PLUMBIC OCULAR NEURITIS IN QUEENSLAND CHILDREN.

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THE title "ocular neuritis" was applied by me to the cases which form the subject matter of this paper in 1897,¹ because the term includes with the optic nerve the external ocular nerves, whereas "optic neuritis" would not include the latter. My 1897 paper diagnosed the cases, but failed satisfactorily to point out a source for the poison—even, in fact, suggested a source since proved to be erroneous, namely, tank water.

My 1904² paper demonstrated the source of the poison, and this matter was further elaborated in a paper read by me at the Seventh Australasian Medical Congress (1905) and published in its *Transactions*. I am anxious that an account of the cases should have a wide circulation, not only because I think they throw more light on the special clinical manifestations of lead on the ocular nerves than has been recorded by others, but because they appear to constitute a type of ocular disease unknown in the medical literature of Europe.

Isolated cases are likely to be regarded as basal meningitis or cerebral tumour. Our Brisbane cases were, in fact, diagnosed in the first instance as an unusual form of basal meningitis and described as such in the *Transactions* of the Third Intercolonial Medical Congress (1892) by A. Jefferis Turner. I then concurred. Since I ascertained in 1897 that they were really cases of lead neuritis, Turner has accepted and supported that diagnosis.

I am satisfied also that cases cannot be very infrequent in warm climates, other than Queensland, where sources of the poison similar to those pointed out in this paper are available.

A recognition of the source of the poison and of the method by which it is ingested is most important for prophylactic reasons, and I have since 1904 found it an aid even in diagnosis.

I shall not more than refer to the cases of wrist-drop and ankle-drop due to lead which are so frequent in Queensland children and to which attention was first called by the staff of the Brisbane Hospital for Sick Children in the *Transactions* of the Third Intercolonial Medical Congress (1892), and later and more fully by Dr. A. Jefferis Turner.³ My Brisbane colleagues have since 1904 agreed with me in attributing the poison in the paralytic cases to the same source as in the ocular ones.

Dr. Sydney Stephenson's note,⁴ "On a Little-known Type of Amblyopia in Children," has reminded me of my neglect to send this paper some years ago. I have seen no reference to my work in any English or American publications except Osler's *System*.

It will be convenient to give first a somewhat comprehensive definition of the acute cases; secondly, some account of the steps which have succeeded in proving the source of the poison, and how it is ingested; and, thirdly, an account of some illustrative cases, including a chronic variety.

The disease is characterized by the sudden onset, with or without preliminary colic, vomiting, and constipation, and with or without severe headache, rigidity of the neck, or retracted head and acute pains at the back of the neck, of paralysis or paresis of one or both external recti, accompanied by optic neuritis or choked disc, and unaccompanied by rise of temperature or by albuminuria; accompanied also by the well-known blue line on the gums (produced by the deposit of the black sulphide of lead), by the finding of small but distinct quantities of lead in the urine, by the fact that the patient has access to painted surfaces which have either been recently painted, or more often whose paint has become powdery and easily detachable, this powder being the very soluble carbonate of lead; accompanied further by the fact that the patient bites his or her nails; the exceptions suck their fingers.

If in Queensland a child under eight years of age is brought to the consulting room suffering from a recently acquired internal squint, and this squint is found to be due to paralysis or paresis of one or both external recti, one expects to find double optic neuritis, and finding this, the provisional diagnosis is lead neuritis.

The acute ocular cases are almost confined to children between the ages of 2 and 8 years. These children are more accustomed to play and hang about verandah railings and garden railings than older children, and the paint of garden and of verandah railings after a year or two of exposure to the weather becomes powdery and easily detachable. It is composed, the Queensland Government analyst has ascertained for me, of a soluble carbonate of lead. It adheres readily to the hands, especially when these are moist from perspiration in warm weather. The cases I have shown to be most common in warm weather. Of 85 cases of lead poisoning, all varieties, admitted to the Hospital for Sick Children during five years, 69 were admitted during the seven summer months and 16 during the five winter months. I have also been able to demonstrate that a majority of the children bite their nails; those who do not do so suck their fingers. This observation explains how the lead is carried to the mouth, as well as the fact that in a family of several children sometimes only one child is affected. It was first suggested to me by the mother of two affected children. Upon my explaining to her that the children carried the powdery paint to their mouths by their fingers, she exclaimed, "And those are the only two of my children who bite their nails."

