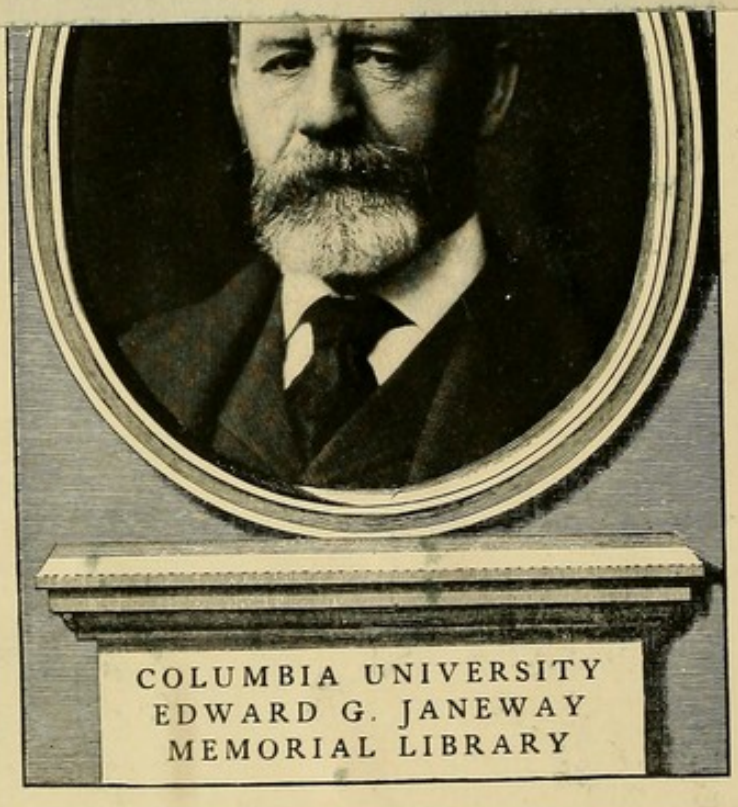
Lead poisoning : in its acute and chronic forms : the Goulstonian Lectures, delivered in the Royal College of Physicians, March 1891 / by Thomas Oliver.



LEAD POISONING.

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IN ITS

ACUTE AND CHRONIC FORMS.

THE GOULSTONIAN LECTURES, DELIVERED IN THE ROYAL COLLEGE OF PHYSICIANS, MARCH 1891.

BY

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PREFACE.

Lead Poisoning is so widely met with, that I need scarcely apologise for the appearance of this monograph.

Almost in the form in which these Lectures were delivered in the Royal College of Physicians, they are now presented to the medical profession.

I take this opportunity of acknowledging my indebtedness to my Colleagues—the Physicians of the Royal Infirmary, Newcastle-upon-Tyne, for their generosity in allowing me to utilise their cases—also to the Surgeons to the Lead Works, for the cases they sent me, and to Mr Foster, Manager of the Ouseburn Lead Works, for the many opportunities he afforded me of being present at the medical examination of lead workers. To Dr Sims Woodhead I am also indebted for the revision of my microscopical preparations, and to Dr Baigent for many of the drawings which are reproduced in the book.

THOMAS OLIVER.

NEWCASTLE-UPON-TYNE,
July 1891.

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LECTURE I.

LET me thank you, Mr President, for the very great honour which you have conferred upon me in inviting me to become the Goulstonian Lecturer for the year. Having accepted the office, I am deeply sensible of the responsibility it involves. Weighty as I feel the duty imposed upon me, it is only equalled by my desire to make these Lectures acceptable and instructive.

In searching for a subject, I considered that there was one upon which, with the time at my disposal for its preparation and delivery, I might be able, from the opportunities afforded me for its study, to say something that might prove of interest to you and of service to the Profession.

Lead poisoning is not only of local but of national importance. The last two years have shown how truly necessary it is for us to realise the dangers that often lurk in the supply of drinking water and of various forms of food. The wholesale poisoning that occurred a short while ago in Sheffield, and which necessitated a scientific inquiry, the poisoning of the inmates of Keighley Workhouse, and the extent to which in Yorkshire at any rate it still follows the use of water, and the repeated announcement in the newspapers of coroners' inquests held in London and the North of England upon cases of lead poisoning, all bear testimony to the ruin wrought by lead, and are my excuse for making it the theme of these Lectures. Apart from the epidemics of lead

poisoning which have passed over certain towns, owing to the faulty conduction of the water supply, it is true that the disease usually affects only a small proportion of the labouring classes, but that is one reason why we who are the guardians of the public health should, by increasing our own knowledge, help those who cannot help themselves. Besides, this College is the Academic centre from which should issue words of counsel and words of warning upon the dangers of a noxious industry, and the subtle influences of a poison which at any time may be widely diffused. It was in the College of Physicians that, in 1767, Sir George Baker, a scientist of celebrity, first drew the attention of the medical profession at once to the production of colica pictonum by the use of cider, and successfully taught how to remedy the evil.

There is a general consensus of opinion that the insidious introduction into the human system of lead in very minute quantities is followed by symptoms of a more serious and persistent character than when taken in larger quantities and for a shorter period. In the latter case we have the symptoms of irritant poisoning, but in the former the symptoms are sometimes so remote that their relationship may escape our attention. So insidiously are the symptoms developed, and so variable, that not only do we as physicians occasionally fail to refer the illness to its proper category, but medical men, the authors of pamphlets on lead poisoning, and who, therefore, have had practical experience of the poison in others, have themselves suffered from the malady without being aware of the nature of their illness.[1] In the cumulative action of the poison lies its danger. Whilst briefly recognising food, water, and fermented liquors as media whereby the poison may be introduced into the system, my experience has been gained mostly from workers in lead factories. If, therefore, I deal with lead poisoning from the industrial rather than from the accidental side, it is simply because I have

[1] Case of Dr Norris, quoted by Harrison, "Contamination of Water by Lead," page 119.

in this way seen more of the results arising from exposure of the system to the influence of lead. Lead under any form, if taken for a length of time, will cause poisoning even in the form of its most insoluble compounds, such as the sulphate. The acetate which is very soluble, and the carbonate and oxide which, though insoluble, are easily acted upon by the gastric juice, are perhaps the most dangerous, unless it be the chromate and arseniate of lead, whose toxic properties would be largely influenced by the chromic and arsenic acid they contain. It is amongst lead workers, however, that we see the metal produce its most disastrous effects, rapidly converting men and women, frequently pictures of health, into human wrecks.

There are other trades besides lead making which are notorious for a high death rate, and in not a few of them lead is manipulated in some way or other. It is upon those engaged in the manufacture of lead that the poison produces greatest havoc, and yet amongst those engaged in other industries, symptoms of a slower but not less severe form of poisoning are also met with. Lead poisoning amongst printers is a thing that has practically gone out of existence. Under any circumstances amongst printers, it would be difficult to attribute death directly to lead poisoning. It can only be done by appreciating the number of deaths due to diseases of organs that are known to suffer in lead poisoning, for example the kidneys and the nervous system. At the present time scarcely a case of lead poisoning occurs amongst printers. The high death rate amongst plumbers, painters, and glaziers, who show a comparative mortality figure of 1202 against 1000, is mainly determined by their liability to lead poisoning.[1] Associated with this mortality directly attributed to lead there is an excessively high mortality from diseases of the urinary organs, diseases of the nervous system and circulatory organs. The tendency to disease of the kidneys in painters has long been known, and by English physicians has been properly regarded as the result of lead. Amongst the file makers of Sheffield lead

[1] Registrar General's Reports, p. 25.

poisoning is frequently met with. Dr Porter[1] in his thesis draws attention to the unhealthy nature of their occupation. Exposed during their work as these men are to all the dangers arising from metallic dust, and in this way with a high death rate from phthisis—a circumstance which is opposed to the opinion expressed by Tancquerel des Planches as to the protective influence of lead in this disease—they are prone to suffer from lead poisoning. This arises from the fact that these men are obliged to use a cushion of lead on which they strike the file, and thus they are daily inhaling metallic dust. Their high death rate comes from diseases of the urinary organs: they suffer also from diseases of the nervous system and of the circulation. The next trades on the list with a high mortality from this cause are plumbers and painters.

The employment of the word plumbism for lead poisoning needs no explanation, and beyond the fact that Saturn was the alchemic name for lead, I need only mention that Saturnine poisoning, plumbism, and lead poisoning are synonymous.

Lead poisoning has long been known. Galen, in his "De Medicina," condemns the use of leaden pipes, stating that the water which was transmitted through them contracted a muddiness from the lead and created a kind of dysentery in those who drank it. Nicander, a century before the birth of Christ, described acute poisoning by cerusse, and in his poetic fantasies can be detected an accurate description of details relating to lead poisoning. The Roman architect Vitruvius, who lived in the time of Cæsar Augustus, forbade the use of lead for conducting water, owing to the cerusse which is formed upon it being hurtful to the human body. Close to the Roman Wall which stretches from Newcastle to the south of Carlisle, built, as many of my hearers know, in the time of Hadrian, there are the remains of Roman camps. Some time ago, in passing over Cilurnum, one of the most perfectly preserved of these camps, I was much interested in finding a leaden pipe jutting out from the wall in

[1] Porter. Thesis for Degree of M.D., Durham University.

a perfect state of preservation, not at all crusted, exactly similar in appearance to that in use at the present day, except that it had been rolled and not moulded. Dr Bruce, an authority on the Roman Wall, informs me that in the station of Cilurnum, lead had been largely used for soldering purposes. I regard these two facts as interesting from the point of view of the antiquary. As a matter of fact, lead would be very little used on the Continent by the ancients. Their reservoirs for holding water were made either of marble or of cement, and their domestic utensils were made of pottery or of copper. In their decorative painting, their pure atmosphere encouraged the use of fresco: the oxides of lead, so essential in oil painting, would be readily dispensed with,[1] and thus, whereas at the present time the victims of lead poisoning in this country are numbered by the thousands, plumbism must have been, in days long gone by, only an occasional phenomenon. How it fared, in this respect, with the soldiers of the Roman Legions transferred from the plains of Southern Europe to the more uncertain climate of Britain, we have no means of knowing.

Lead is introduced into the system in many forms. The lead miner seldom suffers. Dr Montgomery of Blanchland, and Dr Robertson of Stanhope, both of whom have practised for many years in the dales of Durham, have never met with a case of lead poisoning in the miner. Dr J. Bartley, Resident Surgeon, Broken Hill Hospital, Australia, in the *Lancet*, May 23, 1891, p. 1178, says, "that at the lead mines at Broken Hill the miners are very susceptible to lead poisoning, often in a very severe form, more generally colic, but not unfrequently coma and epileptiform convulsions." The smelters also suffer, but more frequently "it is the miner working down below, especially when working in the carbonate ore." In this country we never have this experience, and this is probably due to the lead existing in a different form in the ore. The danger arises in the first in-

[1] It is to the absence of lead in early fresco that the rapid decay of the paintings is attributed.

stance during the smelting of the ore. A few years ago the smelting of lead was a perilous process, but since manufacturers have introduced a hood in front of the furnace, all the fumes are carried up the chimney, and the smelter, to a great extent, escapes. The men engaged in this work are generally strong, but most of them are pale, and show a well marked blue line on the gums. So injurious are the fumes which escape from the molten metal, that cattle, grazing in the neighbourhood, suffer severely from lead poisoning. This has occurred at Leadhills in Scotland, and in some of the dales in the county of Durham, where the animals are said to be "bellond"—an old term taken from the French "bellon."

Frequently lead poisoning thus induced amongst animals has proved of a wholesale character and been the cause of litigation. Cattle grazing in the fields in proximity to the smelting furnaces have died—the symptoms having been pain in the abdomen and constipation. Dogs too, that have lapped the water trickling from lead works, have also suffered. These animals exhibited very peculiar symptoms. The subjects of circus movements they kept wheeling round and round and were then seen to fall. Dogs which had simply slept on the coats or jackets of their masters who were lead smelters, also became "bellond." A few years ago a very peculiar epidemic occurred in France, where women, who had simply washed the clothes of their husbands who were workers in lead factories, suffered from Saturnine poisoning.

