A SUPPLEMENTAL INQUIRY INTO THE FREQUENCY WITH WHICH LEAD IS FOUND IN THE URINE.¹

BY JAMES J. PUTNAM, M.D.,
Instructor in Diseases of the Nervous System, Harvard University.

At a meeting of the Association of American Physicians, in June, 1887, I reported an analysis of eighty-six cases, not presenting unequivocal symptoms of lead-poisoning, where the urine had been examined for lead. In forty-eight of these cases lead was found; in thirty-eight it was absent.

The question which I had in mind in making the investigation was whether long-continued exposure to very small quantities of lead gives rise to other symptoms than those that are commonly reported; and, if so, whether such symptoms are sufficiently characteristic to lead to, at least, a presumptive diagnosis.

The conclusions which that investigation seemed provisionally to justify were the following:

(1) It is not probable that lead is to be detected in the urine of more than fifty per cent., at most, of persons not presenting symptoms of disease, and the finding of lead in a larger percentage, for any special group of cases, would be presumptive evidence that lead was connected, directly or indirectly, with the production of the symptoms.

(2) That lead is an occasional cause of chronic sensory neuritis, and chronic or subacute myelitis, and may give rise to the symptoms of spastic paraplegia.

(3) That lead is an occasional cause of pure neurasthenic symptoms, and the presence of a fine muscular tremor strengthens the probability of this diagnosis.

(4) That workers in rubber are liable to suffer from lead poisoning.

The investigation which is the subject of the present paper was undertaken, primarily, to control the first of these conclusions, as to the frequency with which lead is to be found in the urine of healthy persons.

For this purpose I first obtained thirty specimens of urine from medical students, and had them analyzed by Dr. A. M. Comey, who had so carefully performed the earlier analyses. These gentlemen were living, as a rule, in boarding-houses in the city, and came originally from various parts of the country. Potassium iodide was taken in each case for four days before the urine was collected. Slight traces of lead were found in three cases, or ten per cent. In one of the three cases a second analysis failed to detect lead; in the other cases no second analysis was made. The students in whose urine the lead was found appeared to be in perfect health.

In order to make the same inquiry with regard to a class in the community living under poorer circumstances, I obtained, through the kindness of Prof. F. C. Shattuck, specimens of urine from a number of patients at the Good Samaritan Hospital. Thirteen of these patients were suffering from phthisis or pleurisy; in three cases lead was found in the urine. In four cases, presenting simple anæmia and debility, lead was absent; but as lead might have been the cause of these symptoms the cases are excluded. In a case of acute Basedow's disease, brought on by severe fright, lead was also absent. The same was true in two cases of paralysis agitans, and one of acute chorea.

Besides these cases, I have had analyses made² of

the urine from a number of patients presenting themselves at the out-patient department of the Massachusetts General Hospital, suffering from some disease, either strictly local, or, so far as we know, not attributed to lead, and from a few suitable private patients.

In all, we have fifty-seven new analyses from persons who might be said to be from our point-of-view without symptoms. In ten of these, or 17.5 per cent., lead was found, usually in slight traces.

It is doubtless true that if more refined methods of chemical analysis could have been employed, and larger quantities of urine, besides other secretions and excretions have been subjected to analysis, the number of cases belonging to this group in which lead was present would have been greater. If to these we add the eleven cases reported upon two years ago, we have a final total of sixty-eight cases, in 17.6 per cent. of which lead was found to be present, and so far as we now know, therefore, this proportion may be taken as about representing, or probably rather under-representing, the frequency with which minute quantities of lead are present in the tissues of practically healthy persons.

If, to compare with this group, we add together all the cases from the two investigations of which it cannot be said positively that they presented no symptoms which would commonly be said to be attributed to lead, we have a total of 117, in 62, or 53 per cent., of which lead was found. Even if we add in the cases presenting no characteristic symptoms, raising the total to 186 cases, we still find that lead was present in 40 per cent., or more than twice the proportion found in the first group. This comparison makes it probable that lead is at least a partial cause of a variety of symptoms not usually attributed to that cause.