Many older children, some of them occupants of the blind asylum, with post-neuritic atrophy either partial or complete, can be, when their histories are pieced together, diagnosed as results of lead neuritis or of lead encephalopathy. An occasional case is observed where there has been no history of an acute attack nor of squint, but a child is brought with defective sight and slight or marked post-neuritic atrophy in one or both eyes, whereby process of exclusion lead can be diagnosed as the cause. In some of these rest from near work, correction of the nail-biting habit, and abstention from handling painted surfaces, together with a course of potassium iodide, leads to recovery of sight in one or both eyes. I will quote a case in point.

Since we have known the source of the poison it has been possible to warn parents, and so to avoid fresh attacks, which were formerly very common, especially in the foot and wrist drop cases. Where possible we have induced parents to substitute zinc white or other basis for white lead upon the parts of their houses and railings within reach of children. In some cases where we have known these warnings to be disregarded the children have been brought back with relapses. The fact that we observe in the out-patient department some children with marked blue lines and no acute symptoms attributable to lead, leads us to believe that the acute attacks are often a result of an increased ingestion of the poison during a few successive days.

It is a remarkable fact that we have very seldom observed ocular attacks in a child during an attack of foot or wrist drop, but before we were able to warn parents of the source of the poison we observed occasionally that a child who had been previously the subject of ordinary lead palsy would be readmitted, perhaps the following summer, suffering from ocular neuritis, or vice versa.

I have found it a help, in breaking children of the nail-biting habit, to apply anterior arm splints, so that they cannot carry their hands to their mouths. This not only interrupts the habit, but can be used as a very useful threat upon any tendency to its resumption.

Our first thought prior to 1897, that the source of the lead was water from galvanized iron tanks, was disproved not only by failure to find lead in the water of these tanks, but also by the fact that the presence of metallic zinc in water prevents it from holding lead in solution. Zinc is always to be found in the water of galvanized iron tanks.

Generally speaking, I see 4 to 6 cases each year of ocular neuritis due to lead. Between the years 1891 and 1897, 24 cases were admitted to the Hospital for Sick Children, 7 of whom became quite blind from optic atrophy.

Between the years 1897 and 1905, 30 cases were admitted, making 54. This number is exclusive of cases seen in my own consulting-room. The total number of cases of plumbism admitted to the Hospital for Sick Children between 1891 and 1905 was 200—that is, including, with the cases of wrist and foot drop, the 54 ocular cases. From July, 1905, to July, 1908, 62 cases of plumbism were admitted, 13 of which were ocular cases.

The greater number of our cases of ocular neuritis in Queensland have been of the acute variety, and they are more likely to be mistaken for basal meningitis than for cerebral tumour.

In most of the cases observed by me I have been of the opinion that the optic neuritis was due to direct irritation, not only to the vaginal sheath of the optic nerve, but also to the interstitial substance of the nerve itself. The ophthalmoplegia externa I have attributed to peripheral neuritis. The very severe cases, however, certainly suggest some lead encephalopathy with distension of the ventricles, and it is not unlikely that some of the obscure brain cases which in Queensland children have not been attributed to lead have been of this nature. The ocular cases do not die, and there is an absence, therefore, of post-mortem evidence of the exact nature of the involvement of the retrobulbar portion of the optic nerve.

A peculiar feature of the Queensland cases has been the extreme swelling of the discs, 5 to 6 dioptres of swelling being quite common, less than 3 dioptres uncommon. There are seldom hæmorrhages, and never large ones. One other peculiar feature is the fact that optic neuritis has never been observed unassociated with some paraplegia externa. As a rule, one external muscle is paralysed or paresed, often both. In only one case, where only one muscle was paralysed, was it other than an external rectus. In that case it was an internal rectus. In the most severe cases all the external muscles are paralysed, and the eyeballs are quite stationary. The levator I have not seen implicated. As a rule, when paraplegia externa is complete there is paraplegia interna also. Cases of paralysis of all the muscles have resulted in complete blindness, with post-neuritic atrophy of the discs. The ophthalmoplegia externa has always been recovered from. I have seen only one case in whom the optic neuritis was unilateral. In it the paralysed external rectus belonged to the other eye.