Some interesting facts bearing upon this point have recently come to light from Germany. Schroeder and Reuss,[1] in dealing with the influence of forge smoke upon the animals of the forest, state, that by the road sides and in the meadows and parks near to the forges are to be found many mountain-ash trees. Their berries are a favourite food with thrushes and some species of finches in autumn, and are used as food by nearly all the birds which remain there during the winter. Below and close to these trees were often found birds sickly or dead. Those that

[1] Die Beschäd. der Vegetation durch Rauch, 1883.

were alive had their extremities contracted, and their power of flight appeared paralysed. In a few days they also died—powerless in their attempt to flutter. Without doubt, they say, here is lead poisoning. The birds eat the berries, upon which are deposited the particles of oxide of lead, and thus poison themselves. The wild animals which roam around these forges do not appear to suffer from paralysis of their extremities, but a very peculiar circumstance has been noticed by these observers in regard to stags. Their antlers exhibit certain monstrosities; the branches are short and stunted: the pedioles are small, and sometimes are altogether absent—the rosettes in this case being flat and almost touching the cranium. The branches too are not shed, or only one is shed, and as a consequence, double crowns appear. These monstrosities are most apt to occur amongst stags whose sexual organs have been injured, for stags, like men, have their reproductive organs affected in lead poisoning. On the meadow hay, in the neighbourhood of the Altenan forges, Frëytag found a deposit of lead oxide equal to ·0027 per cent. Since the introduction of the hood in front of the furnace, to which I have already alluded, other arrangements have been made whereby the fumes from the furnace are prevented from escaping, and lead, to the extent of 33 per cent., is obtained from the deposit of these fumes. Gamgee, in his work on the Domestic Animals in Health and Disease, draws attention to the fact[1] that cattle have frequently died from drinking water contaminated by lead, and from eating grass impregnated with the oxide of lead, and he has shown that the deposition upon land of town manure, which contains the refuse of paint, sheet lead from tea chests, and pieces of painted oilcloth, has been followed by an outbreak of what is known as "staggers" in the animals that have been allowed to graze in the fields. Additional interest attaches to the fact, that certain fields are known by graziers to be dangerous to animals; several crops may even have been raised in rotation before the animals have been placed

[1] Page 179, vol. i.

upon them, and yet death has occurred. The process of working the land, and bringing up to the surface lead long buried, and thus allowing it to be influenced by the atmospheric air and rain, explains the danger. Two forms of poisoning, according to Gamgee, occur—the slow form attended by paralysis, and the other, acute, and due to solid lead having been eaten in large quantities, paralysing the stomach and inducing symptoms such as are met with where animals have filled themselves to repletion, causing obstruction of the third stomach.

Thackrah in his book on the "Effects of Trades on Health and Longevity," gives no place to lead making as a dangerous employment. He introduces the subject in his remarks upon plumbers and painters.[1] Plumbers suffer, he says, from inhalation of the volatilised oxide of lead arising during the process of casting; they frequently complain of a sweet taste in the mouth during the beating of lead and the application of solder. Their occupation undermines the constitution. Plumbers are short lived. In Leeds, at the time he wrote his monograph, only two individuals had regularly pursued their employment beyond the age of forty. His book was written in 1831, and speaking now sixty years afterwards, I think we can safely say, that plumbers appear to be a longer lived class than formerly. Painters follow an unhealthy occupation; lead in their case gains entrance into the system, partly through the skin and partly through the lungs. Of the occupations in which lead poisoning is apt to arise by absorption through the skin, that of potters, but only the dippers, suffers most. It is their duty to dip the pots into a solution containing lead. It is this which gives the glaze to the ware. Those who have been long engaged frequently suffer from symptoms of lead poisoning, for example vomiting, constipation, colic, double "wrist drop," and tremors. In industrial occupations, as in things in general, it would appear as if customs die hard. We advance slowly. An old author, Ramazzini, represents the potters of his day as

[1] Page 55.

affected with tremors, paralysis, cachexia, and he remarks, that almost every man thus employed has a cadaverous face. This remark equally applies to the potters of to-day. Amongst other trades in which symptoms of lead poisoning are met with, I would mention leather cutters. Dr Frederic Taylor, of Guy's Hospital, has recently met with cases of lead poisoning amongst men thus employed. These men cut the leather upon leaden slabs, and come to hospital complaining of colic and constipation, and nearly all of them exhibit a well marked blue line upon their gums.

Lindt[1] relates a fatal case of plumbism in a woman who had only been employed for half a year in a type foundry. In spite of warning, she ate her food with unwashed hands; there was no paralysis, but anæmia, jaundice, constipation, colic, blue line on gums, and headache, followed by convulsions and coma—death supervening on the fourth day. Women engaged in gumming bands on to cardboard boxes have also suffered from plumbism.

It is amongst lead workers that the worst types of plumbism are met with. So far as the working of lead is concerned, we have seen that the producer of the raw material seldom suffers; it is the smelter. The next danger arises during the process of the conversion of the metal into white lead or carbonate. There is no process of preparation of white lead so far known, that is equal to the old Dutch or dry method. It is a simple but dangerous procedure. It was introduced into this country about the year 1780, and is briefly this: Thin sheets of lead are placed upon the top of small earthenware pots, which contain strong acetic acid, the pots are arranged side by side in ordinary tan from the currier and covered with planks; another layer of pots similarly arranged in tan is placed upon these, and over these again another, and thus a "stack" is formed of alternations of planks and layers of pots. This is known as the "blue beds," and is of itself not dangerous. The acetic acid slowly volatilises by the heat induced through fermentation of the tan, and the lead is converted into a sub-acetate and subsequently

[1] *Sajous, Medical Annual*, 1889, vol. ii., A 50.

into a carbonate. When the conversion has become complete, girls enter the stack now known as "white beds," and remove the layers of carbonate of lead, carrying it in little trays on their head, first to the rolling mills, where the white is separated from the metallic lead, and subsequently it is taken to the stoves —a chamber not unlike a dairy, shelved all round for the trays to rest upon, and always at a high temperature, seldom less than 190 degrees. It usually takes about fourteen weeks for the lead in a stack to be completely converted into lead carbonate or white lead. Mr Forster, of Messrs James' lead works, Newcastle, tells me, that if girls are allowed to enter the stack too soon—that is, before the lead acetate is fully converted into lead carbonate—they suffer very readily and severely from colic. The readiness with which girls suffer in the white beds, is always an indication to him of the incompleteness of the conversion of the lead acetate. These two, the "white beds" and "stoves," constitute in my opinion the most dangerous stages of lead-making. Nothing injures a young woman more quickly than a few weeks of work in the white beds and stoves. After being thoroughly dried the lead is ground and washed, then dried as a fine powder. In this form it is packed in casks, a process attended by a certain amount of danger, owing to the dispersion of particles in the atmosphere. It was the opinion, when Sir James Alderson delivered the Lumleian lectures in this College in 1852, that women were less frequently affected by lead than men, but such is not my opinion. Women suffer far more frequently and more severely than men. Alderson says, "there is an impression amongst the workers, that the fineness of the pores of the female skin in some way protects them;" this is a very doubtful supposition, it rather explains their susceptibility, a susceptibility not only of the female lead worker but of females generally. It has been shown, that when the two sexes are exposed equally, as in the epidemics of plumbism from water pollution, the fair sex is weaker, and their nerve centres are more easily undermined by lead.

Of accidental lead poisoning, water may be regarded as the principal medium by which the metal is carried into the system. It was towards the end of last century, that in this country medical men began to study the connection of lead poisoning with the drinking of water. Dr Lambe, of Warwick, was amongst the first to examine this subject. He regarded spring water as particularly dangerous and unfit for man, owing to its solvent action upon lead—a power due to the saline ingredients which it contains. Guiton Morveau regarded distilled water as even more dangerous. All the relationship that need be dwelt upon now is the fact, that some waters act upon lead whilst others are destitute of that power. The use of some waters is followed by wide-spread symptoms of poisoning, as witness the outbreak in Sheffield little more than a year ago, where 129 cases are recorded as coming under the observation of six medical men within twelve months. It is scarcely within the scope of these lectures to enter into details respecting the chemistry of lead poisoning and drinking water. Those who are interested in this, will find it ably discussed in Christison's treatise on Poisons, and by Dr Brown of Bacup, in his observations on "Plumbism due to Lead-polluted Water." Water containing acids produced by vegetable decomposition, is prone to act upon lead, and especially that which penetrates peaty soils. In this way, I apprehend, are explained the epidemics of lead poisoning which have recently scourged various towns in Yorkshire. Dr Downes, in his report to the Keighley Board of Guardians, found sixty-eight per cent. of the inmates suffering from lead poisoning, traceable as in Sheffield to the fact, that Keighley had a moorland supply of drinking water which had become acid—the acid having been increased by the preceding drought. Bradford and Huddersfield have also experienced lead poisoning, and in Bacup they suffered severely. Dr Brown found lead to the extent of two-thirds of a grain in each gallon of water. The account published by Tronchin of an outbreak of lead colic in Amsterdam more than a century ago, lends con-

siderable weight to the view here stated. Until that period, lead colic was unknown in Amsterdam. Meanwhile the residents had been substituting lead for tiles on the roofs of their dwelling-houses, and they collected for culinary purposes the water from the house tops. The rain water was of itself sufficient to act upon the lead, but Tronchin showed that as the epidemic was most severe in its ravages in the autumn, the rain water had become impregnated with acid, caused by the decay of leaves from the trees which abounded in the city. Acids generated from decomposing matter, and acid emanations from manufactories, exert a powerful influence in increasing the solvency of water upon lead. Dr Herbert, of Whitby, thinks that a danger arises from sportsmen shooting over the moors, and leaving behind them lead all over the land. Small shot is composed of lead, arsenic, and antimony, and in some places he maintains the ground is simply paved with lead. The effects of this, he says, are terrible to contemplate, when we know that much of the water on the moors, which becomes impregnated, finds its way into the towns. This as a source of danger is I think over estimated.

A very interesting case of lead poisoning from drinking water, came under my notice a few months ago. Last year I was asked by Dr Smith of Ryton, to see in consultation with him Mr N., a gentleman aged fifty, who a few months previously whilst in the hay field became suddenly unconscious and fell. The attack was regarded as syncopal, but from that date he was never well. He complained of severe pain in the abdomen just above the umbilicus, and became extremely depressed in spirits. When I saw him a few months afterwards, he had all the symptoms of general paralysis. He still complained of abdominal pain and constipation; he had delusions and fibrillary tremor of the tongue and lips, his right pupil was dilated compared to the left; he was extremely cachectic, complained of a disagreeable taste in the mouth, and a blue line was noticed on his gums. His heart and lungs were healthy. He was sent to the sea-side for a few weeks, where all his symptoms diminished,

but on returning home most of them reappeared, the blue line deepening, and his delusions and depression came back again. There was no albumen in the urine. There was however a trace of lead in it. The drinking water was analysed, and analysis disclosed lead to the extent of only ·0028 grain per gallon. This patient died of an intercurrent disease, but retained many symptoms of lead poisoning. At the *post-mortem*, the principal thing noticed was a very marked accumulation of fluid in the sub-arachnoid space over the fronto-parietal convolutions, slight thickening of the arachnoid and of the pia mater, extreme pallor and hydræmia of brain, with fluid in the ventricles. His wife had also a blue line on her gums, but none of the children. The water from the well passed through several feet of lead piping into his house.

It may not be unimportant to observe here, that danger from drinking water is mainly when it has become impregnated with vegetable acids. Acetic, citric, tartaric, and malic acids, all act with various degrees of solvency upon lead. To the presence of these acids, is ascribed the lead poisoning which follows the use of wines and ciders. Many of these beverages have doubtless become accidentally contaminated with lead, as in the case of the Seltzer water now supplied in Paris. Others again have been intentionally adulterated. Wine growers have long known how wine that is acid and harsh can be materially improved in taste by the addition of litharge. This is an old established fraud, and as far back as the 15th century, was sternly prohibited in Germany. The endemic colic of Poitou, which broke out in 1572, and which lasted from sixty to seventy years, has been ascribed in modern times, and with justice, to the wines having been adulterated with lead. I may remind you, that it is from this association with a locality that lead colic was described by Citois as colica Poitou.

Milk that is sour may easily become impregnated with lead. Cider, ginger beer, and lemonade, have also been the vehicles for the introduction of lead into the system.

