A CLASSICAL CASE OF LEAD PALSY

BY

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Thirty to forty years ago lead neuritis in house painters was not uncommonly met with in hospital practice among the out-patients, but for a considerable number of years past it has been of increasing rarity, and is now scarcely ever seen. Its frequency among the painters was ascribed to uncleanly habits of eating their food without having previously been careful to cleanse their hands properly of paint. I was always somewhat sceptical of this explanation, which every medical student was familiar with, just as he was always ready to inform the examiner that a common instance of lead poisoning was the bus or cab washer, or other early riser, who obtained cheaply the first draught of beer drawn at "the public," after it had been standing all night in the lead pipe which led from the cask to the beer engine.

I much doubt whether the personal hygiene of the house painter is so much improved in this century as to account for the disappearance of lead neuritis among them. Almost certainly the real reason is that nowadays the various paints required are machine-mixed in the factory, ready for the painter, who formerly used to mix his paints himself.

Before developing lead neuritis he usually suffered once or twice from attacks of lead colic, and the well-known blackish-blue lead line could be demonstrated on the gums around the remaining teeth, usually extremely dirty, and entirely innocent of a tooth-brush's acquaintance.

In early cases the extensors of the fingers were demonstrably weak, and the right, or working, arm was usually the more severely involved. I remember well one such patient who developed total bilateral wrist-drop within three days of being given a mixture containing 5 grains of potassium iodide. The point has been much debated whether the administration of iodide internally influences the excretion of lead deposited in the tissues, but there is a good deal of evidence to indicate that it does so, and even that the lead thus brought into the general circulation may increase the existing toxic symptoms of lead poisoning to a serious extent. Certain peculiarities of muscle selection are usually noticeable in the less severe cases. The legs almost always escape entirely, and the knee-jerks are unaltered. In the forearms the supinator longus generally escapes, as does also the extensor ossis metacarpi pollicis. In the so-called upper-arm type there is additional weakness of the deltoids, and to a less extent of the flexors of the forearm. The flexors of the fingers and wrist are never affected, but occasionally there is seen an additional paralysis and atrophy of the intrinsic hand muscles, the thenar eminence being flattened and the interossei wasted. This atrophy of the hand muscles is permanent, and is due to a sub-acute poliomyelitis, though the dropped wrists and deltoid paralysis, being a neuritis, recover in course of time.

Clinical History

The following case was sent to me a month ago with the diagnosis of scapulo-humeral myopathy, which was made in a Northern hospital in which he had been resident for ten weeks.

A man, aged 51, living in the country in Lancashire, had been losing weight for eighteen months, which was ascribed to financial worry. In May, 1934, he caught a chill through getting wet, and shivered a good deal, this being followed by pains in both shoulders, more particularly in the left. The pains continued for some weeks,

and the arms began steadily to get weak from this time onwards, so that he was unable to raise them at the shoulders. In August his arms became very shaky on effort, and early in September he developed bilateral wrist-drop. The pains by this time had ceased, but the arms had been steadily getting weaker ever since May. In the middle of September he went into one of the large city hospitals in the North, where he remained for ten weeks, and the diagnosis of scapulo-humeral myopathy was made. He returned to his home early in December, and the next day was sent up to see me in London. He told me that towards the end of his stay in hospital he thought his arms were slightly stronger than when he was admitted. He had no complaint of his legs or trunk, his walking was perfectly good, there was no weakness of any of the muscles in the lower limbs, and the knee-jerks and Achilles jerks were brisk. On stripping him to the waist and asking him to raise his arms, there was evidently symmetrical paralysis of both deltoids, as he could not abduct either arm at all from his side. Flexion of both forearms was very weak, but he was able to do so against slight resistance; the biceps muscle was somewhat wasted, and the supinator longus was also very weak. There was complete bilateral wrist- and finger-drop, and no power of extension of the thumb or of any fingers. Flexion of the fingers and of the wrists was normal, and the intrinsic muscles of both hands were quite normal. There was no sensory loss whatever, and there was no muscular tenderness to be found. Examination of his gums for lead line was negative, as he was completely edentulous and wore dentures.

The picture, therefore, was one of symmetrical wrist-drop and shoulder-drop; the triceps muscles, the latissimus dorsi, the pectorals, serratus magnus, the trapezii, and all other muscles were quite normal. Clearly the condition was not a spinal muscular atrophy, and it was much too rapid to be a muscular dystrophy, whereas the picture of double wrist-drop and double deltoid paralysis very strongly suggested lead palsy—so strongly indeed that I was quite certain of the diagnosis, simply by looking at the man.

Source of the Lead Poisoning

On asking him what his work was, he said that he was a traveller in asbestos. This seemed a sufficiently harmless occupation, and in no way suggestive of contamination by lead. The next question was: "What is your water supply?" He replied: "Oh, that is very good. It is a private supply belonging to Sir X. Y., who allowed me three years ago to lay a pipe to my house from his supply. The water itself comes from springs up in the hills to the north." "What length of piping was laid down to your house, and is the piping made of iron or of lead?" "Oh, it is a lead pipe, about ninety yards long." "Do you drink much water yourself?" "Yes. I drink probably more than anybody in the house. I have two tumblers when I go to bed at night and two tumblers again first thing in the morning." "Where do you get your morning drink from?" "The kitchen tap." "Are you the first to go to this supply in the morning?" "Yes."

The source of the lead poisoning was now fairly obvious. A peaty water, and acids dissolving the lead in the pipe-line supplying the house both for drinking and for cooking, and his first drink in the morning was the water which had been lying all night in the leaden pipe. I told him to cut off this supply at once, and that it was not to be used any more for cooking or for drinking, but might be used for washing, and I told him to send me a quart bottle of the water that was drawn first thing in the morning from the kitchen tap. This I sent to a public analyst, Mr. Rowland, who reports 0.7 per 100,000 of lead in the water, which, as he remarks, is a dangerous percentage.

