the demonstration of lead in the urine or feces. In this connection the recent observations of Breinl and Young are most interesting. They examined eight children with early signs of the disease, *i. e.*, slight pains in the epigastrium and legs, but without basophilic granulations and the lead line, and found appreciable quantities of lead in the excreta. They found there was always a larger amount in the feces than in the urine, and that it was often present in the feces when it could not be demonstrated in the urine. Dixon Mann⁷ found larger quantities of lead present in the feces than in the urine. Others have made this observation, and Legge and Goadby say that lead in the urine is not so common nor definite a symptom as supposed.

From the foregoing summary of the more common clinical signs seen in lead poisoning in children it is evident that a definite classification of the cases into different groups is difficult, and when made has to be based on the severity of the predominating symptom. For instance, one patient may present the characteristic picture of the disease, one may show only irritability and indefinite pains in the abdomen, whereas another may become paralyzed or may be seized with convulsions while in apparent health. In spite of the difficulties attendant upon establishing a classification for lead poisoning, Jeffris Turner⁸ speaks of four main groups as follows:

1. Paralytic cases showing symmetrical wrist-drop and foot-drop; spasm of the calf muscles, and as a secondary lesion, a persistent talipes equinus; (2) cases characterized 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) children suffering from ocular neuritis, a neuritis involving the optic, and at the same time the oculo-motor nerves; (4) eclamptic cases suffering from severe and persistent convulsions which often end fatally.

In the cases reported from Australia, convulsions were observed

⁷ Forensic Medicine and Toxicology, London, 1908, p. 457 (C. Giffin & Co.).

⁸ Lead Poisoning Among Queensland Children, Australasian Med. Gaz., October 20, 1897, p. 475.

very infrequently, though mild cerebral disturbances were frequently present. That convulsions may occur in the course of lead poisoning, has been recognized since the observations of Stockhausen⁹ in the seventeenth century. The first careful investigation of the cerebral disorders due to lead was made by Tanquerel.¹⁰ He grouped them under the term *Encephalopathia Saturnina*. Though his researches were published in 1882, but little attention has been paid to the lead encephalopathies except as they are found in adults and in particular among workers in lead. Lead as a cause of convulsions in children has been ignored almost entirely, and especially among observers in this country.

An interesting and extensive series of cases with convulsions has been reported by Stewart.¹¹ They occurred among 64 cases of lead poisoning which he investigated in Philadelphia, due to eating buns colored with chrome yellow. Seven of the 16 cases in which convulsions occurred were in children between one and a half years and twelve years of age. Stewart believed that of the various forms of cerebral disorders produced by lead, the convulsive was by far the most common—that it formed two-thirds, if not more, of all the cerebral manifestations, and that it had the highest mortality. He (Stewart) thought it impossible to make any clinically exact systematic grouping of the different forms of the lead encephalopathies as Tanquerel¹² did, as one form merges into the other.

Stewart cites 2 cases of so-called idiopathic epilepsy which were secondary to convulsions, symptomatic of lead poisoning, and suggested that epilepsy of lead origin may be of much more frequent occurrence than is generally supposed.

The macroscopic changes most frequently found in the brain are an edema and anemia, with flattening of the convolutions. The brain may, however, be markedly congested. There is often a thickening of the pia—it may be adherent to the cortex. There may be hemorrhages into the pia and between the pia and the cortex. There is also a round-cell infiltration within the pia, and the neuroglia cells of the cortex are found to be increased. Spiller¹³ observed a proliferation of the endothelial cells upon the surface of the cerebral pia. Such pathological findings may occur in other conditions, and similar lesions may be found in patients with lead poisoning who have not manifested cerebral symptoms.

There is no way by which the convulsive seizures *per se* in lead poisoning can be distinguished from those due to other causes. All types of convulsions are seen. They may be local or general.

⁹ Quoted by Stewart (see note 11).