One of the most interesting cases of lead poisoning from the use of fermented liquors, that has recently come under my observation, is that which occurred in the practice of Dr Cromie of Blyth, in Northumberland. In September of last year, he was called to see a botanic beer manufacturer. The man, aged forty-four, had been quite well until two years previously, when he lost his appetite. He had never had colic nor sickness, but he had suffered from constipation and dyspepsia. In February 1890 he had "wrist drop," the paralysis appeared in the right fore-finger, which felt at first stiff and numb, and this gradually extended to the other fingers and then to the wrists. When seen by Dr Cromie at the end of August, he had well developed double wrist drop, was very anæmic, had blue line on gums and inner surface of the lower lip. The patient had a convulsion, but soon recovered consciousness. For some time afterwards his speech was slow. With the exception of headache, no warning preceded this fit, which was followed by vomiting and convulsions that reappeared every second or third day. In his last fit—the seventh—patient remained in a state of coma until death. The interesting point in this case is, that the botanic beer was carried through forty feet of lead pipe into the shop, and that every morning before breakfast, the man took a good draught of the beer straight from the pipe. He died undoubtedly from lead poisoning. The history of his household is soon told. His wife, twenty-eight years of age, with formerly a good deal of colour, is now anæmic. This woman had two children, one aged five and the other a little over three. Since the birth of the younger, she has had a miscarriage; her menses are regular, but rather profuse. At her husband's instigation, she has taken botanic beer every day for nearly two years. She was seized with colic twelve months ago, had vomiting, headache, and was constipated, but made a good recovery. Shortly after her husband's death, she was again seized with colic, the order of symptoms being first constipation, then vomiting and headache, accompanied by pain in the

bowels. When I saw her the colic had disappeared, but she was pale, had well marked blue line along the gums, her pulse was 80, not unduly hard; there was no cardiac bruit and no albuminuria. Living in the house with her two children, was a domestic servant. The maid, though frequently pressed to take the botanic beer, refused, as she did not like the taste of it. She has remained quite well, has no blue line, and is not anæmic. The elder child has been living most of the time with his grandmother, and has therefore not had many opportunities of taking the botanic beer. He is not distinctly anæmic, is not constipated, and has only the faintest trace of a blue line on his gums. His sister, a child of over three years, has lived always at home, and since early infancy has received daily a small quantity of this beer; she is very anæmic, and has a blue line on the upper gum. This child, when I saw her, could not talk nor even walk well. Her knee jerks were absent. There was a good deal of staggering, as in locomotor ataxia, with much restlessness and fidgetiness; there was no history of colic, constipation, or of vomiting. Three months afterwards, this child died of convulsions, which began on the 13th of October and lasted until the 24th. The convulsions were never severe, were more of the nature of tremors, and always attended by profuse perspiration; the urine contained a small quantity of albumen. The temperature throughout was normal, except that on the night before her death it reached 100·8. It is interesting to note, that this child had involuntary micturition and defecation, a circumstance of interest when we know how very frequently in animals poisoned by lead, the bladder is found empty, and is seen to contract in convulsive crises.[1]

The use of hair dyes strongly impregnated with lead has been followed, not only by symptoms of Saturnine poisoning, but by death. A ballet girl from the Tyne theatre was admitted into the Newcastle Infirmary under my care, suffering from colic, headache, and amaurosis; there were retinal hæmorrhages; she had been using cosmetic containing lead. She recovered.

[1] Réné Moreau, l'empoison. aigu par le plomb, page 70.

A far more striking illustration of the serious effects of the indiscriminate use of lead hair dye, occurred in the practice of my friend Mr G. B. Morgan, of Sunderland.[1] It was that of a maiden lady, aged seventy-two, who sought advice, because her left eyelid drooped, and that she could not raise it except with her fingers; that she saw objects double, and that for some weeks past both her arms ached continuously, and that the power in them was failing day by day. She also complained of loss of appetite and obstinate constipation. Her mind was perfectly clear, there was partial paralysis of both arms, ptosis and divergent strabismus of the left eye. A week afterwards, when seen by Mr Morgan, there was no improvement in the symptoms, and as the pains had not diminished, he ordered seven grains of potassium iodide to be added to her mixture. Next morning she was found dead in bed. At the *post-mortem*, Mr Morgan naturally expected to find some lesion at the base of the brain. The most careful examination of the brain by himself and Dr Ransom, failed to reveal anything abnormal in the brain generally, or in any part of it. The medulla, pons and ventricles, and circle of Willis, were all healthy. Thus was concluded a *post-mortem* which was to these gentlemen extremely unsatisfactory. At the autopsy, however, both were struck by the luxuriant black hair of such an old lady. One of this lady's nieces had been struck by the beauty of her aunt's hair during her lifetime, and she ventured the suggestion, could her illness have been caused by the use of a hair dye which the old lady made herself by mixing a packet of sugar of lead with sulphur, in some liquid unknown to the niece. By its use, as Mr Morgan suggested, may be explained the palsy of the upper limbs, then of the third nerve, and then rapid death following the administration of iodide of potassium on the theory of Melsens, that a soluble iodide had been formed which is highly injurious.

Dr Macdowell, of the Northumberland County Asylum,

[1] *Transactions of the Northumberland and Durham Medical Society*, April 1877.

informs me, that twice he has seen patients who have worn false teeth made of cheap plates, suffer from symptoms of lead poisoning. In both there was a well marked blue line on the gums; each patient became insane. In one of Dr Moxon's patients, mania, with hallucinations, arose in a woman who, for some inflammatory affection of the vagina, had on her own account made use of a strong lead lotion.[1]

Flour is occasionally a medium through which lead poisoning is induced. In our country we see little of this, but in France there are periodic outbreaks of it.[2] In one epidemic, upwards of 100 people were suddenly attacked with violent symptoms, amongst which colic predominated. Nearly all those who suffered presented the characteristic blue line. The water supply was suspected, but could not be incriminated. On inquiry, it was found that all the persons affected obtained their flour from the same mill, and that those who had partaken of rye bread suffered most. The tin buckets of the elevator which served to transport the rye flour from the grindstones had been tinned with lead, and these were found to have lost 150 grammes of their weight. Only the flour which passed through these buckets had caused symptoms of lead poisoning.

Dr Marshall of Pennsylvania[3] reports, that some time ago chromate of lead was used by the bakers of Philadelphia, and that in France, when eggs are scarce, it is employed to give a yellow tint to pastry. Dr David Stewart analysed 64 cases of lead poisoning traceable to the consumption of cakes dyed with chromate of lead. In most of these cachexia, colic, arthralgia, blue line on gums, were present, and traces of lead were found in the urine.

In Russia, the use of tinned or canned meats has been followed by symptoms of lead poisoning, but on the authority of Professor Attfield and Dr Thomas Stevenson, no acute cases of

[1] *Journal of Mental Science*, 1881, page 130.

[2] *British Medical Journal*, 1887. *Sajous, Med. Annual*, 1888–9.

[3] *Sajous, Medical Annual*, vol. v., 1888, page 251.

lead poisoning have occurred in this country from that source.

It is very rarely that lead has been given for a criminal purpose. Generally it is taken by individuals, in France more than in England, with suicidal intent, or it is taken accidentally, as for instance, where a person has drunk lead lotion in mistake for his medicine.

Trousseau[1] used injections of acetate of lead to the extent of from 460 to 900 grains in a litre, without causing any symptoms.

When lead has been prescribed to be taken internally, it has been followed occasionally by symptoms such as those of gout in Dr Lauder Brunton's case, or as in the case of Fouquier,[2] who prescribed to a student of medicine suffering from phthisis, pills composed of 1 grain of acetate of lead. The first pill caused slight colic, the second produced very sensible effects, but the third created such severe colic, that it resulted in a magisterial inquiry. The apothecary who dispensed the prescription was accused of negligence, but on analysis, only one grain of lead acetate was found in each pill.

Wherever, therefore, lead has been freely used, either in the manufacture of articles of food or as a conductor of drinking water, there has generally been a considerable risk of lead poisoning. For several years now, there has been known in the Tropics a disease spoken of as the "dry belly-ache" or *colique sèche*, a disease which at times has played sad havoc with the sailors of the French Navy cruising in the Tropics. The West Indies and Guiana[3] have been noted as the districts in which endemic colic has been most apt to occur. For a long period its causation was misunderstood, but there has been a gradual consensus of opinion in favour of the view, that *colique sèche*, or

[1] "Clinical Medicine," vol. iv., page 81.

[2] "René Moreau, L'empoisonnement aigu par le plomb," page 35.

[3] Hirsch, "Geographical and Historical Pathology," vol. ii., page 269. New Sydenham Society.

endemic colic, is exactly the same as lead colic, and is traceable in every instance to the use of food or drink contaminated by that metal. The fact that the disease is almost entirely confined to the sailors of the French ships of war, and not in any corresponding degree to the inhabitants of the Tropics, has given rise to considerable discussion, and to not a little doubt as to the disease being Saturnine. By many it has been regarded as a neurosis of the sympathetic, and due to chill; others have regarded it as of cryptogenetic origin; whilst others have claimed for it a miasmatic or malarial origin. Contradictory as all these opinions are, the view now generally adopted is that of Lèfevre, the Director of the Naval Sanitary Board at Brest, who says, that "the *colique sèche* of the French Navy is nothing more than lead colic." Many writers who had previously expressed contrary opinions, have been gradually coming round to the view of Lefèvre, and the outcome of this expression of opinion from so high an authority is already seen in the good results which have followed, for the Ministry of Marine is taking steps to render the disease less frequent in the French Navy. When we know how very much lead enters into the construction of French ships of war, no astonishment need be expressed at the greater frequency of colic amongst French sailors than those of other countries. Either as tanks for holding water, or as pipes for conveying it, as plates for protecting the sides of the ship, in the form of putty and paint, or in the fact that the enamel of the drinking cups and cooking utensils on board a French man-of-war usually contain lead, this metal is more freely distributed throughout their ships than in those of other nations. The reason why English, American, and German sailors have not suffered in the same proportion, is simply because that, in their Navies, everything likely to give rise to lead poisoning has been carefully eliminated.

It is alleged by Dr Stephen Mackenzie, that in the autumn a larger number of lead cases is met with than at any other period of the year. On looking over the sick register at one of

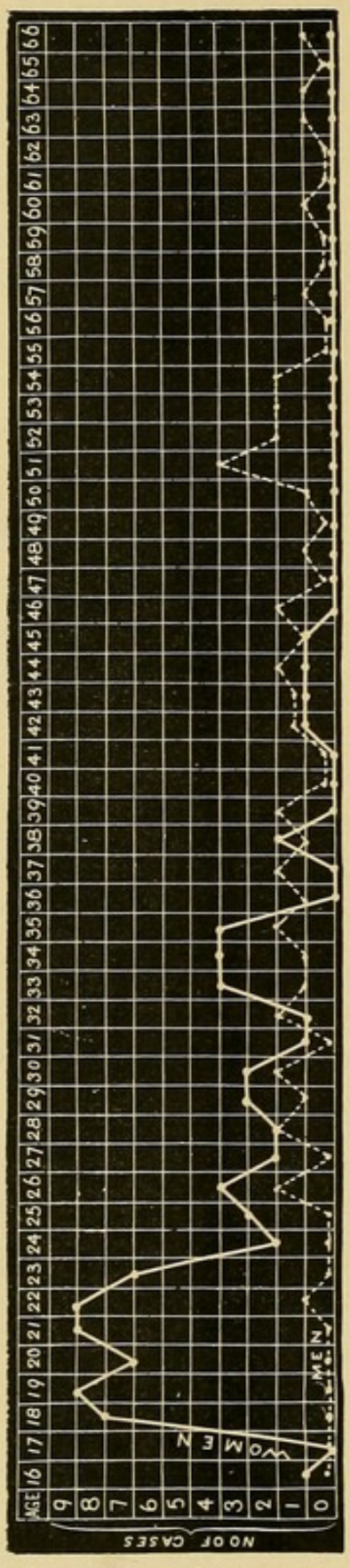
our lead factories, I find that in the months of June, July, and August, the largest numbers of "orders" are given to the workers to obtain medical relief.

How much of the prevalence of lead colic at one particular period of the year is accidental, it is difficult to say. Trade may not always be so brisk at one time as another. Last year we had a strike amongst the lead workers, which, beginning in May, ended in September. This fact renders the returns for the year unavailable for generalisation. Of the 1217 cases observed by Tanquerel Des Planches, the largest number occurred in July. The warmest months of the year showed, according to him, the largest number of cases, and in these months Dr Inglis, of Hebburn noticed, out of 947 cases of lead colic, that there was a preponderance of cases. Inglis supposes that the lead dust is in the warmer months of the year more freely dispersed in the air. In this season vegetables play a larger part in the diet of the workers. Some have thought the winter months have furnished the largest number of cases, and regarded this as due to diminished elimination of the poison. In the early part of this winter I have seen more cases, but I attribute this to the fact that, as it was known amongst my confrères that I was on the outlook for lead cases, a larger number has in consequence come under my observation. The season of the year, like climate, has, in my opinion, little to do with it, although Hirsch attributes the greater frequency of lead colic on board ships cruising in tropical waters to the fact, that the higher temperature there materially conduces to the development of lead poisoning, and that of all the men on board ship, those who are obliged to spend most of the day in an over heated and impure atmosphere suffer most, for example the men employed in the kitchens and engine rooms.

