Remarks

ON

SOME CLINICAL PICTURES ATTRIBUTABLE TO LEAD POISONING

WITH SPECIAL REFERENCE TO THE NEUROLOGICAL MANIFESTATIONS OF PLUMBISM*

BY

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Perhaps it may seem strange that I should have chosen as the subject of this lecture a disorder which is relatively uncommon, and which may be said to be upon the wane. An explanation to justify my choice would consequently seem necessary, although I feel that no apology is called for. Lead poisoning may be acquired from numerous unsuspected sources, and may appear under a variety of subtle guises. The diagnosis (and it is of diagnosis that I propose particularly to speak) may be at once suggested by the patient's occupation, for there are certain industries in which it is common knowledge that the workers are especially exposed. On the other hand, there are many occupations in which the risk is not so generally realized and many different ways by which accidental lead poisoning may be brought about. When the preliminary anamnesis fails to indicate the cause the possibility of lead poisoning will not occur to the practitioner unless it is suggested by the symptomatic features which the case presents. Hence the importance of an acquaintance with the varied symptomatology of plumbism. The clinical pictures which I purpose to portray may consequently prove helpful.

My first consultation after entering practice some thirty years ago (the case was one of lead poisoning) has for various reasons left an indelible impression on my mind. Such memories are invaluable; they constitute, indeed, an essential part of clinical experience, for, when the exceptional and unexpected are again encountered, they may indicate the line of approach or even serve to determine the diagnosis. For the purpose of this lecture I have, with undoubted benefit to myself, and I trust the result may be not unprofitable to you, reviewed my experience of lead poisoning and selected from my notes of some thirty cases, in a number of which the cause had not been previously suspected, several which exemplify some of the diverse manifestations of plumbism and raise certain problems of interest in this connexion.

OCCUPATIONAL AND ACCIDENTAL LEAD POISONING

Since the recognition of the source of poisoning plays so prominent a part in diagnosis it may be well in passing that I indicate some of the industries fraught with special danger and some of the sources of accidental poisoning. Occupational lead poisoning has long been recognized, and periodic inspection of the employees engaged in many industries is now insisted upon as a routine procedure. Medical practitioners are, indeed, under a statutory obligation to notify cases of lead poisoning occurring in a factory or workshop. A great deal of valuable information has been so acquired, and the risks of industrial poisoning, which are now known to be largely due to the inhalation of particles of lead contained in dust or in fumes, have been materially diminished by the application of appropriate preventive measures. Sir Thomas Legge,¹ in a recent paper on industrial lead poisoning, has classified the cases of plumbism reported to him over a period of twenty-five years in his official capacity as the

senior inspector of factories. The various occupations, including that of house painting, with the number of cases reported during the year 1925, were as follows: house painting, 88; electrical accumulators, 73; pottery, 45; ship breaking, 31; coach building, 23; smelting of metals, 22; ship building, 13; white lead, 12; enamelling, 9; printing, 8; paints and colours, 8; plumbing and soldering, 7; red lead, 7; tinning, 4; litho transfer making, 2; indiarubber, 2; other contact with molten lead, 17; paint used in other industries, 14; other industries, 29. There has been, it seems, a material and progressive diminution in the number of cases in all the occupations in which lead poisoning is specially met with, apart from electrical accumulators, a rapidly growing industry, and ship breaking, a new industry which came into existence during the war.

The term "accidental" is applied to cases of lead poisoning attributable to causes other than occupational exposure. These are very numerous, and often obscure. Contamination of drinking water derived from a peaty soil is, because of its comparative frequency, of special importance as a cause of chronic lead poisoning. The acids in the soft water act upon lead pipes, particularly if the pipes are new, and dissolve the lead. Instances are recorded in which epidemics of lead poisoning have been so produced. Poisoning may result when the quantity of lead in the water is not more than 0.01 grain per gallon. But it is not necessary that the lead should be in solution, for Sir Thomas Oliver² reports a case in which lead poisoning was caused by several lumps of white lead left in a cistern by careless workmen. The water was muddy in appearance from the precipitated lead, and lead was deposited on vegetables boiled in it. Among many other possible causes of accidental lead poisoning are the following: drinking aerated water from siphons with lead or pewter valves; drinking beer, wine, or cider which has been drawn through lead pipes; eating tinned articles of diet in which solder has been used in the tinning process; eating confectionery in the preparation of which lead chromate has been utilized for colouring purposes; drinking tea or eating food or sweets wrapped in lead foil, etc. Sucking the fingers and biting the nails after contact with white lead paint was the cause of the epidemic of lead poisoning in children reported by Lockhart Gibson³ of Brisbane in 1897.