¹⁰ *Lead Diseases* (translation by Dana, 1848), p. 263.

¹¹ *Lead Convulsions*, *AM. JOUR. MED. SC.*, 1895, cix, 238.

¹² Tanquerel established the following divisions: (1) delirious forms; (2) comatose form; (3) convulsive forms; (4) above forms combined.

¹³ *Jour. Med. Research*, 1903, x, No. 1.

They may be mild or severe. They do, however, have certain peculiar characteristics of the disease. They have a tendency to recur, and they are

The persistence of the convulsions. Enormous doses of bromides, continued to each patient without effect. It was only by the continued use of chloroform, controlled, and when this was controlled anew. In one patient (Case I) there was a period of seven and a half months after a period of eighteen days in which each recurrent attack they were treated with chloroform. One patient (Case II) had an attack. The convulsions in one patient lasted only one day. He was delirious after which he was comatose for several days. This patient serves to support Stewart's classification to define sharply the encephalopathies.

The high mortality of convulsions in Stewart's cases. The termination of the disease. A series of most remarkable cases in which the fatality of the condition equaled that of the disease. In one case who lived day and night in an attempt to breathe the fumes, the father suffered from the same encephalopathy. Seven children died of frequent cerebral disorders and convulsions. Several of the grandchildren died of the same disease. In 3 of the 4 patients died. There were shown any evidences of a recurrence of convulsions during the third attack, and the other (Case II) died of a second attack, and the other (Case III) died of a third attack.

The temperature, which was normal in (Case II and Case III), preceding the convulsions respectively. Such a hyperpyrexia preceding death from lead convulsions in man is not infrequently present.

Evidences of cerebral involvement are present. In one patient (Case I) there was a positive Kernig sign. He had a paralysis of the rectus muscle of the eye. Another patient had a paralysis of the neck and a temporary paralysis of the arms.

Mosny and Malloizel¹⁴ and Plat¹⁵ have shown that there is very generally evidence of cerebral involvement in lead poisoning.

¹⁴ *Berl. klin. Wehnschr.*, 1874.

¹⁵ *La meningite saturnine*, 1907, xxvii, 50.

¹⁶ *Ueber einen Fall von Meningitis saturnina*, 1874, lx, 234-3.

bral disturbances were frequently occur in the course of lead poisoning, observations of Stockhausen⁹ in the careful investigation of the cerebral by Tanquerel.¹⁰ He grouped them as saturnina. Though his researches the attention has been paid to the they are found in adults and in

Lead as a cause of convulsions most entirely, and especially among

series of cases with convulsions has occurred among 64 cases of lead in Philadelphia, due to eating buns even of the 16 cases in which convulsions between one and a half years and believed that of the various forms of lead, the convulsive was by far the most frequent, if not more, of all the it had the highest mortality. He tried to make any clinically exact system of forms of the lead encephalopathies merges into the other.

Lead idiopathic epilepsy which were idiopathic of lead poisoning, and suggest that it may be of much more frequent occurrence.

It is frequently found in the brain are the thickening of the convolutions. The vessels are edly congested. There is often a thickening adherent to the cortex. There may be a separation between the pia and the cortex. The meninges within the pia, and the neuroglia may be increased. Spiller¹³ observed a thickening upon the surface of the cerebral vessels may occur in other conditions, and in patients with lead poisoning who have no symptoms.

The convulsive seizures *per se* in lead poisoning are from those due to other causes. All

They may be local or general.

They may be mild or severe. The convulsions in lead poisoning do, however, have certain peculiarities which appear to be characteristic of the disease. They are very persistent, they show a great tendency to recur, and they are attended by a high mortality.