Case.	SYMPTOMS.	Former series.	New series.	Total.	Lead present.	Lead absent.	Per cent. in which lead was present.
1	No symptoms not attributable to other causes	11	57	68	12	56	17.6
2	Fine muscular tremor; nervous debility	8	2	11	11	0	
3	Chronic multiple neuritis; neuritis with chronic and subacute myelitis; spastic paraplegia; multiple sclerosis	19	24	43	27	16	
4	Progressive muscular atrophy	4	1	5	0	5	
5	Cerebral and cerebro-spinal cases	7	2	9	7	2	
6	Epilepsy	8	4	12	4	8	
7	Sciatica	2	1	3	0	3	
8	Acute polio-neuritis	0	2	2	0	2	
9	Digestive disorders	0	2	2	0	2	
10	Simple anæmia and debility	0	4	4	0	4	
11	Remaining cases from former series	26	..	26	13	13	
12	Rubber-workers	0	8	8	5	3	
13	Optic neuritis	0	2	2	2	0	
							53 40

No statistical importance is to be attached to these ratios, except as stated in the text.

No doubt the composition of this table is open to a number of criticisms; one of which might be that quite a large proportion of the patients presenting symptoms of organic disease were laboring men, whose lives would expose them to a relatively large number of the many sources of lead-contamination, and, at the same time, to other, and perhaps the real causes of their disease. Nevertheless, the numbers are sufficiently striking to make it worth while to examine the groups of

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² The later analyses were made by Dr. C. P. Worcester, under careful and repeated control experiments.

cases in which lead was found for indications of its influence.

I recognize that this attempt should be made in a conservative spirit, for it is evident that the presence of small quantities of lead is not only compatible with health, but does not necessarily complicate other existing diseases. Thus, one of the cases noted in the earlier paper was that of a gentleman suffering from sarcomatous disease within the pelvis. Lead was twice found in the urine in the course of his illness, yet, in spite of the presence of this poison in his tissues, and the depressing influence of the organic disease, which eventually proved fatal, he exhibited no marked signs of anæmia or impaired nutrition until a comparatively short time before his death.

One group of cases, the patients being a father, mother and three children, is especially interesting in this connection, as indicating the importance of taking lead-poisoning into consideration in obscure cases, and as indicating the difference in susceptibility between different members of the same family. The water used by this family at their country house, where they spent a considerable part of the year, ran for quite a distance through lead pipes, and was found to contain a large quantity of lead. Repeated analyses showed that lead was present in considerable quantities, in the urine of two of the children, the father and the mother, though for some reason, not easily to be explained, not in that of the third child. All these children were in fair health. One of them, however, has been very subject to sharp attacks of gastric indigestion, pain in the left side, and vomiting preceded by sore throat; and also to constipation, which was worse during the attacks of indigestion. When the lead was found, small doses of iodide of potash were given, but seemed to precipitate the attacks above mentioned, and was, after a number of trials, abandoned by advice of her physician, who believed her attacks to be due to imperfect power of digestion. A possible cause for this imperfect digestion was, obviously, the lead. She became, finally, perfectly well, but still has to be careful with her food. Another child, a sister, in whose urine, also, lead was found, is said to have had a few attacks of indigestion and occasional clay-colored stools. A third child, a boy, who was thought to have drunk even more of the water than his sisters, had no lead in his urine, according to two analyses, and no symptoms. The father and mother were perfectly well, though their urine contained a considerable amount of lead. It is a remarkable fact that in the extensive outbreaks of lead-poisoning from drinking-water, occurring in certain English towns, children have seemed to suffer much less than adults, but the lighter grades of symptoms were probably not taken into account.

There is obviously a danger that in seeking to account for pathological conditions of obscure origin that arise in the presence of an agent like lead, we may, by including symptoms that are due to some other cause, obscure instead of illuminate the true clinical picture.

For this reason, I think that it is best to suspend judgment for the present as to the etiological diagnosis of the cases classified in the table, and wait for the further accumulation of evidence.

The largest group of cases analyzed in the earlier paper was that comprising nineteen cases of chronic generalized neuritis, neuritis with chronic myelitis,

myelitis with symptoms of spastic paraplegia, and multiple sclerosis. In fifteen of these cases lead was found to be present, and in four it was absent. I can now add to this group twenty-four similar cases, equally divided between those in which lead was present and those in which it was absent. To these ratios we cannot attach much importance, because the cases do not form a strictly homogeneous group, and those which are essentially alike, are not in sufficient number to warrant statistical conclusions of real value. The greater part of the cases of this group may be subdivided as follows, into:

(1) Those in which subacute myelitis was present, usually associated with profound disturbance of the general nutrition, and generalized neuritis.