As regards the age at which lead poisoning is most apt to occur, an answer is with difficulty returned, owing to the fact that exposure to the poison is not equal at all ages. Children are not employed in lead factories, but it has been noticed that

when an epidemic of plumbism occurs as the result of drinking contaminated water, they suffer readily. The children of the botanic beer manufacturer, whose case I quoted, suffered; one died; but in the case of N——, whose illness was partly due to drinking contaminated water, none of the children suffered, nor did they exhibit even the blue line on the gums. Taylor, in his work on poisons,alludes to the case of a child who died after taking for five weeks one-fifteenth of a grain of acetate of lead twice or thrice daily. There was colic, constipation, and fetid breath, then emaciation, and the limbs became paralysed, death occurring after convulsions and coma. In Keating's Cyclopædia of Diseases of Children, there is an excellent account of lead poisoning as it affects children. By the author of the article, Dr Putnam, it is stated that, if facts really point to a relatively slight liability on the part of children to poisoning from minute doses of lead often repeated, it is a matter of interest, as it indicates in all probability a more active power of elimination on their part. According to Des Planches, the largest number of cases occurs between the ages of thirty and forty, whilst in the epidemic at Bacup, fifteen to fifty is the period over which the liability to lead poisoning extended. Adults may be regarded as more predisposed to plumbism, for it is seldom met with in people after the age of fifty, although I have met with it in men at sixty-six. Whatever doubt surrounds the question of age, there is, in my opinion, no doubt in regard to the very much greater susceptibility of the female to be contaminated by lead compared to the male, and this is not due simply to the fact of exposure in a lead factory, to what may be regarded as the greater dangers, but depends upon sexual idiosyncrasy. This is an opinion so totally at variance with that given by several authors, that I require to explain myself. My experience, drawn from hundreds of cases, is, that both as regards the acute and chronic forms of lead poisoning, women are much more quickly brought under its influence than men. The ratio of men to women employed in lead factories is in favour of the

women, and at first sight it might appear as if the liability was explained by the greater number of women exposed. Taking a period of five years, I find that 135 cases of lead poisoning were admitted as in-patients at the Royal Infirmary in Newcastle. Of these, 91 were women and 44 men. Des Planches, on the other hand, regards women as less liable, even though employed at the same operations at a white lead manufactory as men, and so did Alderson. To me there is no comparison of the greater susceptibility of the female; and that it is not altogether a question of trade, is shown by the fact that, in the recent epidemic of lead poisoning in Yorkshire, out of 1000 cases due to drinking of water contaminated by the metal, the special correspondent of the *British Medical Journal* found the proportion of females to males to be as 4 to 1. Against this it may be urged that women probably drink more water than men, but allowing for this, the proportion would be still in favour of the female. Brown found 153



Lead-Poisoning Cases admitted into Royal Infirmary, Newcastle-on-Tyne, during Five Years ending June 1889.

Fig. 1. (Reprinted by permission from "Transact. Royal Medico.-Chir. Society, 1890.")

males as against 251 females. Not only is the female more susceptible, but she is so at an earlier age than the male, and is more likely to suffer severely, and from such nervous accidents as epilepsy. I find that the ages at which the human female is most susceptible, is from eighteen to twenty-three, and that in men from forty-one to forty-eight; and the interesting point in regard to exposure to lead is, that whilst young women suffer readily from Saturnine poisoning, recovering quickly from colic only to be more readily and severely affected on again exposing themselves, men may go on working for years, ten to twenty, having only one or two attacks of colic, and then, after a very lengthened period of service, may still fall victims, either to lead paralysis, or die from the effects of a kidney lesion due to the poison. It is difficult to say what length of exposure to lead is required to develop symptoms. In a fatal case of acute lead poisoning which I saw lately, the girl had only worked forty days in the factory altogether; these forty days were spread over nine weeks. The table on page 22 shows the ages of lead workers admitted into the Infirmary, over a period of five years, suffering from plumbism, and indicate the differences between the ages of males and females.

Not only is there a sexual, there is also an individual idiosyncrasy to plumbism. "There is a class of women too easily affected by lead,[1] but what that type is, it is impossible to say. Generally the class from which these people are taken are young girls who are practically without the comforts of a good home, many of them lead a questionable life, they expose themselves to cold, and are frequently in a state of chronic starvation before going to the lead works, and are therefore in a fit state for rapidly breaking down under the influence of lead. Those who are careful in regard to personal cleanliness, and are well cared for at home, do not as a rule suffer. I have seen women in the lead works who have followed their occupation for twelve or twenty years, and who have seldom been away from

[1] T. Oliver. "Local Industries," British Association, 1889.

work more than two or three weeks, when they have suffered from constipation and colic. It is the ill-fed, the badly housed, and thinly clad young girl who suffers early ; women who are labouring to support idle or drunken husbands or paramours, or women who have lost their husbands, and having failed at other occupations, betake themselves to the lead factories, tempted by the high wages, where, after a few months' or a few weeks' service, they become the victims of plumbism. Apart from the influence of external conditions, such as want of adequate food and exposure, there is with this, as with most other diseases, not only an individual, but a family predisposition to lead poisoning. Whole families will suffer readily, others scarcely at all. This observation applies to both sexes." I have known a family in which four sons and the father, all strong and well built men, smelters, died from the effects of lead poison ; and I know of other cases where, probably as the result of heredity, the sons and grandsons of men who were house painters have become early and severely affected by lead, the hereditary taint markedly precipitating the attack. Again I repeat, female idiosyncrasy predominates. One of the first noticeable effects of the pernicious influence of lead, is the production of anæmia or cachexia. Nearly all young women, those particularly between the ages of eighteen to twenty-four, when thus exposed, suffer from deranged menstrual function; hæmatosis and ovarian activity are interfered with, and the result is either amenorrhœa or menorrhagia. Once the functional activity of the ovaries and blood-making is interfered with, then is that woman already in a critical condition, and at any moment she may become the subject of any of those explosive outbursts of plumbism, known by the name of lead encephalopathy. To sexual peculiarity I therefore attribute much of the danger from exposure to lead. Lead as a poison strikes early at the functions of blood-making and reproduction, producing sterility, liability to abortion, and amenorrhœa or menorrhagia. Woman, from her constitutional idiosyncrasy, is therefore more liable to be impressed by lead.

Whatever may be the power of sex, of family, and individual predisposition, however potent may be the effects of inadequate food or clothing, if there is one agent which must greatly increase the influence of one or other of these as factors in determining lead poisoning, it is alcohol.

Once the individual has suffered from an attack of lead colic, or any symptom of lead poisoning, he is launched upon a morbid stream of life, where, under the influence of metabolism, which is making for health, he may safely steer his course and recover his health; on the other hand, if he is inattentive or unwilling to regulate the requirements of his constitution by abstinence from alcohol, and again exposes himself—preferring thus to be wafted by the breeze that placed him on that stream—he floats the more rapidly down, heedless of the gulf in which he is so soon to be submerged. Between the first and subsequent attacks there are periods of quiescence; the first attack predisposes to the second, and this to a third; or a first attack is experienced, and without any fresh exposure, there is, long after it may be, another outburst. What is the meaning of this? Is it that during the period of apparent health lead has been deposited in some of the organs in an insoluble state, and that as the result of some particular diet, drink, or medicine, it has again been rendered soluble, circulates afresh in the system, and, for the time being, the person is poisoned with lead absorbed from his own tissues?

LECTURE II.

At the close of last Lecture, we were dealing with the peculiar susceptibility of woman to the influence of lead, and we saw that under any circumstance, once Saturnine poisoning was induced, it was particularly apt to recur on exposure of the patient. Indeed, sometimes the symptoms reappear without exposure, the explanation being that lead, which had been deposited as an inert compound, by some changes either in the blood or in the fluids, had become converted into a soluble lead compound, and in this form was reabsorbed.

I shall deal in my third Lecture with the channels through which lead enters the system. It is sufficient to say, that either in a state of solution, of fine sub-division, or dissolved in some volatile agent such as turpentine, lead is taken into the system. In one or other of these forms, and by channels subsequently to be described, lead is introduced into the body, and soon its insidious and pernicious effects are seen.

Before dealing analytically with the symptoms of lead poisoning, I shall briefly give an outline of three or four types of plumbism; two shall be acute, and two sub-acute or chronic.

An individual in good health, with obviously well filled blood-vessels, engages in a factory where lead compounds are handled, and where powdered lead dust is floating in the atmosphere, or, as the result of accidental conditions of which he is unaware, he receives into his system for a time, either in water or in food, a small quantity of lead. Such a one is ultimately seen to become pale and sallow, and concurrent with this anæmia or cachexia,

the features, which had remained full and fairly rounded, have through failure of nutritive processes become pinched, and the face lost its natural expression. When questioned as to his health, we learn that he has no definite symptoms of disease, no special illness, only he is not well, each function is working sluggishly. Examination discloses a small and rapid pulse; on his gums may be detected a well marked blue line, and we therefore conclude that the case is one of Saturnine cachexia. At this stage the doctor is seldom consulted, the individual scarcely feeling ill enough to consult a medical adviser. Subsequently, however, these symptoms manifest themselves; a disagreeable metallic taste in the mouth, occasional morning sickness, headache, a sense of weakness in the limbs, loss of appetite, and usually constipation. Frequently the constipation is so enduring that the bowels may be closed for weeks. After these symptoms, the patient is suddenly seized with an acute pain in the abdomen. Confined to the region of the umbilicus, sometimes a little above it, but more frequently to the left, the pain is either of a twisting or a grinding nature, or it creates a sense of weight and constriction. These pains are simply excruciating. Sometimes the patient receives relief from pressure, sometimes from warmth; at other times he is unable to be touched. The colic subsides, only to recur in paroxysms, and with a tendency to nocturnal exacerbations. Generally, after the acuteness of the pain has subsided, nausea, retching, or vomiting of a thin greenish liquid occurs, accomplished with difficulty and accompanied by restlessness. The secretion of the urine is diminished, and the pulse is hard and slow. In such a case under treatment, the pain, gradually subsiding in severity, keeps returning in milder and milder paroxysms; the vomiting abates, the bowels yield to purgatives, the spirits improve. In a few days the patient is convalescent. Here I have attempted to portray the conditions involved in an attack of acute lead colic.

I shall take as my second type, a case of acute lead encepha-

lopathy. A young healthy girl of an age ranging from seventeen to twenty-four, well developed and lively, and with normal menstruation, seeks employment in a lead factory, where she is placed in either the "white beds" or the "stoves." In whatever part of the factory she is employed, she is brought into direct contact with powdered white lead. Here, after a varying degree of exposure, she becomes anæmic. It may be that her gums show a very faint blue line, or perchance her teeth and gums are perfectly sound, and no blue line is discernible. Coincident with the anæmia, she has been getting thinner, but so gradually as scarcely to impress itself upon either herself or friends. Sickness, however, is now complained of, but there is neither constipation nor abdominal pain, or if these have been experienced, the condition has long been passed through, and was at the best but slight. Her menstruation is either scanty or profuse, and as a consequence headaches, growing in intensity, are developed. These are frequently attended by obscuration of vision or temporary blindness. Such a girl passes into what appears to her friends and medical adviser as ordinary hysteria. This gradually deepens, or without warning, and more often without than with an aura, she is suddenly seized with a convulsion, beginning in one-half of the face, then involving the arm, next the leg of the same side of the body, until the convulsion, violent and purely epileptiform in character, becomes universal. This is attended by loss of consciousness, out of which she passes into a series of convulsions, gradually increasing in severity, in one of which she dies. Or consciousness, partial or perfect, is regained, either it may be for a few minutes, a few hours or days, during which violent headache is complained of, or she is delirious and excited as in acute mania, or dull and sullen as in melancholia and requires to be roused, when she is found wandering and her speech is somewhat imperfect. Without further warning save the pulse, which had become soft, and with nearly the normal number of beats all at once becomes slow and hard, she is suddenly seized with another convulsion, in which she dies

or passes into a state of coma, from which she never rallies, the urine, previously scanty but healthy, ceasing in the last hours of life to be secreted. In another case, the convulsions gradually subside, the headache disappears and the patient recovers, only to find that she has completely lost her eyesight, a loss that may be temporary or permanent.