The persistence of the convulsions is shown in Cases II and III. Enormous doses of bromides, chloral, and morphin were administered to each patient without causing any effect on the convulsions. It was only by the continued use of chloroform that they could be controlled, and when this was discontinued the convulsions began anew. In one patient (Case I) there were two recurrent attacks in a period of seven and a half months. The convulsions recurred after a period of eighteen days in another patient (Case II). With each recurrent attack they were severe, and were controlled only with chloroform. One patient (Case III) died in the first convulsive attack. The convulsions in one patient (Case IV) were mild, lasting only one day. He was delirious at first, then had convulsions, after which he was comatose for several days. The behavior of this patient serves to support Stewart's contention that it is not possible to define sharply the encephalopathies into definite groups.

The high mortality of convulsions due to lead is shown by Stewart's cases. The termination was fatal in 6 of the 7 children. A series of most remarkable cases reported by Berger¹⁴ emphasizes the fatality of the condition equally well. In a pot-glazier's family, who lived day and night in an atmosphere saturated with lead fumes, the father suffered from the comatose variety of lead encephalopathy. Seven children died in convulsions, four others had frequent cerebral disorders and suffered from peripheral neuritis. Several of the grandchildren died in convulsions. In my series of cases 3 of the 4 patients died. The patient who recovered has not shown any evidences of a recurrence. One patient (Case I) died in convulsions during the third attack, another (Case II) during the second attack, and the other (Case III) during the first attack.

The temperature, which was normal on admission in two patients (Case II and Case III), preceding death rose to 107.5° and 105.5° respectively. Such a hyperpyrexia has been present preceding death from lead convulsions in most of the reported cases.

Evidences of cerebral involvement other than convulsions were present. In one patient (Case I) there was rigidity of the neck and a positive Kernig sign. He had also a paralysis of the left external rectus muscle of the eye. Another patient (Case IV) had rigidity of the neck and a temporary paralysis of the facial nerve.

Mosny and Malloizel¹⁵ and Plate¹⁶ and others have pointed out that there is very generally evidence of involvement of the meninges,

⁹ a, 1848), p. 263.

¹⁰ p. Sc., 1895, cix, 288.

¹³ divisions: (1) delirious forms; (2) comatose forms combined.

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¹⁴ Berl. klin. Wehnschr., 1874.

¹⁵ La meningite saturnine, 1907, xvii, 506.

¹⁶ Ueber einen Fall von Meningitis saturnina, München. med. Wehnschr., 1913, ix, 2343.

as shown by the fact that the cerebrospinal fluid contains an increased number of cellular elements. In chronic lead poisoning they rarely found any change in the cerebrospinal fluid. In this series the spinal fluid from all the patients was examined and changes were found in three of them. In these three patients the spinal fluid was clear, not under increased pressure, and a fibrin clot did not form on standing. In two patients (Case II and Case III) the cellular elements were not increased, but there was a marked globulin reaction (Ross-Jones). In one patient (Case I) there were thirty cells per cubic millimeter, mainly lymphocytes, and a marked globulin reaction. The spinal fluid from this patient was examined on several occasions over a period of eight months, and on each examination there was an increase in cells and a positive globulin. In the fourth patient (Case IV) the spinal fluid was not examined until four weeks after he had recovered, and at this time it showed no changes other than twelve cells per cubic millimeter. Such a slight increase in cells does not signify a pathological change.

Eye changes during the course of lead encephalopathies have been noted by a number of different observers. Wilbrand and Sanger,¹⁷ who have collected reports of most of the cases, believe that optic neuritis may occur primarily through the direct toxic action of lead on the nervous system or by the production of interstitial neuritis or through the alterations of the bloodvessels in the retina and optic nerves. It also may occur secondarily through intracranial changes or changes in the kidney. Gibson, as mentioned previously, has written extensively on the eye changes in children, and believes that they frequently occur without other symptoms of lead poisoning. In two of my patients (Case I and Case II) there were extensive hemorrhages into the retina and optic neuritis. In one (Case I) the condition gradually subsided and the eye grounds became normal after a month. At the time of his second admission there were retinal hemorrhages and a choked disk. The eye grounds were normal in the other two patients (Cases III and IV).

