vertigo from a poisonous dose of quinine. It is, however, not necessary to give it in such large doses. Given a grain at a time, it seems to reduce the irritation of the vestibular nerve, probably by overcoming congestive changes. On the ground that vertigo is due to anaemia of the labyrinth, which, however, is probably not as a rule well founded, Lermoyez suggested the use of amyl nitrite. We are all aware, of course, that in conditions of severe anaemia vertigo is common, but whether this is an aural phenomenon or not, even when associated, as it often is, with tinnitus, is a doubtful point. At the same time it is quite possible that the actual cause of vertigo is not so much either congestion or anaemia as the alterations in the pressure of the endolymph which may be brought about by either of these states. The bromides and iodides are sometimes useful, and pilocarpine, especially in patients in whom the aural condition is dependent on congenital or tertiary acquired syphilis, has sometimes seemed of value. Repeated lumbar puncture has been used with some, though probably only with temporary, benefit. Patients should be advised to eschew alcohol, tea, coffee, and tobacco, to avoid as far as possible mental excitement and loud noises, and to keep the bowels acting well. During a severe attack the recumbent posture should be enjoined, with ice to the side of the head. When all other measures fail, and when life is made practically intolerable, the advisability of destroying the labyrinth—at any rate the static portion of it—should be entertained. I am referring now to cases of non-suppurative ear diseases. This has been carried out in this country by Milligan, Lake, and Yearsley; or, as Ballance suggests may yet be possible, the vestibular nerve, as distinct from the auditory nerve, may be divided before it enters the internal auditory meatus.

ON LEAD POISONING IN CHILDHOOD.*

By A. JEFFERIS TURNER, M.D.LOND., D.P.H.CAMB.,
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THOUGH lead-poisoning is practically unknown among adults in Queensland, except among those specially exposed to it by occupation, such as painters, yet lead-poisoning among children is not uncommon in every township in Queensland. By not uncommon I mean that every medical man practising among children should probably see several cases every year. The causation of the disease has been ascertained, and that similar cases must occur in all those parts of Australia in which similar conditions may happen to prevail.

The symptoms of lead poisoning in children differ from those found in adults in certain important particulars, which have not yet found their way into the textbooks, and as a consequence the real nature of the affection is very frequently overlooked or misunderstood.

ETIOLOGY.

My experience is confined mostly to Brisbane. I see cases rather commonly in private practice, more commonly since I have come to recognize the milder cases and earlier stages. A few of these cases have come from other towns. The records of the Children's Hospital show that 262 cases have been treated as in-patients since 1891, and at present some 20 cases are being admitted every year.

The cause of lead poisoning among Queensland children has been most convincingly demonstrated by Dr. Lockhart Gibson.¹ Nearly all the dwellings in Queensland are built of wood, and in the towns the wood is covered by paint consisting largely of white lead. Exposed to our hot summer sun this paint rapidly withers and becomes

reduced to a powdery condition. This is particularly noticeable on verandah railings. The verandahs are favourite playgrounds for young children, who clasp the railings with their moist hands, which become covered with poison. Thence it finds its way to the child's mouth, especially in children who suck their fingers or bite their nails. Dr. Gibson's investigations explain:

1. Why the disease is found in children from all towns in Queensland, but appears to be unknown in the country, where the houses are of wood but not painted.

2. Why it is most prevalent in the summer months.

3. Why it frequently picks out one child only in a family, attacking the one who bites his fingers or sucks his thumbs, or who plays habitually on the verandah, etc.

Similar cases must occur in any districts of the other States, if such exist, where the dwelling houses are similar; even though such cases may have never been recognized.

I do not mean to deny that lead poisoning may occasionally but rarely occur among Queensland children from other sources than that here given, but I have no doubt whatever that this is the cause of the endemic prevalence of this form of sickness.

SIGNS AND SYMPTOMS OF LEAD POISONING IN CHILDREN.

There are two signs of lead-poisoning in children that are not, properly speaking, morbid symptoms, but which are of great value as indicating the presence of lead in the tissues.