Although poisoning is very rarely caused by lead when prescribed for medicinal purposes, it is sometimes so produced. Plumbism has, for example, been reported in cases in which lead acetate has been given to control diarrhoea. Infants have been poisoned by lead ointment applied to an excoriated nipple. Poisoning has also been observed in cases in which an ulcer has been dressed with lead lotion. The risk of absorption of lead through the unbroken skin is very small, but lead poisoning has been known to follow the application of hair dyes, cosmetics, and lotions.

Lead has seldom been employed with criminal intent for purposes of suicide or homicide; at one time, however, it was widely used, and is still sometimes employed, as an abortifacient. Nowadays the sale of diachylon is restricted under the Poisons Act.

A CASE OF "LEAD NEURITIS" IN WHICH WEAKNESS OF THE RIGHT HAND WAS THE PATIENT'S SOLE COMPLAINT

The first case to which I shall direct your attention is in many respects typical, since it exemplifies the form of nervous lesion most often met with in lead poisoning.

A lead smelter, aged 34, was seen on January 8th, when he brought with him a note from his medical attendant to the effect that he had a right-sided radial paralysis. The patient stated that he had worked as a lead smelter for ten years,

* A British Medical Association Lecture delivered to the Leeds Division, March 26th.

that to his knowledge he had never had any symptoms of lead poisoning, and that he had had no illness prior to the onset of his present trouble some three months previously. At that time, when feeling otherwise perfectly well, he noticed that he was unable to straighten the middle and ring fingers of his right hand. The weakness of the hand, which had come on gradually and without apparent cause, had, he said, been progressive. Although he had had some sharp pains at times, extending from the region of the right elbow to the dorsum of the thumb, he had noticed no numbness or other subjective sensation in the skin. Further, it was ascertained from the patient, only, however, in answer to direct leading questions, that for the previous two months he had been rather constipated, that his appetite had been poor, and that occasionally he had had some pain in the lower abdomen, though this had never been severe, and from his description had not been definitely colicky in character. Again, he stated that there was no weakness of the left hand, and that he had been doing a full day's work up to the previous day.

Upon examination the patient's appearance was that of a healthy man, though he was possibly slightly anaemic. There was, indeed, a secondary anaemia, for the red cells numbered 3,610,000, the haemoglobin was reduced to 55 per cent., and there was a distinct punctate basophilia. The extensors of the right hand, notably those of the middle and ring fingers, of the thumb, and of the wrist, were obviously weak, though there was no definite wasting; there was no paresis of the small muscles of the hand, of the brachio-radialis, or of other muscles of the arm. The muscles of the right forearm and upper arm were hypersensitive on pressure. The deep reflexes of the right arm were less active than those on the left side. There was no sensory loss. Despite the patient's statement that he had noticed no weakness of the left hand, the power of the extensors of the left wrist, but not that of the fingers, was found to be definitely reduced when tested against resistance. There was no weakness of, or alteration in, the reflexes in the lower extremities, and no tremor, nor was there a blue line on the gums, but it should be added that the patient was edentulous. Further examination revealed no additional physical signs. Lead was not found in the urine even after large doses of potassium iodide had been given for three days.

There can, you will agree, be no doubt as to the diagnosis of lead poisoning on the data I have presented to you. The patient's occupation, the bilateral though asymmetrical weakness of the extensors, the history of constipation, loss of appetite and abdominal pain, and the associated secondary anaemia with punctate basophilia place the diagnosis beyond question. But had it not been that the patient was a lead smelter, the possibility of lead might never have suggested itself, and the case might have been diagnosed on cursory examination as a simple radial palsy of undetermined etiology, for the patient's one complaint was of weakness of the right hand, he did not look ill, and it was only in answer to direct questions that the history of constipation and slight abdominal pain was elicited. There is indeed no reason why a lead worker should not develop a coincidental radial palsy. On the other hand, the gradual onset of the paresis affecting, in the first instance, the extensors of two fingers only, and the progressive course would probably, I think, have aroused the suspicions of those of you who have had some experience of lead poisoning.