As a rule there are certain symptoms which precede the development of the convulsions. These may be the common symptoms of plumbism or they may be so slight as to escape notice. Colic has been more frequently observed than paralysis or arthralgia, but more common are irritability, restlessness, and a blue line on the gums. Vomiting and constipation are not infrequent, and many patients complain bitterly of pain in the head. In two patients (Case I and Case IV) of this series there was an interval of several days before the convulsions, when the children were irritable during the day and restless at night. One of them complained of headache, and they both had attacks of vomiting. In one instance (Case III) the indefinite symptoms covered a period of about three months. There

was evident impairment of general health, with vomiting and colic. At times the

The duration of the period between the development of cerebral symptoms and the exposure in the patients of with certainty. Three of them (Cases I, II, and III) were exposed for many months, as the source of eating paint. In one patient (Case IV) it was ascertained.

In addition to the symptoms of lead poisoning, the patients in this series showed other changes. The blood picture was that of a mild anemia, with in all a few red blood corpuscles. In the four patients there was a moderate increase in the polymorphonuclear cells. On the gums a moderate increase in the polymorphonuclear cells was present in three (Cases I, II, and IV). It should be remembered that the characteristic appearance of black dots in the margin of the teeth which are covered with tartar can be seen only with the aid of a hand lens or microscope.

Efforts made to determine lead in the blood (Cases I and II) were unsuccessful. The following are brief histories of the patients reported in this report:

CASE I.—The patient, W. M., 12 months old, complained first of pain in his head. The following day he vomited several times, and his neck was stiff. He appeared ill, and on the fourth day of his illness he was taken to the hospital in a comatose state. He had generalized convulsions, his neck was stiff, and there was internal strabismus of the left eye and a hemorrhagic retinitis on the left eye. The temperature was normal. The temperature was 97.600. The spinal fluid obtained by the first spinal puncture, but subsequent spinal punctures were clear and sterile. It was under slight pressure. There were from twenty to forty cells per cubic millimeter (mostly mononuclears). The Noguchi globulin test (blood) was negative. The Wassermann (blood) was negative. The first lumbar puncture, but he recovered in two days. He gradually improved and was well. The convulsions did not recur.

¹⁷ This case was reported by Thomas and I in *Die Neurologie des Auges*, 1913, vol. v, 377.

¹⁷ *Die Neurologie des Auges*, 1913, vol. v.

cerebrospinal fluid contains an s. In chronic lead poisoning : cerebrospinal fluid. In this ents was examined and changes these three patients the spinal pressure, and a fibrin clot did its (Case II and Case III) the ed, but there was a marked ne patient (Case I) there were ly lymphocytes, and a marked rom this patient was examined of eight months, and on each n cells and a positive globulin. spinal fluid was not examined red, and at this time it showed per cubic millimeter. Such a fy a pathological change. of lead encephalopathies have rent observers. Wilbrand and of most of the cases, believe that through the direct toxic action by the production of interstitial of the bloodvessels in the retina occur secondarily through intra- kidney. Gibson, as mentioned on the eye changes in children, occur without other symptoms : patients (Case I and Case II) into the retina and optic neuritis. ally subsided and the eye grounds t the time of his second admission d a choked disk. The eye grounds tients (Cases III and IV). ptoms which precede the develop- may be the common symptoms of ht as to escape notice. Colic has an paralysis of arthralgia, but more erness, and a blue line on the gums. ot infrequent, and many patients ead. In two patients (Case I and s an interval of several days before ren were irritable during the day hem complained of headache, and ng. In one instance (Case III) the eriod of about three months. There

des Auges, 1913, vol. v.

was evident impairment of general health with frequent attacks of vomiting and colic. At times the onset of convulsions is sudden.

The duration of the period between the first exposure to lead and the development of cerebral symptoms is variable. The exact time of the exposure in the patients of this series could not be determined with certainty. Three of them (Cases I, II and III) had been exposed for many months, as the source of the infection was found to be from eating paint. In one patient (Case IV) the source of lead was not ascertained.