(2) Those characterized by the symptoms of spastic paraplegia, without signs of any marked impairment of general nutrition.

(3) Cases, apparently of chronic neuritis.

(4) A case of subacute neuritis, with neuralgic symptoms.

It will be noted that the changes in the spinal cord characteristic of the first two groups are of the nature of chronic scleroses, especially of the long cerebro-spinal tract, and inasmuch as such changes are not usually attributed to lead, it may be well to inquire what part, in the judgment of the best authorities, lead-poisoning does play in exciting diseases of the cord.

I do not propose to reopen the whole discussion of this subject, but will ask you to admit as representing, at the least, a sufficiently cautious and conservative position the views expressed by Professor Schultze in a critical paper accompanying the report of an anatomical examination, published in the *Arch. für Psychiatrie*, for 1885. This writer points out, after carefully reviewing the evidence, that there is as yet no sufficient grounds for the theory that the well-known and constant changes in the nerves and muscles seen in the typical paralysis of the forearm extensors, are secondary to spinal disease (which some observers have believed to be always present even though latent); and that, in the cases where myelitis is found, of which there are but very few on record that are above criticism,^{*} the spinal changes do not stand in any coördinate relation to the peripheral changes, and are not coextensive with them, but form an independent lesion, and are, as he thinks, indicative of a later stage and a more generalized form of poisoning than that implied by the neuritis alone.

A similar view has been upheld by Zunker, and other writers. It is only open to one criticism, as it seems to me, and that is, I think, an important one; namely, that the evidence before us is as yet, too meagre to admit of our asserting, with any degree of confidence, that myelitis occurs only in cases of "severe and generalized poisoning." If we admit that it is not the cause of the typical paralysis, and that when it occurs, it is as an independent lesion, we might fairly expect that, like the other independent lesions of the same origin, it should come to the front in some cases and remain in the background in others, in the most unexpected manner, until the contrary has been proved.

This probability gains in weight through the inter-

^{*} Neither Schultze nor Oppenheim refers to Dr. Birdsall's case, published in 1881 (*Journ. of Neurology and Psychology*, N. Y.), nor to those of Moritz (Dreschfeld and Young), London, *Journ. of Anat. and Physiol.*, October, 1880; or of Seguin (unpublished) which are cited by Birdsall.

esting clinical distinction made by Gowers, between the muscular atrophy due to degeneration of the peripheral nerves (the usual type), and that analogous to progressive muscular atrophy, which affects by preference a somewhat different set of muscles from the usual form and runs a different course, and may be of central origin. As to the form which the spinal affection probably takes when it does occur, so far as we may judge from the very few cases on record, it is oftenest of the polio-myelitic form, except that v. Monakow found patches of sclerosis, and Vulpian, in a dog, granular cells and other signs of subacute diffused myelitis. Monakow's case is considered by Schultze as not a clear one, since some of the symptoms and some of the lesions, including the spinal sclerosis suggested the presence of paralytic dementia, but it would be begging the question to assert, that, even then, lead might not play a part in the etiology.

It has, further, been asserted by Monakow and by Oppenheim, that the cell groups of the anterior horns which appeared to have suffered the most, were those of the anterior and median portions of the horn, and I was especially interested in this statement because the same condition appears to be present in some specimens of my own. Schultze, however, thinks that this relative scarcity of nerve cells in these parts of the anterior horns is sufficiently common in healthy cords, and, although he found it in his own case of lead paralysis, he does not attach much importance to the appearance, unless associated with other signs of destruction of tissue.

Finally, another important change quite generally noted in the reports of the examinations of the cord, is thickening of the smaller arteries.

As a rule, this was associated with nephritis, and it is a condition often seen apparently as a secondary feature, in chronic spinal cases. Still, it is pretty certain that thickening of the vascular walls may occur in various organs, as a direct result of lead-poisoning, and it may have an etiological significance apart from the nephritis, as will be pointed out later.