The third type shall belong to the neuro-muscular. A man or woman has for some time been brought under the influence of lead, either at his occupation, or he has been receiving lead into his system through food or drink, and after passing through states of failing health, such as I have already mentioned, he has an attack of colic, recovers, again exposes himself, and again suffers from abdominal pain. After several attacks of colic, or perchance without any such attacks, he begins to feel numbness in one or more fingers, or in his arms, accompanied by pains either in the muscles of the forearm or in the wrist, elbow, or shoulder-joints, and he notices that, either insensibly or gradually, he has lost the power in one or two fingers, or in the muscles of the forearms. The hands fall powerless from the wrists, the arms from the shoulders, and in this condition the patient, unable to feed or clothe himself, lies helpless in bed with hands crossed. Or the case is still more acute and the paralysis more extensive, for in getting up one morning, the patient suddenly feels that he has lost the power in one or both legs, and that he is quite unable to stand. In a few hours, or it may be days, the paralysis has extended to the muscles of the back, the shoulders, and the arms. In one or other of these ways, which may be described as characteristic of Saturnine poisoning, though always with a predilection for the extensor than the flexor muscles to be involved, lead paralysis is established, unaccompanied by loss of sensation, although occasionally that loss may supervene. This paralysis is always accompanied by muscular atrophy of a rapidly developing nature. Owning no special relationship to colic, or the nervous symptoms met with in the severe cases, and yet generally following one or other of these, the paralysis

may disappear as quickly as it came; or it may, and often does, last for weeks or months, the cure being frequently incomplete, the paralysis apt to recur on exposure, or it persists, and the patient drifts into the fourth type which receives all. That type is briefly this:—

A man or woman has for many years been exposed to the influence of lead. There is the well-marked cachexia, with a history of repeated attacks of colic, possibly even of convulsions. Or if neither of these has been experienced, for months the patient has had pains in the muscles of the limbs, particularly in his calves or arms and forearms, attended by a sense of lassitude and a feeling of unfitness for work, or pain is complained of in one or several joints, many of which, particularly the metacarpo-phalangeal, are deformed; attacks of gout have been experienced, emaciation is steadily progressive, and certain extensor muscles are noticed to be atrophied; the general health is noticed to be failing; albumen is detected in the urine, and in this condition of chronic lead poisoning, where no one function in particular is affected to the exclusion of another, all suffering more or less from the dycrasia, now no longer depending upon recent exposure to lead, but aggravated by the condition of the kidneys, such a person, prematurely old, lives it may be for years, dying ultimately from nephritis or some intercurrent brain, lung, or heart affection.

Of the various symptoms and physical signs of lead poisoning, the one physical sign above all others which indicates that the system is becoming impregnated with lead, and is suffering in consequence, is the developement of anæmia, which becomes gradually confirmed, and is spoken of as Saturnine cachexia. Almost from the first day of exposure to lead, hematosis is interfered with. I have never found in any lead worker the normal number of red blood cells. Concurrent with the reduction in the number of red cells, a reduction at first rapid, but ultimately checked (I have seen it 2,500,000 in a girl, after twelve days' exposure), the average range varies from 2,500,000

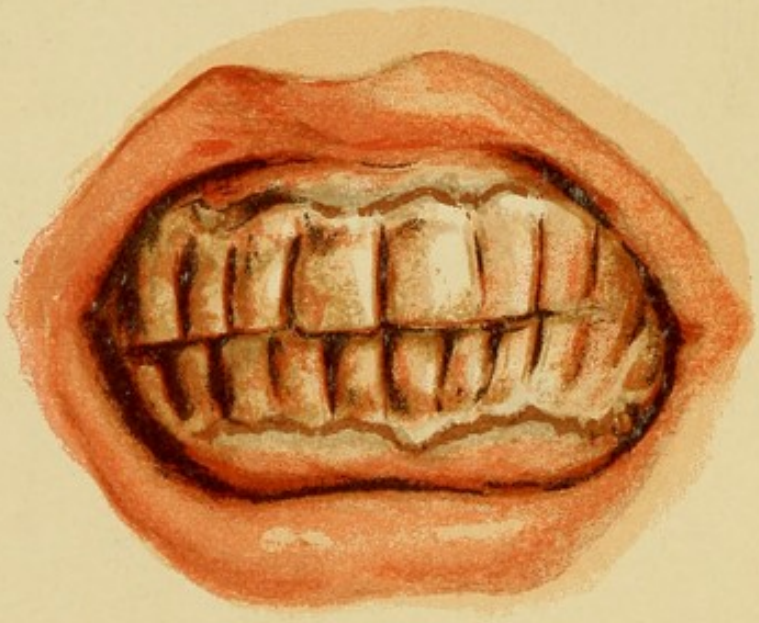


FIGURE II.

FIG. 2.—Blue line on gums. J. K. had only worked amongst red lead for twelve days.

to 4,000,000 in 1 cmm. There is no marked increase in the number of white cells, but the colouring matter falls as low as 45 or 50 per cent. of the normal. Not only does the face assume the appearance of anæmia, but the features become rounded and expressionless. The first effect of lead upon the system, in my opinion, is disturbance of the functions concerned in the making of blood, and closely following upon this comes interference with nutrition. The bones have, in some of my cases, contained lead, and to this fact the anæmia may be partially attributed. Raimondi observed atrophy and degeneration of the bone marrow in lead poisoning.

The physical sign of plumbism, however, which is most looked for, and generally met with, is the presence of a blue or slate coloured line on the gums, noticed by Burton, but previously described by Tanquerel Des Planches. When present, it is a valuable sign, but it may be absent; it generally is when the teeth and gums are perfectly sound, and the mouth is kept cleansed. And yet there may be colic, and no blue line, or a girl may die from acute encephalopathy, with no blue line on the gums, characteristic of the poison present from first to last. There are two blue lines on the gums that must be distinguished from each other—one is a delicate line just at the margin of the gums and teeth, but not involving the gum: it is a deposit in the interstices between the teeth, and is seen in workers who have been exposed to white lead dust for a few hours. This is removed by cleansing the teeth and rinsing the mouth: it is simply a deposit of lead sulphide on, and not in, the gum. The other, the characteristic line, is bluish black, and occurs also at the margin of the gum, where it is not in complete apposition with the teeth. There the gum is frequently ulcerated and irregular in outline. The teeth are discoloured, and covered with a brownish soft pulpy fur, the odour of the breath is disagreeable, and the patient complains of a metallic taste in the mouth. Where the teeth are absent there is no blue line. I have seen this line develop the day after a large dose of acetate of lead had been

taken: the line persisted for a few days, and then disappeared. Out of several scores of cases examined by me, I find when the blue line is absent from one gum, that it is more frequently found in the lower than the upper; if present in both gums, that it is usually more deeply developed in the lower. The deepest tint is observed underneath the outer incisors and canines. The fact of the line being absent, where the teeth are perfectly white, and the gums healthy, suggests as a necessity for the development of this physical sign, that there must be a broken surface of the gum. This alone, rather than bad teeth, although the two are generally associated, makes it difficult to assign to each its proper share. That the gum, however, contributes most to this is shown by the fact that, in girls who have been exposed to lead dust for a few hours, or a day or two, and in whom no blue line has been observable, I have on several occasions found the gums swollen and red, but not painful, as if they were the seat of irritation. It is in gums such as these that we subsequently meet with a blue line. An interesting point about blue line in the gums is, that I have never found it in animals poisoned by lead. The blue line, however, though noticed most frequently, and as being most highly developed along the outer margin of the gum, is also seen running along the inside towards the molar teeth. The blue line may be present for months, and yet no other evidence of lead poisoning may be present, save that of Saturnine cachexia, general mal-nutrition, occasional constipation, and slight colic. It is difficult to say, positively, how long the blue line of plumbism will remain after removal of the individual from the influence of lead. Tanquerel speaks of the difficulty of removing it except by friction, and the use of water acidulated with sulphuric acid. No kind of mouth wash has, under my observation, hastened its removal. It disappears under internal treatment. In some of my own cases it has disappeared very quickly: in one within eight days, usually, however, from two to three weeks in its minor forms, three months or longer in the severer cases. This is the limit which I expressed at a

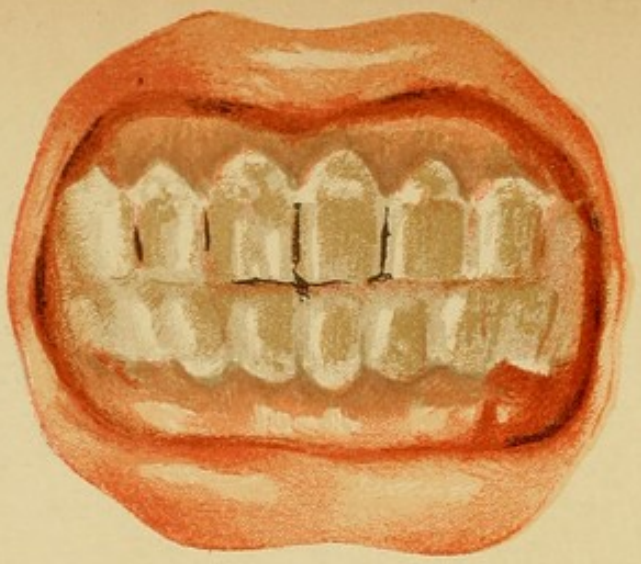


FIGURE III.

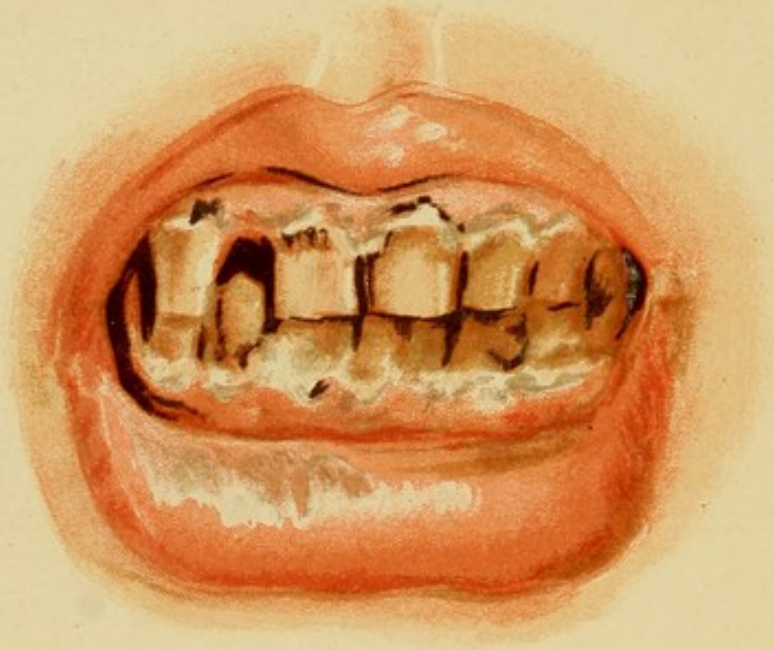


FIGURE IV.

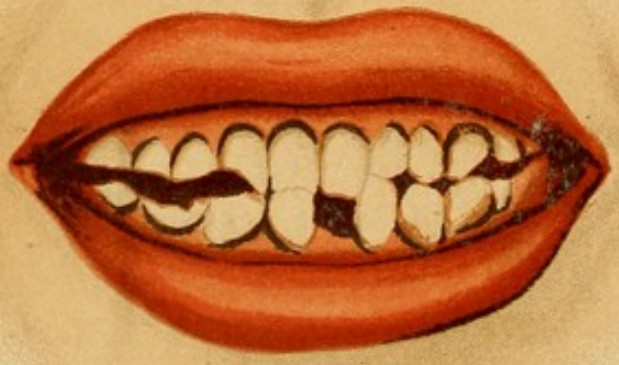


FIGURE V.

FIG. 3.—Blue line on gums; rapid recurrence of symptoms of lead poisoning. M. W. had entirely lost the blue line on her gums. Circumstances obliged her to return to the lead factory; after working there one week she came to the Infirmary suffering from colic.

FIG. 4.—Blue line on gums; also blue patch on inside of cheek, opposite lower incisors and canines (right). Rachael H., æt. 35.

FIG. 5.—Black line on margin of gums and teeth.

meeting of the Royal-Medico Chirurgical Society two years ago, in answer to a question of Stephen Mackenzie, who, with Gowers, regarded a year as the period during which the line may be present. Since that date I have carefully watched the disappearance of this line, and can confidently say that within three months all traces of it may have passed away, whilst ulceration might still remain.