The excellent sight possessed by some of the cases with several dioptres of swelling in the discs is astonishing. If the cases can be removed from their surroundings and confined to bed before swelling has existed for long and sight has remained good, the outlook is hopeful, although not without reservation. Fortunately the presence of squint draws attention to the child even more perhaps than the complaint of defective sight, unless it be very defective. In the mild—that is, early—cases squint only is complained of. Only when it is noted to be paralytic and accompanied by optic neuritis is the serious nature of the case recognized.

If the cases are to be explained by interstitial neuritis of the optic nerve itself, it is not difficult to understand that the optic nerve fibres, which from their possession of no neurilemma or grey sheath must be more vulnerable to the pressure of inflamed interstitial tissue than the fibres of any other cranial nerve, should be damaged beyond recovery, although the sixth and other ocular nerves invariably recover from a neuritis which is surely interstitial.

The chronic cases have a history of defective sight at least for months; and if the optic discs are not atrophic, they are pale, with somewhat irregular and blurred edges. They present, in fact, a halfway appearance between a normal disc and one in a state of complete post-neuritic atrophy.

The acute cases which do not run a favourable course present ultimately appearances which vary according to the amount of damage done—that is, more or less complete post-neuritic atrophy.

The defective vision in both acute and chronic cases has been an all-round diminished acuity with a concentric narrowing of the fields. As a rule, the colour sense is not relatively more reduced than the light sense. A child with vision reduced to fingers at 5 or 6 yards will show no central scotoma for either white or colour.

Some of the apparently chronic cases were probably cases which at the outset were acute and unrecognized. Occasionally the history suggests this and sometimes tells of more than one successive summer attack.

The comparative infrequency of plumbic cases in the Southern States of Australia may be explained by the comparative infrequency of verandah railings in the houses and cottages there. In Queensland all cottages or wooden

houses are raised several feet from the ground by posts or stumps, and verandah railings are therefore necessary.

One difficulty in diagnosing these cases when isolated is the fact that every case does not furnish every aid to diagnosis. Sometimes a blue line is absent from the gums, though possibly present in an unaffected member of the same family. Sometimes the sample of urine sent fails to yield lead on analysis. In one case this occurred until after the administration of iodide and of pilocarpin, when lead was found in the urine. Failure to find lead may often be explained by failure to collect the urine immediately on the admission of a case to the hospital—that is, until after it has been removed from the source of the poison.

Of four cases under treatment at the same time in 1904,⁵ all presenting the same symptoms, hardly differing in degree, all recovered perfect sight, two failed to give lead in the only sample of urine (a week's) sent to be analysed, while the urine of the other two cases contained lead; one of those whose urine contained lead had no blue line, and one whose urine failed to yield lead had no blue line. Three bit their nails. The fourth, aged 2 years, sucked his fingers. His playground was a verandah, which not only possessed painted railings but whose floor was painted. The paint from the floor was easily detachable in the form of a fine powder, and yielded abundant lead on analysis. In two of the other three cases also lead was available in the form of powdery paint, and in the other the paint was still sticky from its freshness.

This paper is largely a synopsis of my 1905 paper,⁶ and as I cannot improve on some of the illustrative cases given there, I shall use them again, and add a chronic case.

I shall first relate three types of differing intensity, and then a few other illustrative cases.

1. A Mild Attack, but by no means the Mildest.

E. B., aged two years, sent to me by Dr. Clowes with a history of squinting for five days and of defective sight. Symptoms came on suddenly; otherwise in good health, though a little fretful. Slight tendency to retraction of the head. Paralysis of right external rectus; paresis of left external rectus. Double optic neuritis, with several dioptres of swelling in each disc. Pupils not enlarged, and react well to light and accommodation. No blue line in gums. Sucks his fingers. Special opportunities for powdery paint to adhere to his hands from the verandah railings and verandah floor, upon which he spent his days; 0.32 mg. of lead found per litre of urine examined. Result, complete recovery.