In addition to the symptoms preceding the convulsions the patients in this series showed other evidences of lead poisoning. The blood picture was that of a moderate secondary anemia, and in all a few red blood corpuscles showed basophilic degeneration. In the four patients there was a leukocytosis of about 20,000, with a moderate increase in the polymorphonuclear cells. The blue line on the gums was present in three of the four patients (Cases I, III, IV). It should be remembered that a continuous lead line is seldom found. The characteristic appearance is the presence of minute black dots in the margin of the mucous membrane around the teeth which are covered with tartar. It is difficult to see and without the aid of a hand lens may readily be overlooked.

Efforts made to determine lead in the urine of two patients (Cases I and II) were unsuccessful. The feces were not examined.

The following are brief histories of the 4 cases which form the basis for this report:

CASE I.—The patient, W. M.,¹⁸ aged five and one-half years, complained first of pain in his head, and was very restless at night. The following day he vomited several times, and it was noticed that his neck was stiff. He appeared very ill, became rapidly worse, and on the fourth day of his illness severe generalized convulsions began. On the fifth day, August 22, 1913, he was brought to the hospital in a comatose state. He continued to have recurrent generalized convulsions, his neck was retracted, and there was an internal strabismus of the left eye. There was an optic neuritis and a hemorrhagic retinitis on the right. The left optic nerve was normal. The temperature was normal. The leukocytes were 27,600. The spinal fluid obtained on four occasions was clear and sterile. It was under slightly increased pressure at the first spinal puncture, but subsequently the pressure was normal. There were from twenty to forty cells per cubic centimeter (mostly mononuclears). The Noguchi globulin reaction was positive. The Wassermann (blood) was negative. The convulsions ceased after the first lumbar puncture, but he remained in coma for the next two days. He gradually improved and after a week seemed nearly well. The convulsions did not recur and he was discharged

¹⁸ This case was reported by Thomas and Blackfan: *Am. Jour. Dis. Child.*, 1914, viii, 377.

September 20, 1913. The abnormal changes in the right optic nerve gradually subsided, but the spinal fluid contained an increase in cells and a positive globulin reaction. With the exception of an occasional attack of vomiting and abdominal pain, he remained in excellent condition for five months.

On March 1, 1914, he complained of headache and he vomited. The convulsions recurred, and on March 3 he was again admitted to the hospital. His condition was almost identical with that at the time of the first admission. There were generalized and persistent convulsions, a definite rigidity of the neck, and an optic neuritis and hemorrhagic retinitis of the right eye. At this examination the characteristic lead line was seen on the gums. The red blood corpuscles were 4,480,000, the white blood corpuscles 23,000, and the hemoglobin 55 per cent. There was well-marked stippling of the red blood corpuscles (Grawitz granules). No lead could be demonstrated in the urine. The convulsions subsided after lumbar puncture. Two or three days after admission an internal strabismus of the right eye developed. The patient made an uneventful recovery and was discharged on May 1.

He remained in apparently good health for three weeks, when on May 25 he had a severe convulsion and died. No postmortem examination was obtained. The source of the lead was determined when his lips were found covered with white lead paint which he had nibbled from the railings of his crib. On investigation at the orphanage where he lived it was found that the white paint on his bedstead had been entirely gnawed off.

CASE II.—R. B. (7094). This patient was aged two and one-half years. On March 8, 1915, he had a convulsion which lasted about two hours. From then until March 12 he seemed well. On the morning of March 12 convulsions began about 7.30. He was brought to the hospital at noon the same day in convulsions, which continued for about twenty-four hours. These convulsions involved the eyes, the left side of the face, and the left arm. He had no fever. There was a leukocytosis of 19,000. The red blood cells were 4,000,000. Hemoglobin, 50 per cent. There was well-marked stippling of the red cells.