A number of pertinent questions are raised by the case to which I have just referred. Why should a lead smelter develop symptoms of lead poisoning for the first time after working at his occupation for ten years? Sometimes, in similar circumstances, a history may be obtained to the effect that the patient had had influenza or some preceding illness, that he had been indulging in alcohol to excess, that he had been doing extra work, or that he had been especially exposed to lead immediately prior to the onset of his trouble. But in other cases, as in the present instance, no clear evidence of a determining cause is obtained. Then this case demonstrates a point I wish especially to emphasize—that wrist-drop due to a "lead neuritis" may be the first and only

obvious manifestation of lead poisoning. Why should this be? The problem is an interesting one, since it raises the whole question of the selective action of poisons. Is it not an interesting fact that the musculature of the upper limbs, and particularly the extensors of the wrists and fingers, should be picked out so often by lead, whereas the lower limbs so rarely suffer? I know of no form of multiple neuritis, apart from occasional post-diphtheritic types, in which this is so. The study of the exceptional sometimes throws light upon the commonplace. Numerous reported observations appear to indicate that muscular effort plays a part in determining the incidence of the paralysis. The facts in this case are suggestive in this connexion, for the patient, whose work chiefly consisted in ladling lead, informed us that he was in the habit of grasping the end of the iron ladle with his right hand while he supported the shaft of the ladle with his left. The employees in the works in which this patient was engaged were, he informed us, inspected once a month, when the inspector examined the gums for the presence of the blue line, looked at the eyes for anaemia, and noted whether there was any tremor of the hands. With the exception of a slight degree of anaemia, however, none of these phenomena were present here. On the other hand, this case demonstrates that a paresis of the extensors of the wrist and fingers may be the first noticeable manifestation of lead poisoning, and that unless this is pronounced it may cause little if any disability. Consequently it would seem to be advisable to test the power of the extensors of the wrists and fingers as a routine procedure. J. P. Leake⁴ has devised a special instrument for this purpose, but an ordinary dynamometer, which I am in the habit of using for recording progress in cases of radial palsy, appears to be all that is required.

ETIOLOGICAL CONSIDERATIONS

The cause of lead palsy is still the subject of debate. For long the paralysis has been regarded as secondary to the histological changes which are met with in varying degree, in individual cases, in the peripheral nerves and spinal cord. Gowers⁵ recognized two clinical types, in one of which the symptoms resembled peripheral neuritis, in the other, a progressive muscular atrophy of spinal origin. Messing⁶ in 1913 described inflammatory changes in the muscles which were not, he thought, to be accounted for by interruption of the nerve fibres. Hyslop and Kraus⁷ in 1923 were of opinion from their careful anatomical studies that lead palsy is due to a neuronitis which may affect any part of the neuro-muscular apparatus while Aub⁸ and his co-workers conclude that "in the light of more recent work on the chemistry of muscular activity and of lead within the body, it seems probable that the physiological lesion of lead palsy is in muscle."

CHRONIC LEAD POISONING SIMULATING MALIGNANT GASTRIC DISEASE

The following case is of considerable interest from the diagnostic point of view:

A business man, aged 62, whose home was in the North of Scotland, was seen on August 31st, 1900. A history was obtained from the patient to the effect that he had been an exceptionally healthy man up to two years previously, when he had had influenza, since which time he had been troubled with occasional attacks of abdominal pain, not related to food, and unaccompanied by vomiting. In February, 1900, six months before we saw him, he had a second bout of influenza, and after this never seemed to regain his strength. For some months he had been rapidly losing weight; in the spring he weighed 11½ st., whereas he now weighed only 8 st. 5 lb. Further, he informed us that for six weeks or more his arms had been becoming progressively thinner and weaker, and that he had been much troubled with what he described as boring pains in the arms, especially about the shoulders.

The patient was sallow, rather anaemic, and somewhat emaciated. There was pronounced symmetrical wasting and weakness of the upper limbs. So weak were the deltoids that he could only raise his hands for two or three inches from his sides. The extensors at the elbow were relatively weaker than the flexors, the extensors of the wrists and fingers were definitely weaker than their opponents, and the brachio-radialis had, I see from my notes, escaped. An occasional fibrillary tremor was to be seen in the wasted muscles. There was no sensory loss, nor was there any weakness or wasting of the lower limbs. The knee-jerks were present, equal, and not exaggerated, and the plantar reflexes were definitely flexor. The spine showed no deformity, limitation of movement, or localized tenderness. No tumour was detected in the abdomen, the stomach did not appear to be dilated, the liver and spleen were not enlarged, nor was there any glandular enlargement. The radial arteries were slightly thickened and tortuous, the blood pressure was not high, the heart was not enlarged, and there was no albumin in the urine.

Such were the leading facts. What was the diagnosis? Malignant disease of the stomach or bowel had been suggested some months previously, a diagnosis which might naturally suggest itself in view of the patient's age, the abdominal pain, the constipation, and the loss of weight. At a later date, the pain in the arms and associated wasting and loss of power had been attributed to secondary growths involving the cervical nerve roots. This diagnosis now appeared to be very unlikely in view of the facts that, although the abdominal pains had been present for two years, no tumour could be felt, that no peristalsis was observed, and that there was no enlargement of the liver or glands. Further, it seemed very improbable that the condition of the arms was to be explained by secondary growths involving the nerve roots in the cervical region, for pain in the back had not been complained of, nor was there any evidence of disease of the bone or of implication of the spinal cord.