2. A More Severe Case.

K. R., aged 6½ years, ill two weeks, began with vomiting, violent abdominal pains, and constipation. Eyesight began to get weak a few days ago; to-day cannot see at all.

Condition on Admission.—Very restless, continually crying out as if in pain. Head slightly retracted and neck held stiffly. Pupils sometimes large, at others small, react slightly to a lighted match when large. The only indication that ophthalmoplegia externa is not absolute is a right eye very slightly directed outwards as compared with the left. Six dioptres of swelling in each disc. For the first week the patient's pulse was irregular at times. Four days after admission she was still unable to see a lighted match, and ophthalmoplegia externa was absolute. Twenty-five days after admission only one external rectus remained quite paralysed, the other muscles all acted slightly, some fairly well. Counted fingers at one yard with her left eye. Three weeks later at seven yards, with left eye. Very little sight with right eye. Now (three months after admission) left eye sees certainly $\frac{1}{2}$, probably $\frac{3}{4}$. Right eye cannot count fingers. The swelling in the discs had practically disappeared a month after admission. Discs both present an appearance of partial post-neuritic atrophy. It is possible that this case had a little lead encephalopathy. She is now very well. The sight recovered was exceptional when we consider how complete was the paraplegia externa. Neither of these cases has had any further attack. The source of poison was demonstrated to their parents.

3. A Still More Severe Case.

A. R., aged 5½ years, was seen in consultation in 1900, before I had grasped the source of poison. He had then been ill for two or three weeks, and his case had been diagnosed by Dr. Meek as lead neuritis. With this I concurred. Illness began with pain down the arms, spasmodic and not always in the same arm; no paralysis. No temperature. Retraction of head and pains about head and back of neck. Cries out with these pains on movement. No colic and eats well. Lead had been found in the urine. Both external recti paralysed. Five to six dioptres of swelling in each disc. Counts fingers at 18 in. When seen two months later he had in the meantime suffered from wrist-drop but not from ankle-drop, and he was still unable to dorsiflex his wrist. Pupils natural. Paralysis of external recti had nearly disappeared. In good health; each disc atrophic.

Four years later he counted fingers at 2 ft. with one eye and at 6 in. with the other. No paralysis of any muscle.

4. The Gradual Improvement in Sight which may Occur.

M. S., girl, aged 5 years, an attack of severe lead ocular neuritis in 1899. Five dioptries of swelling in the discs, and much neuro-retinitis encroaching on the macular regions. Paralysis of right external rectus; paresis of left external rectus. Recovered with damage to the retinae and some post-neuritic atrophy of discs. A year later counted fingers at 6 yards and saw $\frac{1}{2}$. Two years later right eye saw $\frac{1}{2}$, left eye $\frac{1}{2}$ partly. Now (1905) sees right eye $\frac{1}{2}$, left eye $\frac{1}{2}$. Her age discounted to some extent the accuracy of these tests. Her discs still show marked evidence of previous neuritis, and are pale and irregular. There are marked evidences of old retinitis between the disc and macula in her left eye. In each fundus the veins about the macular region are somewhat dilated. She has no scotoma for colour, and her fields show only slight concentric contraction, central vision being most acute.

A sister of this patient suffered from colic and very marked blue lines in her gums, also from twitching of shoulders and arms.

5. Monocular Diplopia.

E. M., a peculiarly intelligent girl of 8 years, was admitted in January, 1904, with a typical attack of lead neuritis, with lead in the urine and blue lines on the gums. She recovered quickly, had ultimately regained perfect vision. She had been ill for three weeks, and had squinted for one week before admission. On admission, she saw double with both eyes open and also with the right eye closed, but single with the left eye closed. The swelling of the discs only amounted to 2 dioptries. I noted that there appeared to be relatively more exudation in the horizontal meridian of the left fundus than in the vertical. The case was, however, examined hurriedly, and a more careful examination left for two days later. The monocular diplopia had, however, then disappeared. It had lasted for seven days. I believe it to have been due to temporary astigmatism.