On examination the positive findings were a lead line about the upper and lower teeth and an enlarged spleen. He was entirely relaxed between the convulsions, the reflexes were not exaggerated, and Kernig's sign was absent. The eye grounds were normal. The spinal fluid was clear and not under pressure. There were seven cells per cubic millimeter and a positive globulin reaction. Examination of the urine for lead was negative.

The child had no convulsions in the hospital, and was discharged on March 25. He was brought back to the hospital on March 26, twelve hours after discharge, in a convulsion which involved the face and the left arm. He continued to have convulsions for twenty-four

hours in spite of energetic treatment with bromides. By the administration of bromides could be checked, but they recurred. The temperature rose to 107.5°.

Source of Lead. The father stated that he had read a newspaper article, and that he and his wife had set of parlor furniture by eating

CASE III.—(9182) H. B., age 18 months. Case No. II) died in the Johns Hopkins Hospital on March 27, 1915.

The patient was a normal child at sixteen months. Shortly after the age of sixteen months the abdomen and was operated on for a hernia (February, 1915). The appendix was removed. The months preceding the onset of his illness he often vomited and complained of abdominal pain. He vomited more than usual on October 1, 1914, and had a convulsion. The convulsions continued until the following day, October 2, when he was admitted to the hospital. The convulsions were general in character. The rigidity of the right than the left side was not rigid; there was no Kernig's sign. Examination of the mucus membranes failed to show a lead line.

Ophthalmoscopic Examination. The fundus was obscured. The vessels were filled with retinal hemorrhages. Left: The fundus was obscured by a large retinal hemorrhage, and the vessels were not visible throughout. The leukocytes were 3,600,000 and the hemoglobin 50 per cent. The stippled red cells were present. There was an increase in pressure. There was no reaction for globulin was present.

Course in Hospital. The child required enormous doses of chloral and morphine to control them by the use of chloral and morphine for temporary benefit, as the convulsions were discontinued. The patient died on October 1, 1914, temperature, which was 97.6° on October 1, just before death reached 105.5°.

Source of Lead. Same as in Case II.

CASE IV (8491).—J. N., aged three years. One of two children, was of normal development until the onset of pertussis at two years, had no history of convulsions. The patient died on July 30, 1915, with drowsiness. He

mal changes in the right optic spinal fluid contained an increase in protein. With the exception of an increase in abdominal pain, he remained in good health.

ed of headache and he vomited. March 3 he was again admitted as almost identical with that at March 1. There were generalized and periodicity of the neck, and an optic atrophy of the right eye. At this examination the fundi were as seen on the gums. The red blood cells were 23,000, hemoglobin 15 per cent. There was well-marked stippling (Pappenheimer granules). No lead could be detected in the urine. After the convulsions subsided after lumbar puncture and admission an internal strabismus was noted. The patient made an uneventful recovery.

He remained in good health for three weeks, when a convulsion occurred and he died. No postmortem examination was made. The source of the lead was determined to be the white lead paint which he had used on his crib. On investigation at the home it was found that the white paint on his crib had been removed.

The patient was aged two and one-half years. He had a convulsion which lasted about three minutes. Until March 12 he seemed well. Convulsions began about 7.30. He had on the same day in convulsions, lasting four hours. These convulsions involved the face, and the left arm. He had a temperature of 100.0. The red blood cells were 19,000. The hemoglobin was 15 per cent. There was well-marked

findings were a lead line about the mouth and enlarged spleen. He was entirely normal. The reflexes were not exaggerated, the eye grounds were normal. The cerebrospinal fluid pressure was normal. There were seven cells per cubic millimeter and a positive globulin reaction. Examination of the urine was negative.

He was admitted to the hospital, and was discharged on March 26, after a convulsion which involved the face and arms. He had no more convulsions for twenty-four

hours in spite of energetic treatment with morphin, chloral, and bromides. By the administration of chloroform the convulsions could be checked, but they recurred as soon as it was discontinued. The temperature rose to 107.5° just before death.