When the clinician is confronted by a complicated case, and when the most suggestive line of inquiry fails to lead up to a diagnosis, other lines of approach must be explored. Now in this case, setting aside for the moment the history of abdominal pain and the loss of weight, the striking feature was the more or less symmetrical wasting and weakness of, and pain in, the arms. These phenomena were certainly indicative of peripheral nerve involvement. But a symmetrical neuritis confined to the upper limbs is very rarely met with apart from lead. Might the facts of the case perhaps be so explained? This would account for the frequent attacks of abdominal pain. The suggestion appeared very feasible. True the atrophic palsy was not limited to the extensors of the wrists and fingers, but this is by no means always so in lead poisoning. The fact that the brachio-radialis muscles had escaped was in itself suggestive. But the patient had had a great deal of pain in the arms, and pronounced pain is an unusual feature in cases of lead palsy. It might be, however, that some additional etiological factor, possibly the influenza, would account for this. The patient lived in the Highlands, and was it not consequently possible that there might have been a contamination of a moorland water supply? These facts were indeed most suggestive. Was any further evidence available? The gums were next examined, and a blue line was observed opposite the two lower incisor teeth. Although the blue coloration of the gums was very faint—indeed, it would probably have passed unnoticed unless it had been specially looked for—its presence was sufficient, in association with the other features of the case, to practically clinch the diagnosis. Upon further inquiry the patient informed us that, although it was true that the water supply of his house came direct from the moor, the present system had been laid down some eight years previously. Two additional lines of inquiry were still open to us. The urine was examined by a skilled analytical chemist, who reported

that he could find no trace of lead, but in his report he referred us to a recent authority who had examined the urine in fourteen cases of chronic plumbism and was only able to detect lead in two of these. On the other hand, on analysing the tap-water from the patient's house, he detected 0.12 grain of lead to the gallon of water. Conclusive proof of the diagnosis was afforded by the progressive improvement in the patient's condition after the correction of the faulty water supply. Among features of particular interest in this case were the great loss of weight and the unusual severity of the pains in the arms. No doubt the two attacks of influenza had, by lowering the patient's resistance, determined the symptoms.

Two methods are employed in arriving at a diagnosis; that is, the diagnosis may be based on the positive findings or arrived at by a process of exclusion. This patient was examined thirty years ago, and the diagnosis was suggested directly from the physical examination, was supported by the history, and confirmed by the detection of lead in the water supply and the rapid improvement after this had been corrected. Had the patient been seen to-day a number of diagnostic methods might have been employed for purposes of exclusion or confirmation which had not at that time been introduced—for example, the examination of the gastric contents for free acid, radiograms of the gastro-intestinal tract and of the spinal column, and the examination of the cerebro-spinal fluid and blood. But apart from the possible detection of a punctate basophilia, the occurrence of which in lead poisoning had not at that time been described, no additional evidence of a positive kind would have been obtained from these procedures.

ACCIDENTAL LEAD POISONING SIMULATING GASTRIC ULCER AND PROGRESSIVE MUSCULAR ATROPHY

Another case in which the source of the lead poisoning was the drinking water is of interest. The facts were as follows:

A married woman, aged 33, the wife of a gamekeeper, living in Inverness-shire, was seen on January 4th, 1915. The patient, I was informed, had suffered for some years from progressive muscular atrophy, and had been troubled from time to time with symptoms which had been attributed to a gastric ulcer, though she had never, so far as was known, vomited blood or passed blood by the bowel. The history was instructive. Bloodless since childhood, the patient stated that at the age of 19 she married and left her home in the South of Scotland to live in Inverness-shire. She had two healthy children, and had had no miscarriages. Some six years before I saw her, up to which time she said she had had no illness, the patient began to be troubled with attacks of abdominal pain, vomiting, and diarrhoea. About the same time she noticed that her hands, particularly the right hand, were shaky. These symptoms had continued to recur, although it was significant that when she left home to stay with her relatives in the Border country, as she did for a few weeks each year, her health always improved. A medical man whom she consulted sent her to see a distinguished surgeon, who, the patient said, agreed with the diagnosis, but was of opinion that operation was not called for. (It would be of interest to hear from surgeons of large experience whether they have ever met with, or operated upon, cases of lead poisoning sent to them with a diagnosis of gastric ulcer, intestinal obstruction, or appendicitis. I know personally of one case which was admitted directly to a surgical charge as a case of chronic appendicitis in which the symptoms were, I think, almost certainly to be explained by the lead poisoning from which the patient was suffering.) For some months before I saw her the patient had also been suffering from severe pains in the knees and elbows with, so she said, some swelling of the joints.