First, the so-called "blue line" on the gums. A continuous line is rather exceptional in children; what we usually see are small blackish dots at the extreme edge of the gum opposite some of the teeth, more particularly those that are not so clean as the rest. They are frequently minute, and need to be looked for in a good light, and sometimes with the aid of a lens. I repeat, they have to be looked for; on a casual inspection they will certainly be missed. Where there is actual pus exuding from the gum I have seen a dark black line on the edge of the sinus, and even discoloration of the cheek opposite to it, but this is rare. A discoloration of the base of the tooth itself is no sign of plumbism; and doubt as to the site of the blackness may be at once removed by inserting the edge of a piece of stiff notepaper between the gum and the tooth. These dots in the gum are pathognomonic. But they may occur in the absence of symptoms. When a child exhibits symptoms of plumbism an examination of the teeth of its brothers and sisters will sometimes reveal the presence of the dots in them, though they are in apparently good health. This *stigma* of plumbism may, as I have observed, be followed later by the usual symptoms. On the other hand, the complete absence of lead dots is of no importance in diagnosis. The lead dots are particularly likely to be absent if the child has a clean set of teeth.

The second *stigma* of plumbism is the presence of lead in the urine. The detection of traces of lead in this fluid should be the task of a trained analyst. Lead is by no means constantly present in the urine of these children, therefore little weight, if any, should be given to a single or even several negative findings. On the other hand, the presence of lead in the urine is positive proof that the metal has been absorbed

into the system. For clinical purposes an analysis of the urine is, I think, seldom necessary.

1. Gastro-intestinal Symptoms.

These correspond to the lead colic of adults, but are less defined. The child complains of abdominal pain, which is often plainly of a colicky nature. There is no abdominal tenderness, but pressure frequently gives relief. Vomiting, often persistent and lasting for several days, is a usual accompaniment. During this time all food is often rejected, with complete impartiality. The bowels are

* Presidential Address delivered at the Section of Diseases of Children of the Australasian Medical Congress held in Melbourne, October, 1908.

usually constipated, but this is not invariable; sometimes there are rather frequent small stools containing much mucus.

If these symptoms are accompanied by dots on the gums the diagnosis is easy. If the latter are absent it may be difficult, but may often be made with a high degree of probability.

One feature of these attacks is that they are independent of the nature of the food the child has taken, and another is their periodicity. If a child is brought with a history of attacks at intervals of a few weeks or months, during which it is continually vomiting and complaining of abdominal pain, while in the intervals it eats everything without suffering, plumbism should be strongly suspected. If pain in the legs accompanies that in the abdomen, plumbism may be safely diagnosed; and when the child—perhaps after several attacks—begins to drop its toes when walking, there is no longer any room for doubt.

In doubtful cases the fingers should be inspected to see if the nails are bitten, and if the house is visited the condition of the paint on the verandah railings and other parts, together with the habits of the child as to play, etc., should be inquired into.

2. Paralysis of the Muscles of the Limbs.

Lead poisoning and wrist-drop are so closely associated in the mind of the profession that it is necessary to say at once that wrist-drop is *not* the most characteristic paralysis in childhood. It is frequently absent in children under 12 years of age. When present it is never the first paralytic symptom. The feet are invariably affected first, and are invariably the last to recover. This is so in my experience at least.

The earliest muscles to become paretic or paralysed are the tibialis anticus and extensor longus digitorum pedis. The child drops its toes in walking, and the nature of its ailment can be recognized as it walks into the consulting room. There are, of course, other causes of this form of paralysis, such as arsenical and alcoholic poisoning and beri-beri; but I have not so far met with any instance in which these have complicated the diagnosis. The peronei are also frequently attacked and occasionally suffer worse than any other muscles. In the forearm the extensors of the fingers are attacked before those of the wrist, as is the case in adults. Paralysis of the small muscles of the thumb, accompanied by wasting, is often seen in severe cases, and the interossei may be similarly affected. In the muscles of the hand paresis and wasting appear to be simultaneous, and in this respect they differ from the muscles of the forearm and leg.