Source of Lead. The father stated that the child would gnaw any painted article, and that he and his brother had recently ruined a set of parlor furniture by eating the paint from it.

CASE III.—(9182) H. B., aged two years. A brother (R. B., Case No. II) died in the Johns Hopkins Hospital of lead poisoning March 27, 1915.

The patient was a normal child, and was well until he had varicella at sixteen months. Shortly after this he complained of pain in the abdomen and was operated on by an orthopedist for appendicitis (February, 1915). The appendix was normal. For two or three months preceding the onset of his acute illness he was not well. He often vomited and complained a great deal of abdominal pain. He vomited more than usual on October 15, 1915, and at 11 P.M. had a convulsion. The convulsions continued, and he was admitted to the hospital the following day, October 16. The patient was comatose on admission and was having repeated and severe convulsions. The convulsions were general in character, with possibly greater involvement of the right than the left side of the body. The neck was not rigid; there was no Kernig's sign. The knee-kicks were not obtained. Examination of the mucous membrane about the teeth failed to show a lead line.

Ophthalmoscopic Examination. Right: The margin of the disk was obscured. The vessels were full and tortuous; there were small retinal hemorrhages. Left: The margin of the disk was obscured by a large retinal hemorrhage, and there were other hemorrhagic areas throughout. The leukocytes were 20,000. The red blood cells were 3,600,000 and the hemoglobin was 55 per cent. Many stippled red cells were present. The spinal fluid was clear and not under increased pressure. There were nine cells per cubic millimeter and the reaction for globulin was positive.

Course in Hospital. The convulsions persisted in spite of enormous doses of chloral and morphin. It was only possible to control them by the use of chloroform. This, however, was of temporary benefit, as the convulsions commenced again when it was discontinued. The patient did not regain consciousness. The temperature, which was 97.6° on admission, gradually rose, and just before death reached 105.5° (fifteen hours after admission).

Source of Lead. Same as in Case II.

CASE IV (8491).—J. N., aged three years. The patient, the older of two children, was of normal development and, with the exception of pertussis at two years, had always been well. There was no history of convulsions. The present illness began acutely on July 30, 1915, with drowsiness. He slept the greater part of that

day, was very weak, refused his food, and vomited. At night he was delirious. He jumped out of bed, cried out several times, and threw his head back as though in pain. The next day, July 31, he appeared better, though still weak and drowsy. Again, at night, he became delirious and had several attacks of crying and throwing his head back. On August 1 he was comatose, and he had several convulsions. He did not improve, and was admitted to the hospital August 2.

The temperature on admission was 101°, pulse 124, respirations 20. He lay quietly in bed, with his eyes closed, and made no effort to speak. He was well-nourished and did not appear very ill. The neck muscles were slightly rigid and attempts to flex the neck caused pain. The reflexes were active but not increased. The examination of the heart and lungs was negative. The spleen was palpable. There was a well-marked lead line present in the mucous membrane about several of the upper teeth. It was especially well defined about the upper lateral incisors. The optic discs were normal.

The red blood corpuscles showed stippling in a few cells. The white blood corpuscles were 7500 per cu.mm. The differential count was:

	Per cent.
Polymorphonuclears	67
Small mononuclears	20
Large mononuclears	6
Transitionals	2
Polymorphonuclear eosinophiles	5

The Wassermann (blood) was negative.

Course in the Hospital. The child lay in a semistupor for most of the day after his admission. He was easily aroused, and at times appeared conscious but would not speak. The respirations were full and deep, varying between eighteen and twenty-four. They were irregular, and a few periods of apnea were observed. There was a well-marked *tâche cérébrale*, and Kernig's sign was suggestive. The neck was rigid. The condition remained the same for the next two or three days. There were no convulsions. On August 5 the patient seemed much better. He sat up in bed and could walk when placed on his feet. On this day an incomplete paralysis of the right facial nerve was observed. Improvement in his condition continued. The temperature gradually became normal, and six days after (August 8) admission he appeared quite well, except that he was very weak. The facial paralysis slowly became less marked, and it disappeared after about ten days. When he was discharged on August 12 (ten days after admission) he was apparently well.