Upon examination there was a complete symmetrical paralysis, with very pronounced wasting of the extensors of the fingers and wrists. There was, too, great weakness and wasting of the small muscles of the hands. No fibrillary tremors were observed. The brachio-radialis and the muscles

of the upper arm and shoulder girdle had escaped. There was no sensory loss, nor was there any weakness or alteration in the reflexes of the lower limbs.

The distribution of the paralysis in association with the history was most suggestive of lead poisoning. There was no blue line on the gums, but the patient was edentulous. Upon further inquiry it was ascertained that a new water supply from a peaty well had been introduced into the patient's house a few months before she first commenced to suffer from the symptoms above described. Corroboration of the diagnosis was obtained when the tap-water was examined and found to contain 0.4 grain of lead to the gallon.

Among a number of interesting features presented by this case were the following: the occurrence of diarrhoea and vomiting in association with the abdominal pain; the improvement which occurred when the patient left home; the arthritic pains which are observed in some cases of lead poisoning; the great wasting and weakness of the small muscles of the hands in addition to the extensors; and the patient's statement that, although her husband and children looked pale, and had never felt well after the introduction of the new water supply, they had not suffered from colic or paralysis. Women are, it is affirmed, more susceptible to lead poisoning than are men.

So pronounced was the wasting of the hands and fore-arms in this case that I was very doubtful as to the ultimate recovery of the muscles, but when I examined the patient in January of this year she had completely regained the use of the hands and arms, and was looking in robust health.

LEAD ENCEPHALOPATHY, DUE TO DIACHYLON POISONING, SIMULATING BRAIN TUMOUR

Cases of lead encephalopathy are rarely met with nowadays. I can only recall two occasions upon which I have made this diagnosis.

A widow, aged 28, was admitted to hospital under my care on October 9th, 1929, when she complained of intense headache, vomiting, and double vision, which symptoms had troubled her for a month. The patient was somewhat confused and agitated, and when first seen it was not possible to obtain from her a very precise history of her illness.

Upon examination she was well developed, well nourished, but distinctly anaemic. There was a right internal strabismus, which the patient said had been present since early childhood. The pulse rate was rather fast (104), but there was no febrile disturbance. The retinal vessels were engorged, and the right optic disc definitely swollen. Otherwise the cranial nerves presented no abnormality, and there was no history of otorrhoea. There was no weakness, inco-ordination, or tremor in the upper limbs. A slight degree of ataxia was present in the lower limbs, but no distinct weakness. The knee-jerks and one ankle-jerk were absent, as were the abdominal reflexes. Both plantar reflexes were definitely flexor. There was no tenderness of the muscles on pressure. Apart from a soft mitral systolic murmur nothing abnormal was detected on examination of the thorax and abdomen. The radial arteries were not thickened, the blood pressure was not raised, and the urine contained no albumin. The result of the examination of the blood was as follows: Red blood cells, 3,650,000; haemoglobin, 50 per cent.; white blood cells, 12,800. Differential count: polymorphonuclears, 80 per cent.; small lymphocytes, 20 per cent.; basophils, 1 per cent.; no punctate basophilia. The Wassermann reaction was negative in both the blood and the cerebro-spinal fluid. The latter contained two cells per c.mm. There was no increase of protein, and the colloidal gold test was 0011000000. No lead was found in the urine, which was examined on two occasions by the Ganassini test. The Zondek-Aschheim test for pregnancy was negative.

The diagnosis of lead poisoning, which was at once suggested by the blue line on the gums, was confirmed by further inquiry, and by the subsequent history of the case. The patient was obviously agitated and worried,

and upon questioning her directly she admitted that, after missing a menstrual period two months previously, she thought she might be pregnant, and had bought an ounce of diachylon and two pennyworth of aloes, which she had made into pills and taken during the first fortnight of September. Further, she said that the headache and vomiting commenced immediately after she began to take the pills. Three days after the patient's admission to hospital she developed hallucinations and delusions of sex, and commenced to have severe attacks of abdominal colic. She was not taking potassium iodide at the time, a point of some interest, for cases have been reported in which symptoms of acute lead poisoning were precipitated by potassium iodide, in some instances long after exposure to lead had ceased. Gradually the patient's symptoms passed off, and when she was discharged, on November 21st, she was practically well. When seen on January 17th, 1931, she said that she felt in perfect health; she had had no further headache, abdominal pain, or vomiting, the knee- and ankle-jerks were present, the fundus oculi was perfectly normal, and she was no longer anaemic.