A very common symptom attending the onset of the paralysis is pain in the muscles of the legs. This comes on especially at night, when the child frets and cries and cannot go to sleep. The pains are usually accompanied by tenderness of the muscles, but may be relieved by warm fomentations. When severe they are accompanied by evident cramp, affecting particularly the calf muscles. I have seen extremely painful cramps of the back muscles, and of other muscles, also, as in a case of which I made the following note during an attack: "The child lies on its face, embracing a pillow with both arms. When the pain comes on the head is thrown back by spasmodic contraction of the back muscles, the muscles of the arm are in tonic spasm, the legs are flexed, and hamstrings forcibly contracted. The legs are tender, and the slightest touch causes the child to cry out. The abdomen is not tender, and stands firm pressure wall. The mind is perfectly clear; the child is very intelligent." This was an extreme and very unusual condition.

If the child is removed from the source of the poison, the forearm muscles usually recover in a few weeks, those in the hand more slowly. The leg muscles are much more slow to recover; three months may suffice in recent cases, but some take much longer. Talipes equinus may persist after the anterior muscles have regained contractility, and it is then necessary to divide the tendo Achillis. Complete recovery is usual. In old cases with relapses—in some the history had spread over several years—I have seen apparently incurable paralysis, but in the light of recent advances in the treatment of paralyses I am not sure that any case should now be regarded as hopeless.

3. Diaphragmatic and Cardiac Paralyses.

One of the less frequent symptoms of plumbism is paralysis of the diaphragm, of which I have seen a fair number of cases, but only in patients suffering from the usual paralyses of the legs. In itself it is not a serious symptom, for this paralysis gives rise to no embarrassment of respiration in a child lying in bed, and will, indeed, escape observation unless carefully looked for. Most of my cases were old-standing cases of plumbism, but I have seen diaphragmatic paralysis in a first attack. When it comes on acutely, I regard it with grave anxiety, for two or three times I have seen it accompanied or followed by fatal cardiac weakness. This is characterized by excessive weakness and rapidity of the pulse, intense general depression, and vomiting, without any intellectual disturbance; death occurs usually within twenty-four hours. I am unable to explain these symptoms, but they are very similar to some fatal cases of poisoning by diphtheria toxins, which occur in a late stage of that disease.

4. Remote Effects: Anaemia, Albuminuria, Nephritis.

Old-standing cases of plumbism are frequently distinctly anaemic, with sallow complexions. This is not so in recent cases.

Transitory albuminuria is not uncommon. Chronic interstitial nephritis is, I believe, an occasional but rare complication. A case which ended fatally has been reported by Dr. T. E. Green.²

5. Eclampsia.

Now and again children suffering from chronic plumbism develop eclamptic seizures. These do not differ symptomatically from convulsions due to other causes, but are of a severe type and not infrequently fatal, even when treated with the greatest skill from the onset. I believe that a good many cases of fatal convulsions in Brisbane are the effect of undetected plumbism. A few months ago I was called in consultation to see a boy dying from protracted convulsions. Careful inquiry failed to elicit any exciting cause, but an examination of the gums revealed the typical appearances caused by the deposit of lead sulphide.

6. Acute Optic Neuritis associated with Oculo-motor Paresis.

The far-reaching effects of plumbism in childhood are not yet exhausted. The most insidious and most damaging I have not yet mentioned. There is a totally distinct clinical complex, first recognized by myself long before I had any idea of its causation, and described at the Inter-colonial Medical Congress of 1892 as "a form of cerebral disease characterized by definite symptoms, probably a localized basic meningitis." I mention this purposely, as it is with meningitis and cerebral tumour that this form of lead poisoning is most likely to be confounded. This mistake has been made frequently in the past, and will no doubt be repeated in the future. Nevertheless, the diagnosis of this condition is not as a rule difficult to those familiar with it.