With regard to the possible influence of lead in the etiology of the chronic system-scleroses, I do not in the least suppose that it is the one special cause, or, mean to assert that it is certainly a direct cause at all, though this is no more intrinsically improbable than that syphilis, ergot, and lathyrus should excite spinal scleroses, as these appear to do, — but it does seem to me possible that lead may at least act as an indirect cause of spinal sclerosis by causing increasing general anæmia, and by exciting hypertrophy of the small vessels.

If we glance over the conditions under which the scleroses in general arise, we find, I think, that among the most prominent are an inherited tendency to degenerative weakness, low or depressed vitality, and toxic influences. Thus, syphilis certainly plays an important part in causing locomotor ataxia, and even in causing lateral sclerosis and that the scleroses associated with parietic dementia; ergot and lathyrus seem to excite lateral sclerosis; and malaria, and other zymotic poisons have been suspected of a similar influence.

The influence of heredity is most marked in Friedreich's disease, of which a form of combined sclerosis is the most conspicuous lesion, but there are many partial or irregular cases on record, which it is difficult to classify, where the local disease appears to be closely

associated with a generally poor state of nervous vitality.

A recent suggestion of Lauder Brunton is interesting in this connection. Referring to the importance of the study of the action of drugs on the production of lateral sclerosis, and especially to the observations of Tuzec on the sclerosis of ergot-eaters, he points out that this result is not produced experimentally by injections of ergotine, and thinks that the poverty and famine of the peasants who fall victims to the disease may have a good deal to do with the result. Of course, many well-nourished persons suffer from typical lateral sclerosis, but then some other distinct cause, especially syphilis, may come in.

To speak of another possible partial cause in this case, and others of the same kind, I will say that our patient's mother suffered for some years before her death from symptoms which are thus described, from memory, by her daughter:

"For a number of years she was troubled with diarrhœa at times: then she complained of numbness in her fingers at first (presumably, afterward in the feet), feeling like coarse sand under the skin, with occasional return of the looseness of the bowels. Then there was swelling of the feet and ankles, which finally disappeared. She had the numbness and diarrhœa for a year before the dropsical symptoms, shortly after which she was unable to sit up for several months. She afterward rallied and was about the house for nearly a year, but remained very feeble, complaining of great soreness across her bowels, and having very little control over her limbs. She had an attack of jaundice in July, and died in October. I do not think her head was affected to any great extent, or her back. She complained of her knees being cold, and wrapped them up even in very hot weather."

Apart from the possibility of relationship of developmental origin, between the disease of mother and son, I was interested in this account from the fact that three other of the patients who belong in the group of which I have been speaking, suffered from excessive diarrhœa before and along with the spinal symptoms. I have never felt sure whether there was any connection at all between them, nor whether to consider the diarrhœa as a partial cause of the anæmia, and thus indirectly of the spinal disease, or as itself a result of some anatomical changes in the sympathetic system.

I will not take the time of the Society by describing the other cases in which lead was found in the urine. I will only say here that the list comprises other cases more or less analogous to the one just described; also two cases of optic neuritis apparently from lead-poisoning; for one of which I am indebted to Dr. J. J. Minot and Dr. O. F. Wadsworth; while the other is of interest as exhibiting symptoms strongly suggestive of parietic dementia and yet not justifying that diagnosis; further, several cases of chronic sensory neuritis, and one highly interesting and rare case of subacute neuritis with distinctly neuralgiform symptoms, nerve tenderness, and impaired electrical reactions, affecting one nerve after another until in the course of a year all four limbs became involved, the legs only to a slight degree. Lead was found twice or three times in the urine, once in considerable amount. For this case I am again indebted to the kindness of Prof. F. C. Shattuck.

With regard to the diagnostic value of the finding of traces of lead in the urine, it is needless to say again that I regard it as of no necessary importance. Dr. Gowers thinks its significance is much less than that of the "lead-line." Perhaps this is true, but a person may have a marked lead-line and yet no impairment

of health. This was strikingly shown not long ago at the Massachusetts General Hospital, where a typical and very bad case of universal drinking-water paralysis presented itself. The patient's aunt, who accompanied him, denied all symptoms of the disease, but her gums showed a well-marked lead-line.

I think the result is that it is never safe to say how large a quantity of lead a person may bear, nor how small a quantity may effect him badly, and that we probably do not yet know all the manifestations of the poison.

In conclusion, I wish to say a single word about the source of lead-poisoning in drinking-water, an old but ever new subject for investigation and discussion.