Lead encephalopathy, which is characterized by such symptoms as headache, vomiting, papilloedema, passing on it may be to optic atrophy, hallucinations, mental depression or exaltation, dementia, tremor, paresis, and epileptiform seizures, is undoubtedly very uncommon. Sir Thomas Legge¹ met with this diagnosis in only 1.1 per cent. of the male and 2.1 per cent. of the female cases reported between the years 1920-24. Byrom Bramwell² long ago insisted upon the importance of keeping this possibility in view in suspected cases of brain tumour. It is possible, indeed, that some of the reported cases of recovery from brain tumour are to be so explained. Mott³ was of opinion that lead encephalopathy was of the nature of an encephalitis. Aub⁴ and his collaborators take the view that the process is meningeal, basing their conclusions on the observations of (a) Hassin, who found a marked proliferative meningitis and relatively slight changes in the brain, of (b) various observers who have found increased pressure and an excess of lymphocytes in the cerebro-spinal fluid, and of (c) Camus, who produced excitability, "hallucinations," and convulsions just before death in dogs in which lead had been injected into the subarachnoid space by cistern puncture, whereas no symptoms followed when the lead was injected into the brain substance.

W. B. Ransom¹² drew attention in 1900 to the then very prevalent use of diachylon as an abortifacient, and suggested that something should be done to restrict the indiscriminate sale of the drug. Although diachylon is now included in Part I of the poisons schedule, the case to which I have referred shows that it may still be obtained and is still used for this purpose. Indeed, one should always bear in mind the possibility of lead poisoning when one is consulted by a woman of child-bearing age who complains of severe headache and vomiting, who is perhaps anaemic, and in whom there is a history of amenorrhoea. It is to be remembered that a single dose of diachylon may produce symptoms of poisoning, that these may persist for months, and that they may be aggravated by an intercurrent febrile illness.

SPASTIC PARAPLEGIA A CONSEQUENCE OF LEAD POISONING

Doubt was at one time expressed as to whether a pronounced spastic paraplegia is ever produced by lead. The following case is of interest in this connexion:

A gentleman, aged 56, was seen on July 18th, 1911, complaining of difficulty in walking, which had troubled him for three or four years. He had, he said, had no previous illness, apart from an attack of typhoid fever some twelve years previously. The history he gave me of his illness was

as follows. Four years before I saw him the patient had been troubled for some weeks with a pain about the right knee. At the same time, or soon after, he noticed some weakness of the hands and a tendency to drag the right foot when walking. The right leg became gradually stiffer, and soon after the left leg became similarly affected. He then began to have attacks of severe abdominal pain, vomiting, and constipation. His brother, who lived in the same house, commenced at the same time to be troubled with stiffness in the legs. After the weakness of the hands and attacks of abdominal pain had persisted for some six months the patient's medical man recognized that he was suffering from lead poisoning. He wrote me: "It was about this time that I suspected plumbism, and found that the patient had a blue line on his gums. The drinking water was analysed by Dr. Falconer King, who reported the presence of 0.15 grain of lead per gallon. Since then the water has been taken from a well free from lead contamination, and the patient has greatly improved in health and power of walking. He seemed always better away from home." According to the patient's statements, the attacks of abdominal pain disappeared at this time and have not recurred, the power of the hands was gradually regained, but the difficulty in walking has persisted.

Upon examination the patient had a good colour, and was well nourished. The only symptom of which he complained was difficulty in walking. His general health was, he said, excellent. The gait was very spastic, both lower limbs being affected to the same degree, though the patient was able to walk across the room with the aid of a stick. The muscles of the thighs and of the anterior tibial group were somewhat wasted. No fibrillary tremors were observed. The knee-jerks were exaggerated, and there was bilateral ankle-clonus and a bilateral extensor response. There was no sensory loss, and nothing abnormal was detected on examination of the upper limbs, cranial nerves, or other organs.

I heard nothing more of this patient until January of this year, when, in reply to my inquiries, he wrote:

"I am now 74 years of age, and there is little difference in my health. The loss of power has not extended to any other part of my body. This would seem to indicate that the disease has been stationary ever since I discontinued using the poisoned water. My brother, who also suffered from lead poisoning, died about two years ago, aged 70. He never recovered the use of his limbs. Latterly he had one or two shocks, and was bed-ridden for a while before his death."