One of my cases, Mrs W., aged 38, a lead worker for eighteen years, and who had suffered seven times from colic, showed a blue line on her gums in the month of February 1889, although she had not been at the factory for six months. In November of the same year she brought her daughter to the Infirmary suffering from lead poisoning, and I took the opportunity of examining the mother's gums. All traces of blue line had disappeared, but the gums were ulcerated along their margins. Owing to the daughter's illness, the mother returned to the factory, from which she had been absent for eighteen months. In the following month, namely December, after having worked only one week, she returned with a most intense blue line.

In addition to the blue line on the gum, there are occasionally to be noticed discoloured patches of mucous membrane, varying in size from a split pea to a shilling, inside the cheek, most frequently met with opposite the lower canine and outer incisors. It is said by some authors that the administration of potassium iodide will develop a blue line in plumbism when absent. I have never succeeded in doing so, nor has Inglis, who has repeatedly tried the experiment, managed to induce it by these means. The other metallic poisons which give rise to a line on the gums are copper and bismuth. I show you, however, a black line due to some form of carbon, met with in a miner who was under the care of my colleague, Professor Philipson, in July 1890. The man, aged 40, was admitted into the Infirmary complaining of pain in the back and loss of power in the legs, of three years' duration. He was cachectic, had drunk largely of beer, but never out of a metallic vessel. He came from Cum-

berland, where he had been working in a 20-inch seam; the coal was soft, and there was much dust. In this man there was a distinct bluish black line at the margin of the teeth, which struck observers differently, as being black or bluish according to the light in which it was examined. That on the lower gums, when looked at from above, appeared as a delicate line, bluish black in colour, but on careful examination it was noticed that the line depended upon a deposit of dark pigment upon the teeth, whilst underneath the gum was retracted and exhibited a slight red line. There was no deposit in the gums. In diseased conditions, attended by marked deterioration of health, as for instance phthisis, a delicate red line is occasionally noticed on the gums, and in copper poisoning the gums are ulcerated and a greenish blue colour is noticed, depending upon a deposit in the depth of the ulcer.

In Fagge's "Principles and Practice of Medicine," there is an admirable description of the Burtonian line, and of its differentiation from the bluish purple border noticed in the gums as the result of irritation of metals other than lead. On microscopical examination, the blue line of plumbism is found to consist of a series of black dots, granules of sulphide of lead. Fagge thought that these granules occupied the interior of small blood vessels, the ramifications of which were mapped out by their presence. It seemed to him as if the blue line was due to an excretion of lead from the blood. An examination of the accompanying drawing does not show this relation of the black granules to the blood vessels: part of the gum in which the dots occur is non-vascular. That the blue line is due to a deposit of sulphide of lead, there is no doubt. The sulphur in all probability comes from the sulphuretted hydrogen generated during the decomposition of particles of food left between the teeth. I regard this as the source rather than the sulphocyanogen of the saliva. I cannot altogether support the view that the lead has been eliminated from the blood circulating in the gum, and that it is deposited therein in an insoluble form around the vessels.

FIG. 6.—Section of gum with blue line in lead poisoning. (× 250).

FIGURE VI.

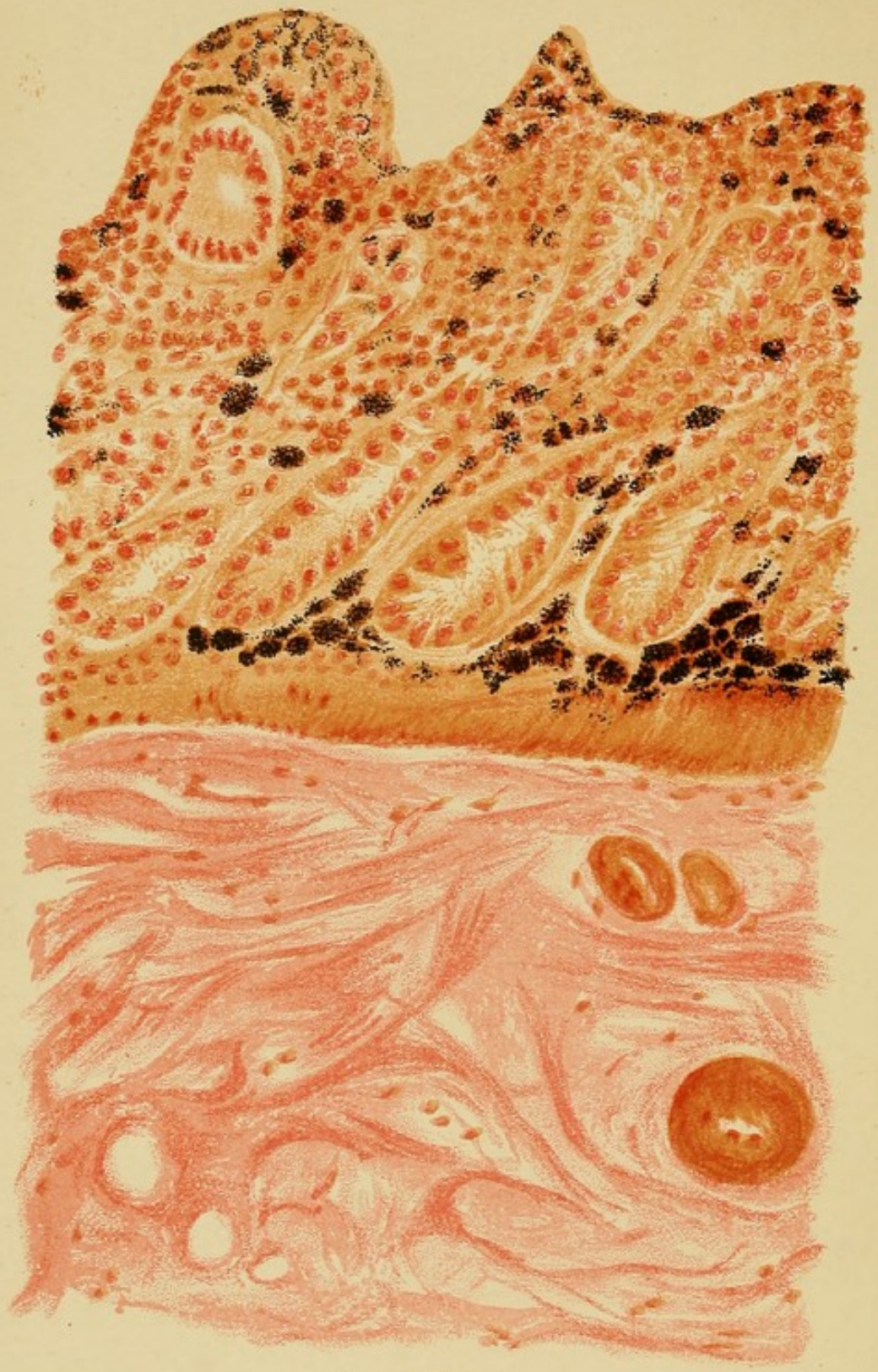


FIGURE VII.

FIG. 7.—Large intestine ; section showing deposit of lead in mucous membrane. (× 250). Case of Barbara R.

Lead is eliminated from the blood. It may be thrown out by the salivary glands, for example. I am inclined to think that there is an absorption of lead from the mucous surface, either by the lymphatics or by the cells themselves.

The mucous membrane of the intestine occasionally shows bluish-black discolourations. These patches, on microscopical examination, exhibit a similar condition. Numerous large black dots are noticed on the surface of the mucous membrane also in and underneath it, but the granules do not appear to be specially associated with the blood vessels. It is rather with the lymphatics and the large cells of the mucous membrane.

One of the earlier symptoms complained of by patients suffering from plumbism, is sudden and severe pain in the abdomen. Generally it is preceded by such prodromata as a metallic taste in the mouth in the morning with vomiting, or only by a feeling of sickness with loss of appetite and constipation, and occasionally diarrhœa. Colic occurs early after exposure to lead; in one of my cases, seven days after. The pain is frequently so very acute, that the patient rolls about in bed in agony, generally obtaining relief by pressure and warmth, but frequently he is unable to bear the slightest pressure. Sometimes localised, the pain, when paroxysmal, is felt in the neighbourhood of the umbilicus, or a little above it, and to the left; at other times it is felt in two or three diffierent places in the abdomen, or it may be generalised. Occasionally in males, the pain radiates into the testicle. These attacks are varied by remissions, during which the pain is lessened in severity, but never quite disappears. Constipation and pain, as a rule, go together, but in a few cases there is no constipation, the bowels are regular, or there may even be diarrhœa. The pain has no special relationship to the colon, nor can it altogether be regarded as simply ordinary colic. Pressure is said by most writers to relieve it, but sometimes the patient cannot bear to have the abdomen touched. There are two kinds of pain: the paroxysmal or acute pain—the true lead colic, which is generally relieved

by pressure; and the other is the dull continuous pain that follows it in the intervals. It is this pain which is usually aggravated by pressure. In all cases where colic is well marked, there is excessive restlessness. Accompanying the colic, or preceding it, there is very obstinate constipation, with headache and inclination to vomit. In one of my cases—a painter—there had been no constipation until the first attack of colic, when, for twenty-one days, constipation was absolute. In each succeeding attack the colic has been preceded by constipation, and the only relief the patient experiences, is when he leans over a chair and firmly presses his abdomen thereupon. The abdomen is retracted, or if not markedly retracted, it is resistant to pressure, and as a rule, this resistance to pressure continues for some time after the acute pain has disappeared. Where repeated attacks of colic have been experienced, there generally remains a degree of tenderness, limited to the half of the abdomen which was the primary seat of pain. Deep pressure on that side is not only opposed by increased resistance, but pain is felt. On the whole, I am inclined to think that this occurs more frequently on the left than on the right side of the body. In one of my cases, a painter in a shipyard, who is the subject of recurrent lead colic, with its attendant depression of spirits, firm pressure can be made on the right side of the abdomen, which remains perfectly limp, but the moment similar pressure is applied on the left, that side of the abdomen becomes resistant, and severe pain is felt. It is difficult to say what produces the acute pain in lead colic. In a guinea pig, that had lead mixed with its food, I found the small intestine in a state of varicosity, portions of normally distended intestine alternating with other portions so extremely contracted as to have their calibre almost completely obliterated, and the piece as hard as whip-cord. There existed a contraction, which pressure, when applied to the more widely distended tube, could not overcome. In other words, a veritable stricture, amounting to tetanic contraction, existed for several inches—due to extreme shortening of the circular muscular fibres

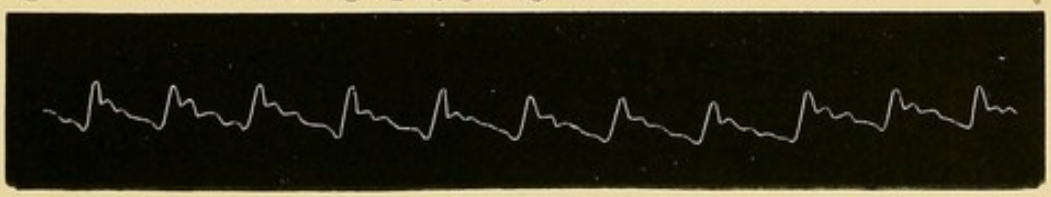
of the bowel. A spasmodic constriction of the small intestines, occurring at irregular intervals, appears to me to be the cause of the pain in lead colic by pressure upon the nerves, and this is aggravated by the efforts made by the distended portions of the intestine to propel their contents into the contracted tube below. Lead is regarded by some, particularly Gusserow, as having a special affinity for unstriped muscular fibre—more particularly that of the intestine. No matter the channel by which it has been introduced into the system, as for example, when Moreau injected solution of lead acetate into the crural vein of a dog, whose abdomen he had at the same time opened, so as to watch the intestines, Moreau noticed, after a time, irregular contraction of portions of the small and also of the large intestine. How far does this circumstance support the view of Henle, that the action of lead is primarily on muscular tissue, or that of Heubel, that its first effect is on the nerve centre? My own impression is, that lead may act directly upon the muscle-fibre-cell, both around the arteries, which are generally found thickened in chronic lead poisoning, and also upon the intestine; but it is much more likely that it acts primarily upon the large nerve ganglia which are in the abdomen, or, according to Harnack, upon ganglia in the intestinal wall. Unstriped muscular fibre does not respond quickly to stimuli: nerve tissue, on the other hand, is much more irritable. Gusserow maintained that muscular fibre had a special affinity for lead, and that post mortem lead was always found to predominate in the voluntary muscles. Heubel found, however, just the opposite of this, namely, that brain and spinal cord contained the most lead after the liver and kidneys. The intestine is found at times to be irregularly, but extremely contracted: hence the spasm would cause pain, and the arterial ischæmia, which follows, would diminish the secretion in the intestine, causing dryness of the fæces and constipation. But I have found liquid stools in guinea pigs, and yet there were irregular spasms; and in the human subject, constipation is not a necessary accompaniment of colic. If the effect

were upon the nerve centres, we might expect that a larger part, or the whole of the intestine, would be involved. The extent to which this spasmodic contraction of the intestine occurs, explains the retraction of the abdomen, and yet, if we were to draw an inference from the retracted state of the belly, as to whether the determining cause is in the muscle or nerve centre, from a disease in which this condition is most noticeable, namely, meningitis, we would at once say it was in the nerve. Harnack[1] inferred from experiments upon animals, that the intestinal ganglia were thrown into a state of excitation by the poison, whereas Riegel thought the intestinal arteries first were narrowed, and that this was followed by intestinal contraction. This spasmodic contraction, once established, is capable of persisting for a variable length of time, and by its opposing the downward movement of the intestinal contents, must give rise to constipation, a condition which is admittedly nearly always present in colic.