A lumbar puncture was made several times during his stay in the hospital, but blood was obtained each time, and for this reason a cytological examination was not made. Four weeks after the onset

when the child was apparently under increased pressure, and millimeter. The globulin reaction source of lead was not determined.

Case	I.—W. M. 2 years.	II.— 2 1/2
Onset of convulsions	4th day	1st
Leukocytes	27,600	20
Spinal fluid:		
Cells	30	
Globulin	+	
Temperature, on admission	99.4° F.	99
Lead line	+	
Stippling red blood cells	+	
Eye	Hemorrhagic retinitis; choked disk	Same Case
Neurological	External rectus palsy; convulsions; coma; rigidity of neck	Convulsions; coma
Source of lead	Eating paint	Eating
Result	Died	Died

CONCLUSIONS. In conclusion, prophylactic measures be taken with painted articles in order to guard against poisoning. Since my attention has been called to this, I have found a number of children in enameled cribs.

In all patients with convulsions is not clear, lead should be suspected, as in the majority of instances of the condition, *e. g.*, the lead presence of lead in the feces.

The examination of the spinal fluid to the seriousness of the affection. In the four patients changes were present in the spinal fluid. In one patient eventually succumbed. It has recovered, and in whom the spinal fluid could not be examined. Four weeks later the spinal fluid globulin reaction was normal.

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when the child was apparently well, the spinal fluid was clear, not
under increased pressure, and contained twelve cells per cubic
millimeter. The globulin reaction at this time was positive. The
source of lead was not determined.

Case.	I.—W. M. 2 years.	II.—H. B. 2½ years.	III.—R. B. 2 years.	IV.—J. N. 3 years.
Onset of con- vulsions	4th day	1st day	2 to 3 mos.	3 days.
Leukocytes	27,600	20,000	19,000	19,500
Spinal fluid:				
Cells	30	9	7	12 ¹⁹
Globulin	+	+	+	0
Temperature, on admission	99.4° F.	99° F.	97.6° F.	101° F.
Lead line	+	0	+	+
Stippling red blood cells	+	+	+	+
Eye	Hemorrhagic re- tinitis; choked disk	Same as Case I	Negative	Negative.
Neurological	External rectus palsy; convul- sions; coma; rigidity of neck	Convulsions; coma	Convulsions; coma	Delirium; convul- sions; coma; rig- idity of neck; facial paralysis.
Source of lead	Eating paint	Eating paint	Eating paint	Not known.
Result	Died	Died	Died	Well at present time.

CONCLUSIONS. In conclusion, I would urge that energetic pro-
phylactic measures be taken with children who habitually eat
painted articles in order to guard against the development of lead
poisoning. Since my attention has been directed to lead poisoning
I have found a number of children who nibble the white paint from
enameled cribs.

In all patients with convulsions in which the etiological factor
is not clear, lead should be suspected. This can be readily deter-
mined, as in the majority of instances there are other evidences
of the condition, *e. g.*, the lead line, basophilic degeneration, and
the presence of lead in the feces.

The examination of the spinal fluid may prove to be an index as
to the seriousness of the affection and of prognostic aid. In three of
the four patients changes were found. In one patient (Case I)
changes were present in the spinal fluid for many months, and the
patient eventually succumbed. In another patient (Case IV) who
has recovered, and in whom the convulsions were not severe, the
spinal fluid could not be examined at the time of the convulsions.
Four weeks later the spinal fluid contained twelve cells and the
globulin reaction was normal.

¹⁹ Four weeks after the convulsion.