That the patient had suffered from lead poisoning is clearly shown by the history of attacks of abdominal pain, the weakness of the hands, the presence of the blue line on the gums, the detection of lead in the water supply, and the improvement that occurred after the source of poisoning had been eliminated. There can be practically no doubt that the spastic condition of the lower limbs was due to the same cause, for the difficulty in walking developed at the same time and ceased to progress after the water supply was changed. It is interesting to note that the patient's brother was similarly affected.

J. J. Putnam,¹³ so long ago as 1887, described some cases of spasticity of the lower limbs in which he demonstrated lead in the urine. Kinnier Wilson,¹⁴ who in 1907 reported four cases of amyotrophy in men who, from the nature of their occupations, must have been exposed to lead, in each of which the onset was with double wrist-drop, while in three there was a certain amount of spasticity in the lower limbs, remarks, "One cannot but be struck with the progressive nature of the affection, which is so contrary to the ordinary forms of lead poisoning where cures are by no means uncommon." In the present case, however, the progress of the nervous symptoms appears to have been definitely arrested.

COMMENTARY

The cases I have referred to illustrate some of the varied clinical pictures attributable to lead poisoning. Is it not a remarkable fact, though it is an everyday

experience, that the same disease-producing agent may affect individuals so differently? We see this constantly exemplified, and yet we are often quite unable to account for the incidence of symptoms, and have to content ourselves by attributing the special symptomatology the case presents to some peculiarity of the virus, to personal idiosyncrasy or individual predisposition, or to the presence of additional etiological factors which it is not always possible to demonstrate or assess. While the majority of patients suffering from lead poisoning no doubt come to the practitioner complaining of attacks of colicky pain referred to the lower abdomen associated with constipation, of cramps, of general debility, and of other symptoms associated with anaemia, these symptoms may, as exemplified by more than one of the cases to which I have referred, be relatively insignificant while the nervous phenomena predominate. When this is so, the nature of the condition, as I have emphasized, may never occur to the clinician, even when he happens to be conversant with the clinical features which may be met with as a consequence of lead. Personally I have a guilty feeling that I have probably failed to recognize cases of the kind. Sometimes, perhaps, because one has not realized the occupational risk, sometimes because one has not elicited the history with sufficient care, sometimes because the symptoms have suggested to one a different etiology, and sometimes because, although one has suspected lead, one has not been able to obtain confirmatory evidence.

When, in any given case, one suspects lead poisoning from the character of the nervous symptoms, what are the points to which special attention should be paid for purposes of corroboration?

1. The patient's occupation may raise the possibility. Reference has been made to those industries in which there is a special risk.

2. Upon inquiry one may obtain a characteristic or suggestive history of attacks of intermittent, colicky, lower abdominal pain which is relieved by pressure.

3. The blue line on the gums, when present, is distinctive. It is, however, merely an indication of exposure, and not necessarily of poisoning, for many lead workers show the blue line and manifest no symptoms. But when symptoms are present which raise the possibility of plumbism the blue line is of the greatest diagnostic value. It is, however, to be remembered that this sign fails us when the patient is edentulous or the teeth well cared for.

4. The examination of the blood in most cases of lead poisoning shows a secondary anaemia with, in almost all cases, a punctate basophilia, which may, however, vary from day to day. To the latter appearance much importance is attached as an indication of early lead poisoning. Although not pathognomonic (for an occasional cell showing punctate basophilia may, it is affirmed, be sometimes seen in normal blood, and cells of this type are also met with in pernicious anaemia and leukaemia), the presence of definite stippling is a most suggestive sign in a case of suspected plumbism.

5. Lead may be found in the urine, and its presence is then a clear indication that it has been absorbed into the system. On the other hand, its absence does not exclude lead poisoning, for the lead may be retained in the tissues. Of 200 patients with suspected lead poisoning examined at the Mayo Clinic¹⁵ 100 were shown to be excreting lead in the urine. Lead may indeed be found in the urine of individuals who are admitted to hospital for various conditions, who have not encountered obvious occupational hazards, and who present no symptoms suggestive of lead poisoning. Consequently, while further observations are called for, the presence or absence of lead in the urine is a point to which too much importance must not be attached.

From what I have said you will see that, while in many cases the diagnosis of lead poisoning is obvious, in others the possibility may never suggest itself, while

even in cases in which one suspects lead as the cause of the clinical picture a definite conclusion may only be arrived at after a careful consideration of all the facts if the source of poisoning is not detected.