As regards treatment, I thought it wiser not to prescribe any iodide mixture, but to allow the neuritis gradually to get better with treatment by massage and galvanism, and the use of light splints to keep up moderate extension of his fingers and wrists. Aeroplane splints for maintaining abduction of the shoulders in order to avoid chronic overstretching of the deltoids are too cumbersome and impracticable, and I thought it best to leave them alone, except for massage, passive movements, and electricity.

It is worthy of note that this patient on no occasion had complained of colic, though severe lead neuritis had been present for several months.

A Case for Comparison

A case I saw twenty-three years ago may be recalled in comparison with the origin of this case. The man had about a year before taken a house with large grounds, and as the water supply was from an indifferent well, he had his house connected with a main water supply by a pipe laid for a distance of a quarter of a mile up the drive to his house. This had been contracted for to be laid in iron pipes. After living there for several months he began to suffer from chronic indigestion, which was diagnosed as being due to duodenal ulcer. Medical treatment did not improve this, and as he was getting worse a surgeon from Manchester decided that an operation was necessary for the duodenal ulcer, and this was arranged to be done on a certain day, and the operation would have been performed had not his butler on the day previous to that arranged for the operation developed bilateral wrist-drop. Although the butler had not suffered from colic, the wrist-drop was recognized as being due to lead neuritis, and this led to a revision of the diagnosis of duodenal ulcer in his master, and the operation was postponed. Investigation of the water supply proved that the piping which had been laid down consisted of lead piping and not of iron pipes, as had been contracted for. My patient was removed to a nursing home in another district, and he soon lost his abdominal pain, which was clearly due to lead colic and not to duodenal ulcer. He was therefore saved from operation by the illness of his butler, whose dropped wrists due to lead poisoning were incidentally an excellent testimonial to his character as a water drinker.

E. Gundersen, in a supplement to *Norsk Mag. f. Laegevid.* for December, 1934, records an epidemiological study of mumps during a hundred years in Norway (1834-1934). The disease never wholly died out, and it swept over large areas of the country in great epidemic waves, which recurred at remarkably regular intervals of eight to ten years. Of late there has been a tendency for these intervals to be shorter. They almost invariably began in the autumn, and reached their peak in January and February. From then onwards there would be a decline till the epidemic ebbed out in mid-summer. The ages of the patients were from 4 or 5 to over 30 years, and as long ago as 1852 it was remarked that children under 4 years were immune. Among the notified cases (and they constituted a minority) there was one death for every 2,900 notifications—a total of fifty-three deaths since 1867. They were due to such complications as suppuration of the parotid with septicaemia, brain disease, oedema of the larynx, and nephritis. Considerable variation was observed in the character of the different epidemics, some being very mild, and the patients being for the most part children. Other epidemics would show a preference for adults, who reacted with high fever and were prone to develop metastases. In some epidemics the skin would be often involved, in others the nervous system, the mucous membranes, the digestive or the urino-genital tract. Epidemic parotitis must therefore be considered as remarkably polymorphous in its extraparotid manifestations.

ACHRESTHIC ANAEMIA*

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Summary of Cases

Symptoms referable to the anaemia, such as dyspnoea, palpitation, and undue fatigue were severe in five cases, less prominent in two of them; pyrexia was not a feature. Digestive disorders were usually absent and the bowels were unaffected (except for occasional diarrhoea in Case I). Glossitis was absent. Icterus was present in some degree, but was not severe. Two patients lost weight. Involvement of the central nervous system was not found in any of the cases. Only one patient (Case II) had retinal haemorrhages. The liver was somewhat enlarged in two cases and the spleen was palpable in one. The teeth and upper digestive tract showed no peculiar involvement. The circulatory system presented no unusual features of importance.

The Wassermann reaction was negative in all cases, while the van den Bergh was negative in three, "indirect positive" in two, and gave a direct delayed response in one. Fractional gastric analysis established the presence of free hydrochloric acid in all cases, the quantities varying from hypochlorhydria to hyperchlorhydria, but large amounts of acid were secreted under the influence of histamine.

There was a megalocytic anaemia in every case; the mean diameter of the red cells varied from 7.78 to 8.43 microns, megalocytosis from 12.6 to 46.7 per cent., and the coefficient of variability from 9 to 17.7 per cent. The anaemia when first seen was severe (that is, less than two million red cells) in four cases; there were between two and three million red cells in the other three cases. All the patients except one (Case V) showed severe anaemia at some time. Nucleated red cells—between 4 and 16 cells per 400 leucocytes—were present in five cases. Anisocytosis and poikilocytosis were observed in all cases and varied according to the severity of the anaemia. Polychromasia was usually seen and occasionally punctate basophilia. Reticulocytes, except after intensive parenteral liver therapy, were low, varying from 0.3 to 8 per cent.; Case II showed a maximum of 14 per cent. when the anaemia was severe. The fragility of the red cells was normal in all cases where it was examined.

RESPONSE TO ANTI-PERNICIOUS-ANAEMIA THERAPY

Parenteral liver treatment was given in six cases, but there was no satisfactory response in four of these. Case I in the early stages of the condition responded to large doses of liver with reticulocyte peaks; but later this treatment had no effect. In Case III there was a small rise in reticulocytes (maximum 12.5 per cent.) after intensive treatment, but there was no subsequent response or rise in the red cell count.

Desiccated stomach was given in all cases, and a moderate blood level was maintained for a time at least. Severe relapses, in spite of treatment, occurred in four

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