Dr. E. M. Greene, of Boston, has recently been making some observations on this subject which show the danger to which dwellers in our large cities are still exposed.

My own house being unused this summer, and therefore a good subject for study, I asked Dr. Greene to make the following analyses, the results of which are appended.

(1) First drawing of water (Ov in each case) from kitchen faucet; the water having lain in a pipe about seventy feet long: Large amount of lead found.

(2) Water drawn after faucet had discharged for five minutes: Lead still present.

(3) Water drawn after faucet had discharged for fifteen minutes: Traces of lead.

(3a) Ibid; and then allowed to remain closed for half an hour: Larger traces than in last case.

(4) Water drawn after faucet had discharged for an hour: No lead found.

(5) Ibid; and then allowed to remain closed for three and a half hours: Traces of lead.

The water from the faucet at the top of the house (five stories) was found to contain lead after it had been running for half an hour.

OBSERVATIONS ON THE OCCURRENCE OF LEAD IN BOSTON DRINKING-WATER.¹

BY E. M. GREENE, M.D.,

Pathologist to Boston Dispensary and Carney Hospital.

THE attention of the writer was directed to this subject last April, when he became acquainted with a family of which several members were evidently suffering from lead-poisoning, the source of which was not readily apparent. Finally the drinking-water was examined and found to contain a large amount of lead.

In order to determine the frequency with which lead is found in Boston drinking-water, a number of examinations were made of water taken from different parts of the city and coming from Lake Cochituate. In each case the houses were supplied with lead pipes, and the specimens for examination were taken after the water had been allowed to run from two to five minutes. The reagents used for the lead analysis were first tested in an examination of distilled water for lead, and no trace of lead was found. The first two specimens came from the house occupied by the family above-mentioned, who lived on the third floor.

(1) Bennet Street: water, after running two min-

utes, contained a large amount of lead, very nearly one grain to the gallon.

(2) Same house, from an adjoining room: water, after running five minutes, contained a considerable amount of lead, about one-half as much as the preceding. A similar specimen, sent to Prof. E. S. Wood, was found by him, also, to contain a considerable amount of lead.

(3) Marlborough Street: after running five minutes contained considerable lead.

(4) Hancock Street, first floor: after running three minutes contained much lead.

(5) Same house, next room: from a leaky faucet which was constantly running a small stream, no lead.

(6) Another house on Hancock Street: after running four or five minutes, considerable lead.

(7) Ash Street, first floor: after running several minutes, considerable lead.

Following Dr. J. J. Putnam's suggestions, a number of examinations were then made of water taken from different parts of the same house, and after running for various intervals. The following nine specimens came from 106 Marlborough Street:

(8) Drawn after standing two days and practically for several weeks, much lead.

(9) Same tap, after running full stream for five minutes, considerable lead.

(10) Upper story, first drawing, much lead.

(11) Same, after being allowed to discharge for fifteen minutes, small amount of lead, perhaps a thirtieth of a grain.

(12) Upper story, after running thirty minutes, a slight trace of lead.

(13) Kitchen faucet, after running fifteen minutes, small amount of lead.

(14) Same, after running fifteen minutes, then closed thirty minutes, considerable lead.

(15) Kitchen faucet, after running one hour, no lead.

(16) Same, after standing three and one-half hours, considerable lead.

It would be interesting to know whether injurious results have followed from drinking the water above analyzed. In three of the above places, where I have had opportunities to investigate, such has been the case.

On Bennett Street, the family consisted of a man, his wife and son, all of whom spent the greater part of every day at home. The wife enjoyed fair health. The father and son, especially the latter, a young man, suffered from general debility and marked anemia. He had been unable to work for several months, and suffered from severe, frequent, abdominal colic, gastro-intestinal disturbances and general muscular weakness. The physician called to attend him made a diagnosis of lead-poisoning before the drinking-water had been examined. After giving iodide of potash, the urines of both father and son were examined and found to contain a considerable amount of lead. The father's urine showed a slight trace of albumen, and contained uric acid crystals and hyaline casts. Under iodide treatment and spring water, after several weeks the colic and gastric disturbance disappeared entirely, and the general health in both cases was much improved.

In the first house on Hancock Street, the family consisted of a middle-aged lady and two adult daughters. The mother and younger daughter were very

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