Coincidences are sometimes remarkable. Two days ago, within an hour of writing the above lines, I saw at the hospital a man, aged 50, who had been under my care in June of last year with an anterior crural neuritis of undetermined etiology. The patient has now practically recovered, but the symptoms, as the notes showed, were anomalous. I recalled a remark of that great clinical master Sir William Gowers,¹⁴ who in writing of the diagnosis of lead poisoning says, "It is not possible to avoid error in such cases except by the habit of remembering lead as a possible cause in obscure cases." Was it possible that this man's symptoms had been caused by lead? Upon inquiry as to his occupation he informed me that he was a house painter, and, further, I found that while he was in hospital the house-physician had noted the presence of "a slight blue line round the gums." The teeth had since been extracted, and the line had consequently disappeared. Further, the patient stated that, although he had worked as a house painter for thirty-five years, he had never been troubled with abdominal pains, weakness in the hands, or, so far as he knew, any symptom of lead poisoning. The probabilities would, however, appear to be that this was the cause of his symptoms in view of their anomalous character and the absence of any other apparent etiological factor.

There is a natural tendency to associate symptom-complexes with individual diseases. Now while this is of undoubted value in furthering the general recognition of the disorders in question, it has its disadvantages. There can, for instance, be no question that the symptomatic triad—intention tremor, nystagmus, and scanning speech—emphasized by Charcot in disseminated sclerosis, was too restricted and retarded diagnosis in so far as it failed to include many cases which we now know to be due to the same pathological process. Similarly the three symptoms of lethargic encephalitis to which so much importance was originally attached—lethargy, febrile disturbance, and ocular palsies—concentrated attention upon one particular type of the disease. The same applies to the diagnosis of plumbism, for when we think of lead poisoning most of us, no doubt, at once recall the characteristic wrist-drop, whereas we are apt to forget that a variety of nervous symptoms may be so produced. There is an aphorism so familiar that it savours of a platitude—"More mistakes are made by not looking than by not knowing." Mistakes in the diagnosis of lead poisoning are often due to not expecting. My purpose will have been achieved if I have succeeded in impressing you with the importance of thinking of lead poisoning as a possibility in every case with which you meet which presents anomalous nervous symptoms.

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CUMULATIVE EFFECTS OF INFINITESIMAL
DOSES OF LEAD*

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There are two types of plumbism: one, the classical lead poisoning of the textbooks, caused by massive doses; the other, little known, a slow, subtle, insidious saturation of the system by infinitesimal doses of lead extending over a long period of time, and producing a group of symptoms altogether different from the recognized forms of plumbism.

In the type of plumbism to which I refer there is rarely colic, seldom a blue line on the gums, and wrist-drop will occur only if the significance of the early symptoms is not recognized. There is rarely noticeable anaemia. I cannot say if an examination of the blood would reveal any approach to the blood destruction characteristic of the grosser forms of lead poisoning.

SIGNS AND SYMPTOMS

A strange lethargy creeps over the sufferer; he feels as if a cloud had settled over him; he loses interest in life; everything is a trouble—a weariness of flesh and brain. He prefers to sit over the fire to tackling his work, though when he forces himself through his tasks he finds no diminution of brain or bodily power. As the condition continues he becomes gloomy and taciturn. Instead of joining in conversation with relatives and friends he sits silent and apathetic, as if overcome by thoughts too melancholy to utter. All his faculties and bodily powers are sluggish. His bowels are constipated and stubborn, he derives no satisfaction from his food, and has perhaps abdominal discomfort, which he puts down to indigestion. The strange lethargy of body and mind increases. Sleep brings welcome respite, but he gets up tired and weary, as if he had not been to bed, though he has slept heavily all night; so tired, indeed, that to bathe, wash, and dress are ordeals he wishes he could shirk. Breakfast bucks him up, but during the day, oppressed with a weariness of body and misery of mind, and unaccountably tired, he succumbs to his desire to sleep. In neurotic females emotional outbursts, with flatulency and abdominal pain, may punctuate the melancholy.

Apparently lead puts a brake on metabolic processes. The listlessness may be due to deficient renal excretion or to a check to thyroid secretion. The hebetude is not unlike that of myxoedema, and I am sanguine enough to think that lead will lessen thyroid activity. The old doctors were not afraid to administer lead to their patients. The still officinal pil. plumbi cum opio was their favourite prescription. It has passed into the limbo of forgotten remedies, but it is possible that it might prove an efficient weapon in the hyperthyroidism of Graves's disease.

The patient gets no sympathy from those about him. If his expression is saturnine, his complexion is fresh and healthy, he sleeps well, forces himself to do justice to his meals, and his relatives set him down as a gloomy person who will not take the trouble to be chatty and lively. He is usually the only person in the house to behave so strangely, for lead has a selective action, and the freedom of others from symptoms increases contempt for the sufferer. Perhaps he sees a doctor, who suggests change of air. He goes away, and in a few weeks returns home a different man, full of life and energy, with all the

* Read before the North Wales Branch of the British Medical Association.