We cannot easily dismiss this question of colic, it being part of another and much larger problem. During the attack of colic the pulse becomes hard and resistant, and is very decidedly retarded. A pulse which has been beating at the rate of from 70 to 80 in the minute, and has exhibited no character other than normal, will, during an attack of colic, sink as low as 40, and become extremely hard and incompressible. The average rate of pulse beats during lead colic, is rather less in the male than in the female, and a few hours after the attack of colic has passed off, the beats will return to their normal number, and the pulse resume its softness. I would not call the pulse of lead colic a feeble one, it is rather hard than feeble. It is interesting to note the slowing of the heart's action, as part of this process. It is explained, I think, as Eulenburg and Guttmann have shown, by reflex inhibition, through the mesenteric and cœliac plexuses of the abdominal sympathetic—the fibres which convey the impression being sensory, and running,

[1] Arch. für exper. Pathol., ix., p. 152.

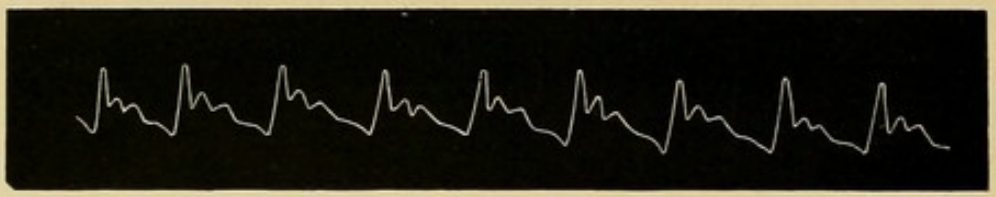
according to Bernstein, along the mesenteric artery. I agree with Riegel that colic, high arterial tension, and retarded action of the heart, are all present together. Are they coincident? The high arterial tension only lasts at first during the colic, hence the absence in the early stages of lead poisoning of the hypertrophy of the heart which is noticed in the later stages. It is difficult to separate, in point of time, the three states, colic, high tension, and slowed action of heart, but it seems to me as if lead first irritated the sympathetic and intestinal ganglia; this is followed by spasm of portions of intestine and arteries, and by inhibited heart's action. That such is the course of events is confirmed by a consideration of the functions of the kidney. During lead colic, very little urine is secreted. In some of my own cases there has been suppression of urine, or from 6 to 12 ounces of urine only have been secreted daily. Wherever there was severe colic, and heightened arterial tension, just in proportion was the amount of urinary secretion diminished, and correspondingly its specific gravity increased. The interesting point observed in my cases is this, that after an interval of freedom from pain which had been accompanied by high tension, and its attendant deficiency of urination, when the colic had returned days after, arterial tension again rose, and the same deficiency was observed in the amount of urine secreted. The facts here enunciated were also observed by Riegel. The following sphygmograms indicate this :—



Cath. Reynolds, aged 23. Severe headache, lead poisoning. June 28, 1889. Made good recovery; returned to lead works, and followed her employment.

Cath. Reynolds, aged 24. Completely blind, marked optic atrophy. No albumen in urine. July 16, 1890. Arterial tension has become considerably increased.

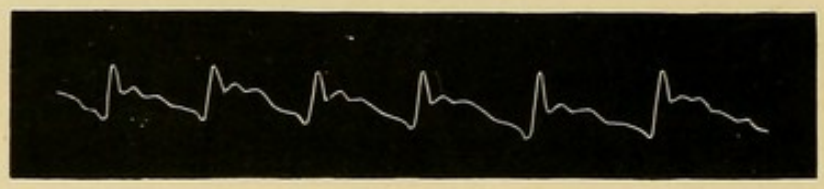
Barbara R., aged 33. Acute lead encephalopathy. Convulsions. Tracing taken a few hours before death. Patient comatose. Urine contains albumen. 5th Dec. 1890.



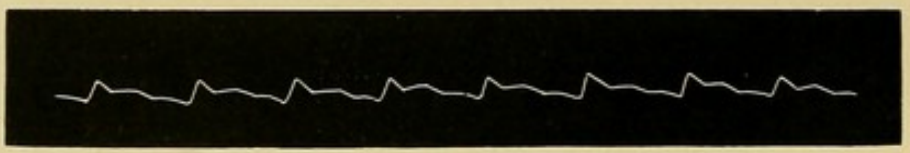
Annie K., aged 27. Lead worker. Taken at factory. Oct. 2, 1890, shows normal tension.

Annie K. Right radial ; has recently had colic ; is still occasionally pained. Dec. 24, 1890.

Annie K. Left radial. Dec. 24, 1890.

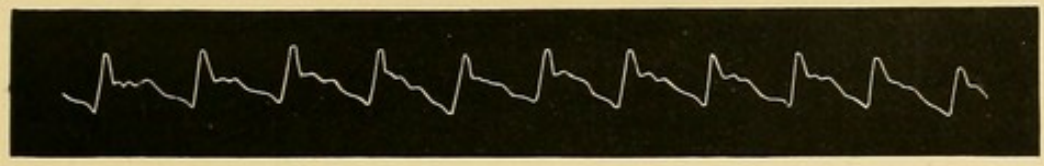


Catherine M'G., aged 19. Oct. 2, 1890. Taken at factory. Never had colic.

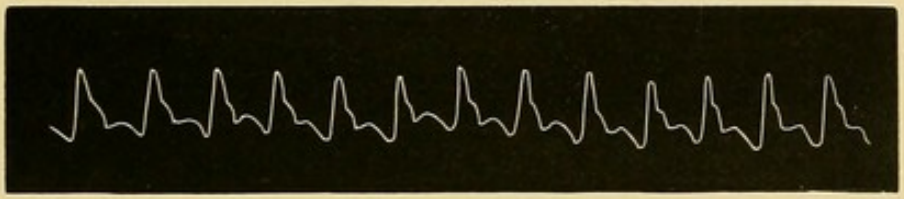


Cath. M'G., admitted into infirmary 28th October 1890 ; is suffering from lead colic ; right radial.

Cath. M'G. Left radial. 28th Oct. 1890.



Harriet E. Taken when cramp-like pains passing off.



Harriet E. Colic had quite disappeared.

Thomas K., aged 40. Lead colic ; had only worked in factory twelve days. March 20, 1890.

Eliz. B. Lead poisoning. 31st Oct. 1886.

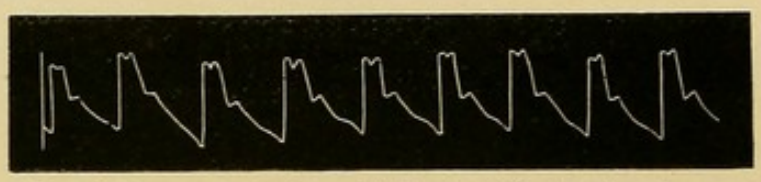
Eliz. B. Lead poisoning ; shows increased tension since last. 7th Nov. 1886.

August P., aged 31. Dec. 31, 1876. Violent abdominal pain.[1]

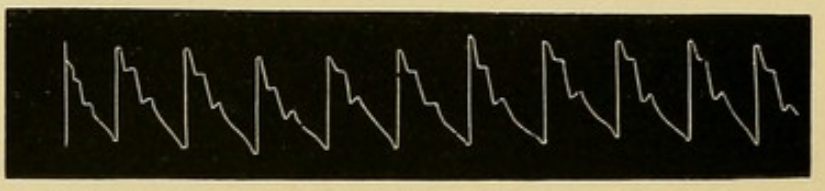
2nd January 1877. Considerable decrease of pain.

[1] Reprinted from Riegel's Article, Deutsch. Archives für klin. Med., 1878, p. 195.

4th January 1877. More violent pain.



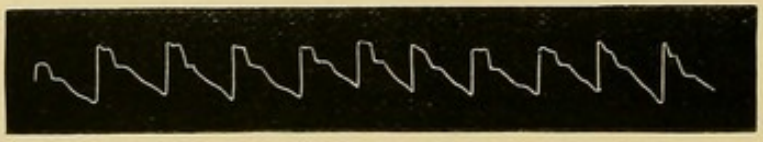
6th January 1877. Violent abdominal pain.



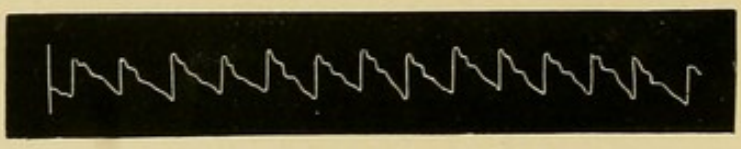
7th January 1877. Considerable decrease of pain.

8th January 1877. Painless.

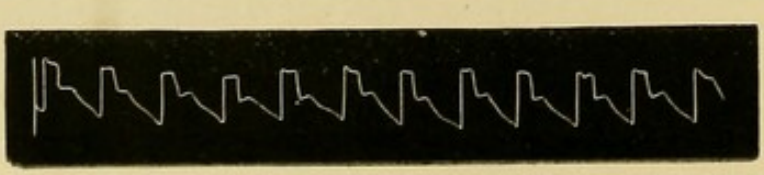
11th January 1877. Painless.



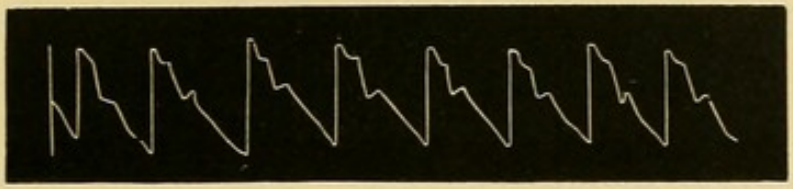
12th January 1877. Again moderate pain.



13th January 1877. Pains less.



15th January 1877. Pains more violent again.



16th January 1877. Painless.

24th January 1877. Convalescent.

Arterial spasm in the splanchnic area must, therefore, be very general, and whilst explaining the high tension in the vessels, and the accentuation of the second aortic sound noticed in these cases, it seems to be accompanied by irregular spasms of the intestinal tube, generally the small intestine, and this is the cause of pain. I do not agree with Traube, when he says that the pains are merely a consequence of the violent intestinal peristalsis set up by an accumulation of hardened fæces, for pain may be present whilst there is no constipation, and may be absent notwithstanding the most obstinate constipation. If present, this becomes an additional factor. Pain stands associated, not with constipation, but with heightened arterial tension, and the height of the arterial tension is the measure of the severity of the colic, and the deficiency of renal function. In my own cases I have often noticed this. Frank and Riegel[1] have attempted a solution of the problem, as to whether lead when it is absorbed causes pain as the primary condition, or whether there occurs first high arterial tension, and then pain, or whether the two may be simultaneous. Patients were treated by hypodermic injections of morphia during the attacks of colic, but whilst the pain was allayed, no reduction of arterial tension was observed. By this means sensation was alone affected. It became necessary, therefore, to obtain other results, and by means which would not involve sensation, or the conduction of

[1] Deutsches Archiv für klin. Med., 1878.