These cases are usually brought to us complaining of headache, which may be severe and persistent, and may be accompanied by vomiting. We observe at a glance that they have an internal squint. Sometimes the squint is the symptom for which they are brought, but on the other hand it may be so slight as to have escaped the mother's observation. On inquiry we learn that the squint is recent, and on examination we find that it is due to paresis of one or both external recti. This ascertained, we proceed to examine the optic discs, and discover optic neuritis, so intense in most cases as to be evident even to the tiro in ophthalmoscopy. This symptom complex is due to lead poisoning. We proceed to verify our diagnosis. In default of any corroborative evidence of lead, we must remember that a cerebral tumour, cerebral tubercle, or cerebral syphilis, might produce exactly these symptoms and no other; but it has happened in all the cases of tumour or cerebral syphilis that I have seen, that other symptoms not characteristic of lead have been present; so that the probability of a case such as I have described, occurring in Brisbane, or any town similarly painted, being due to any other cause than plumbism appears to be very small.

In more severe cases, which are less often seen, the cerebral and ocular symptoms are more pronounced. We

see these children usually in bed. In addition to headache, there may be severe pains in the back of the neck running down into the arms. The head may be retracted. Both external recti are completely paralysed, and some, or all, the muscles supplied by the third nerve (except the levator) may be paralysed also. There may even be immobility of the pupils. These cases are frequently confounded with meningitis. It is a natural, perhaps an inevitable, mistake, but inspection of the gums may save even the inexperienced from error. If the gums are clean the bitten finger-nails may arouse his suspicions. The course of the case is not that of an ordinary meningitis. The child almost always makes a perfect recovery except as to vision. I can remember only one of these cases that proved fatal, unless convulsions supervened, but there is almost always some loss of sight, which may be total.

Blindness due to optic atrophy, and therefore permanent and hopeless, is the unfortunate consequence of a large percentage of these cases of plumbic ocular neuritis. It may occur in the milder cases in which only the optic and sixth nerves are involved; when the third nerve is implicated, escape from at least partial loss of vision is rare. In many cases the blindness is absolute, in some there is only great impairment of vision. The suddenness with which this is lost has struck me forcibly. One day there may be nothing to suggest defective vision, within the next two days the child may be found apparently quite blind. Fortunately the great majority of the milder cases recover perfect vision if they come under treatment early.

It is a remarkable thing that children who suffer from ocular neuritis do not suffer from limb palsy. I have never seen the conditions combined. Dr. Lockhart Gibson has once observed wrist-drop in a child suffering from ocular neuritis, and two or three cases in which the limb palsy had preceded or followed ocular neuritis at an interval of many months. Cases of the two forms occurring in the same family are not so rare.

A large proportion of lead poisoning in children take on the form described in this section. Of 89 cases of plumbism admitted during the last four years into the Children's Hospital, Brisbane, 20 were of this variety, that is, 22.5 per cent.

T. Chronic Optic Neuritis and Atrophy without Paresis of Sixth Nerve.

These cases are much less often seen than the preceding. I have no experience of them, and Dr. Lockhart Gibson has only recognized them within the last three years. The symptom complained of is loss of sight, and the ophthalmologist is most likely to be consulted. The course is slow, and they respond well to treatment, if not too advanced. Diagnosis can only be made by finding evidence of lead in the system, or of exposure to the causes of plumbism.

PREVENTIVE AND CURATIVE MEASURES.

There may be, and doubtless are, other conditions attributable to lead than those recognized, but those I have described are sufficient to show that lead poisoning among children is a serious evil. Prevention is easy. Paint containing lead should never be employed on outside surfaces, such as fences, walls, and particularly verandah railings, in places where children, especially young children, are accustomed to play. Zinc-white, or some other paint free from lead, should be substituted. Unfortunately we in Brisbane still from ignorance, no longer excusable, allow our houses to be poisoned traps for children's fingers, and every year furnishes its quota of ill-health and suffering, crippling, hopeless and permanent blindness, and occasionally death as the consequence. This is certainly a matter which calls for legislative interference.