6. A Chronic Case.

C. B., aged 11½ years. Seen July 28th, 1905. History of defective sight for last year or two; difficulty in seeing black-board at school; reads $\frac{1}{2}$ partly; right eye a little the better; discs large and pale, with ill-defined edges and seen well with +2 D, whereas the general fundus did not bear a + glass; no paralysis of any ocular muscle; nails bitten; skin of hands moist, even in winter; very distinct blue lines in gums round incisor teeth; no central scotoma; diagnosis probably lead. To stop biting nails, to stop school, and take iodide of potassium in 5-grain doses three times daily. October, 1905, tried $\frac{1}{2}$, and saw all of $\frac{1}{2}$. January, 1906, saw two letters of $\frac{1}{2}$; said to be in much better health; allowed to go to school. April, 1906, sees $\frac{1}{2}$. Discs do not bear a + glass; they are large and rather pale, with the edges slightly irregular, in a state, in fact, of slight post-neuritic atrophy; no difficulty in seeing at school. The slight optic neuritis in this case when first seen had no resemblance to the intense neuritis seen in the acute cases.

Treatment has consisted in removal from their homes, confinement to bed, dilute sulphuric acid and saline laxatives, and hypodermic injections of pilocarpin daily—the last continued for six weeks. After the first few days, iodide of potassium in the proportion of 5 grains thrice daily to a child of 8 years.

In cases with symptoms of encephalopathy I have found latterly that lumbar puncture and the draining of some 2 drachms of fluid is a help, and repeat it every few days if the first puncture gives indication of increased tension.

REFERENCES.

- ¹ Australasian Medical Gazette, 1897. ² Ibid., 1904. ³ Ibid., 1897. ⁴ BRITISH MEDICAL JOURNAL, July 18th, 1908; Ophthalmoscope, August, 1908. ⁵ Australasian Medical Gazette, 1904. ⁶ Transactions, Australasian Medical Congress, Seventh Series, 1905.

A NOTE ON THE INFLUENCE OF THE NERVOUS SYSTEM UPON INFECTIVE PROCESSES.

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In previous publications which related to the influence exerted upon cancer by the nervous system I usually mentioned cases in which certain infective processes occupied the same nerve areas as the cancers under description.

In the present article I wish to enlarge on the mere statement of fact, and to add something further. The relationship between the nervous system and infective processes can be described under two heads—neuro-vascular and neurotrophic. I believe they are quite distinct from one another.

1. Neurovascular.

It must be admitted that in most cases the neurovascular system is normal immediately before the actual moment of inoculation, and that in most cases the changes which occur in the neurovascular system after inoculation are due to the presence of the infective processes. On the other hand, it is obvious that if abnormal vaso-constriction or abnormal vaso-dilatation were present at the time of the

Fig. 1.—Area of neurovascular disturbance in a case of infection by *Streptococcus pyogenes* (Lenhartz).

inoculation, then the infective process might be modified by either of these conditions. However this may be, my object now is to point out that there is a marked change in the neurovascular system associated with some processes of infection.

The shaded area in Fig. 1 represents an intensely red skin due to the dilatation of blood vessels supplied by the posterior spinal root ganglion of the left third cervical nerve. The case is one of infection by *Streptococcus pyogenes* affecting the subcutaneous tissue of the chest,

Fig. 2.—To show sterno-mastoid area. (With Dr. Henry Head's kind permission, this diagram is taken from his article in *Brain*, vol. xvii, On the Disturbance of Sensation, with Especial Reference to the Pain of Visceral Disease.)

neck, and shoulder. Compare the red area in this case with the sterno-mastoid area in Fig. 2, which also corresponds to the area supplied by the posterior spinal root ganglion of the third cervical nerve. It is impossible to state the exact mechanism by which the blood vessels of this area became dilated. It may have been due to the direct irritation caused by the presence of the micro-organism in the posterior root ganglion, or by a reflex act induced by stimulation of the peripheral nerves. It is very unusual