The curative treatment consists essentially in removing the child from the source of the poison. We adopt also other measures calculated to encourage the elimination of lead from the system, but to what extent these are efficacious must be a matter of doubt. By themselves they are useless. Removal of the child into a hospital is a short way of taking it out of danger. But after its discharge it will surely relapse if the home conditions remain the same. We must find out exactly where the child got the poison, and warn the parents to keep him away from

it, remembering that lead paint is only dangerous (1) when it is fresh and sticky, or (2) and more especially when it is dry and powdery. It is also well to break the child of the habit of nail-biting or thumb-sucking, which by the way is no easy task. The most effectual method is to put straight splints over its elbows. This may seem harsh treatment, but milder methods usually fail.

REFERENCES.

¹ *Australasian Medical Gazette*, 1904; The Transactions of the Australasian Congress. See also *BRITISH MEDICAL JOURNAL*, 1908, vol. ii, p. 1498. ² *Australasian Medical Gazette*, October, 1897.

A CASE OF CROSSED PARALYSIS.

By GORDON LAMBERT, M.D. CANTAB.,

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THE following case, though not very unusual, presents, I think, certain features of interest:

W. J., aged 59 years, farmer and dairyman, had been very "irritable" for some days before February 7th. On that day he got out of bed at 4 a.m. to pass urine. Immediately after doing so, he became very giddy and fell down; but he did not lose consciousness. He returned to bed, with assistance, and afterwards insisted on making several attempts to walk. I was summoned to him at 8 a.m., and I found the following physical signs:

Nervous System.—Upper segment paralysis of the face on the left side, partial paralysis of the right motor oculi nerve, ptosis, slight divergent strabismus, limitation of movements of the eye (chiefly inwards), diplopia. On my arrival the right eye was completely closed; but the patient persisted in making voluntary efforts, until he completely raised the upper eyelid. The pupils were equal and reacted to light and accommodation. The left arm and leg did not show any loss of motor power; but the knee-jerk was distinctly brisker on the left side.

Heart.—Apex beat not palpable. Apex localized by percussion and auscultation in fifth left interspace, nipple line. Heart sounds faint and distant; aortic second sound not accentuated. No bruit.

Pulse.—Regular, 90 odd, of small volume, moderate tension. Radial arteries tortuous and uniformly thickened; no discrete, nodular thickenings.

Urine.—Specific gravity, 1018. No albumen. (Urea not estimated.) Patient has passed urine during the night for several years, nearly every night. He estimates that he passes from three to four pints per diem.

Previous History.—The patient had been a temperate man, and had done a great deal of heavy manual work. He had had one severe illness only, an attack of influenza 16 years ago, during a severe epidemic.

Progress and Result.—Motor power was rapidly regained during the first week, and on February 22nd the facial paralysis was no longer evident. Diplopia persisted; but the limitation of movements of the right eye was distinctly less. The ptosis had quite disappeared. The pulse was regular, 82, of moderate volume and tension. The physical signs in the heart were as before. The urine—specific gravity 1018—contained no albumen.

The case is clearly one of crossed paralysis due to a lesion in the right crus cerebri, involving the fibres of the motor oculi nerve and the facial fibres of the pyramidal tract. It is justifiable to assume that the nucleus of the motor oculi is not involved.

The chief interest centres on the nature of the lesion. Is it a very limited haemorrhage, presumably from one of the deeper small arteries (of the "ganglionic system") or thrombosis of such a vessel? Arterio-sclerosis is certainly present; but the condition of the heart and pulse, taken in conjunction with the history of severe influenza, seems to make the probability of haemorrhage doubtful. The onset is interesting. The giddiness and falling are, of course, not unusual after evacuation of the bladder—the splanchnic vessels being rapidly filled with blood and transient ischaemia of the brain established. There is no evidence that the act of micturition is accompanied in this patient by any special effort of straining, which might produce a haemorrhage, that is, by raising tension, in spite of the upright position and the evacuation of the bladder. The involvement of the facial fibres and the escape of the arm and leg in a lesion of the crus cerebri is also interesting, as indicating very limited damage.

THE Committee on Legal Affairs of the Massachusetts Legislature, to which the Antivivisection Bill was referred this year, has reported leave to withdraw. This, it is said, is the eleventh or twelfth time that such a bill has been presented to the legislature and has met with the same fate in committee.