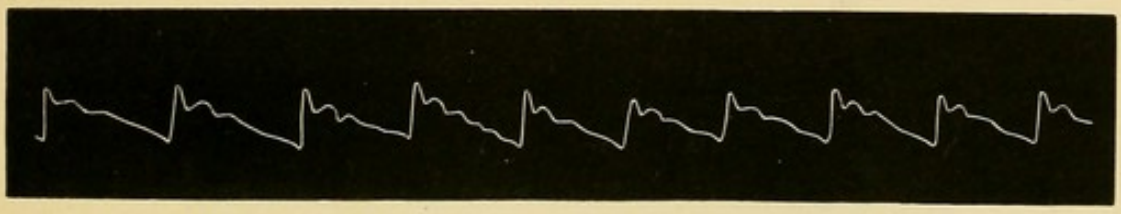
sensory impulses. Nitrite of amyl was inhaled by patients suffering from lead colic, and the consequence was an immediate fall of high arterial tension, and sudden disappearance of pain. As the effect of the nitrite of amyl passed away, the pain soon returned, and with its return heightened arterial tension.

For a similar purpose Bardenheuer tried pilocarpine, and here, as in the experiment with nitrite of amyl, a sudden fall of arterial tension took place, and with it disappearance of pain—both returning again when the effects of the pilocarpine had passed away. According to Riegel, the heightened arterial tension is the cause of the pain in lead colic, owing to the sensitiveness of the vascular nerves in the abdomen being in some way exaggerated, but he leaves out of consideration the accompanying spasms of portions of the intestinal tube which I have observed, and in which I am supported by the observations of Réné Moreau.

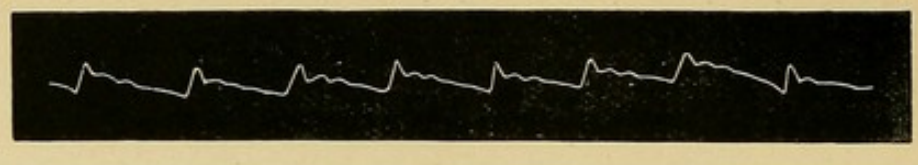
How far a degree of heightened arterial tension, associated from time to time with the minor attacks of pain in the abdomen, and the constipation which so usually accompanies it, aids in developing anæmia or cachexia, by causing malnutrition through impeded flow of blood, it is impossible to say. It is this very marked constriction of artery which causes almost complete suspension of the renal function. Dealing still with this question of colic, I have been much interested in the following facts, which I have lately observed, and which, in my opinion, help to prove that lead acts primarily upon nerve tissue, and not upon muscular fibre. In nearly every instance where lead colic has been experienced, and the minor degrees of it keep returning, there will generally be noticed a slight inequality of the two pupils; sometimes the right is the larger, sometimes the left. This is interesting when looked at in the light of Dr John Ogle's observations upon the altered state of the pupil, and its relation to pressure upon the thoracic ganglia. My idea is that deranged function of certain parts of the abdominal sympathetic, or of sympathetic fibres, coming, as Gaskell has

shown, from the thoracic part of the spinal cord, passing to the solar plexus and through it to the intestine, causes inequality of the pupils. I have observed this inequality in abdominal aneurism, where there was no positive sign of thoracic pressure upon the sympathetic, also in peritonitis; and the other interesting fact is, that where there has been repeated attacks of colic, or where there has only been one severe attack, and sphygmographic tracings have been taken of the radial pulses, there will nearly always be found higher arterial tension in one pulse than in the other. Admitting that even in health there is a degree of difference in the fulness and tension of the radial pulses, this difference in lead colic is so pronounced as to stand out prominently as something peculiar to the illness, and it differs from the healthy tracings, in so far as it is not always the pulse of the limb that is most used that shows the higher tension. Like the inequality of the pupils, it stands in some way related to primary disturbance of abdominal sympathetic fibres, and through them of the vasomotor centre. In a female inmate of the Newcastle Workhouse, Dr Bowlan noticed, not only the difference in the radial pulses already alluded to, but he found that the left hand was always in a state of profuse perspiration.

Eliz. H. (Dr Bowlan's case and series of tracings.) Lead optic atrophy. Right radial. 1st August 1890.

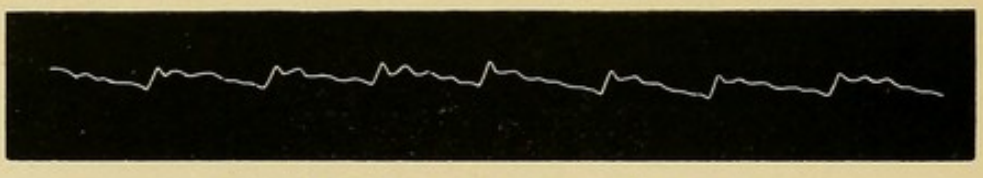


Eliz. H. Left radial pulse; has profuse palmar perspiration left hand; shows lower arterial tension. 1st August 1890.



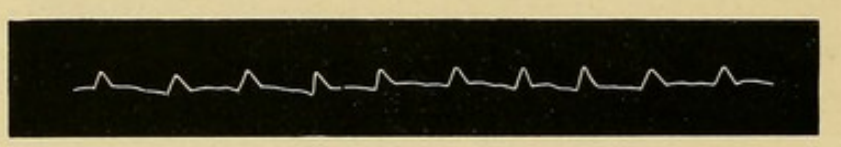
Eliz. H. Right radial. 22nd August 1890.

Eliz. H. Left radial. 22nd August 1890.

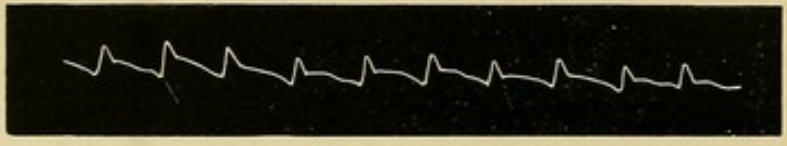


Eliz. H. Right radial. 6th Oct. 1890.

Eliz. H. Left radial. 6th Oct. 1890.



Mary C., age 17. Is experiencing her first attack of lead colic ; right radial ; shows difference between the two radial pulses noticed in colic. 21st November 1890.



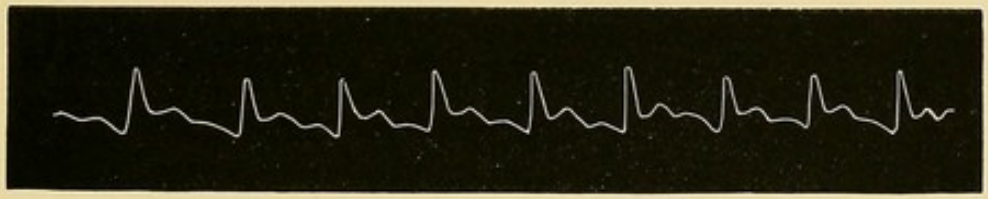
Mary C. Left radial. 21st Nov. 1890.

Wm. M'F., a paint mixer. Severe colic ; left radial. 29th Dec. 1890.

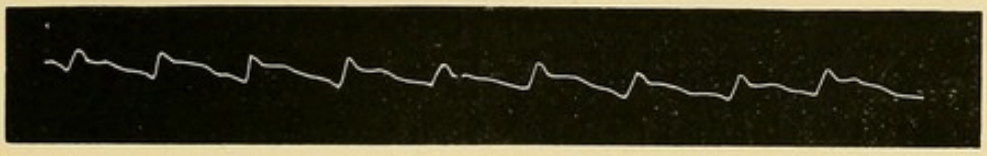
Wm. M'F. Right radial. 29th Dec. 1890.

Eliz. S., age 44. 11th Dec. 1890. Has only worked six weeks ; colic ; right radial.

Eliz. S. 11th Dec. 1890. Left radial ; shows difference in radial pulses in attack of colic.



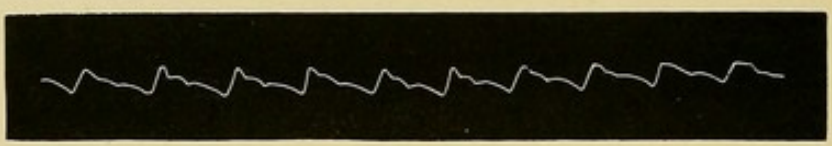
Charles B., age 34. ; ship painter. Right radial. Colicky pain on left abd. only. 20th Dec. 1890.



Chas. B. Left radial ; pulse of higher tension on same side as abdominal pain.

Thos. S., age 48 ; plumber. Lead colic ; has mitral systolic murmur ; right radial. 17th Dec. 1890.

Thos. S. Left radial ; shows pulse to be of higher tension than opposite radial. 17th Dec. 1890.



Dorothy R., age 39. Tracing taken at factory. Right radial ; has never had colic ; shows equality of pulses. 24th Dec. 1890.

Dorothy R. Left radial. 24th Dec. 1890.

A very early symptom noticed in lead poisoning, is *alteration of the menstrual function ;* the menses become either excessive or deficient. A very large number of female lead workers suffer from menorrhagia,—even young girls of from seventeen to twenty-four. I find nearly 50 per cent. of those engaged at the factories, and whilst regarding this as a condition which furthers the development of the anæmia so often present, it is not the cause, for the cachexia affects men equally with women. Other women again suffer from scanty menstruation. Lead disturbs the utero-ovarian function, and the earlier this occurs, the more likely is the individual to suffer from plumbism. Lead workers miscarry in a much larger per centage than other women,

and the children generally die in convulsions. Dr Barnes[1] alludes to the subject of abortion and lead poisoning. He quotes the experience of M. Paul as to the fifteen pregnancies of four women who had worked in a type-foundry: ten of these ended in abortion, two in premature labour, one in still birth, and one child died within twenty-four hours. In a second series, five women had borne nine children before working at lead, without one abortion. After exposure to lead, there was a total of thirty-six pregnancies; of these, twenty-six ended in abortion, one in premature labour, two in still births, and five children died, four in the first year. A woman had five pregnancies, all ending in abortion; she left the factory, and bore a living child. Another having ceased work at the factory, had two children; she returned, had two abortions. This is also my experience. In one of my cases, E. C., aged twenty-seven, there was first a living child, then one miscarriage. She left the lead works and went into the country; there, her second child was born; she returned to the lead works, and had two miscarriages. M. W., aged thirty-nine, lead worker for eighteen years, has had twelve children, of whom four are now alive. The remaining eight died at ages varying from five days, to four, six, and fourteen months, in convulsions. She suckled her children whilst following her employment at the lead works. She has had in addition five miscarriages, three of these were in succession. There is no specific history, no albuminuria, but she is anæmic. In another case, Mary A., aged forty, whose mother, too, had been a lead worker, we have a history of eight children, all of whom died of convulsions. Mary C., aged twenty-four, was healthy until four months ago, when she became a lead worker. She is married, and until a few months ago lived in Scotland, on a farm, where she had one child. She entered the lead works four months previous to my seeing her, and she aborted in the third month of pregnancy. Mary Ann T., aged twenty-five, had one living child at full

[1] Obstetric Medicine and Surgery, page 496, vol. i.

term, eight years ago; she then became a lead worker, since which she has had two miscarriages.

The pernicious influence of lead affects equally the reproductive organs of the male, and through the spermatozoa causes abortion; or if a child is born, it is ill nourished, and dies almost immediately after its birth, generally from convulsions. I have been on the outlook for such a case, and a few months ago was fortunate enough in seeing, with Dr Yoakley of Heaton, the child of parents who were lead workers. The mother was aged twenty. She had been for four years a lead worker. During this time she had been off work twice on account of colic, constipation, headache, and vomiting. For a third time she was invalided, but on account of severe uterine hæmorrhage, prior to which she had noticed that her menses were too profuse. Since her marriage, she has had one miscarriage, and during the first four months of her last pregnancy, she followed her occupation as a white lead worker. No hæmorrhage occurred during this last pregnancy, but she had a good deal of sickness. The confinement was natural, in no way precipitate, and there was no excessive hæmorrhage at the time. Her husband is tall, very pale, aged twenty-seven. He has been for the last two years a white lead grinder, and has had two attacks of colic; his gums show a deep blue line; his wife's gums are ulcerated at the margin, but do not show a blue line. I saw the child of these people three weeks after its birth; in size and weight it resembled a six months fœtus, although born at full time; its finger nails were perfect. It was a tiny object, and had a weak, feeble cry, but so far as I could make out, all its internal organs were healthy. The child lived another fortnight; it was unable to assimilate food. During the last few days of its life, it was the subject of diarrhœa. I made a *post-mortem* examination of this child eighteen hours after its death, and was much struck by a circumstance which I have noticed in animals that die of lead poisoning, namely, the rapidity with which decomposition sets in. This was all the more marked from the fact, that the

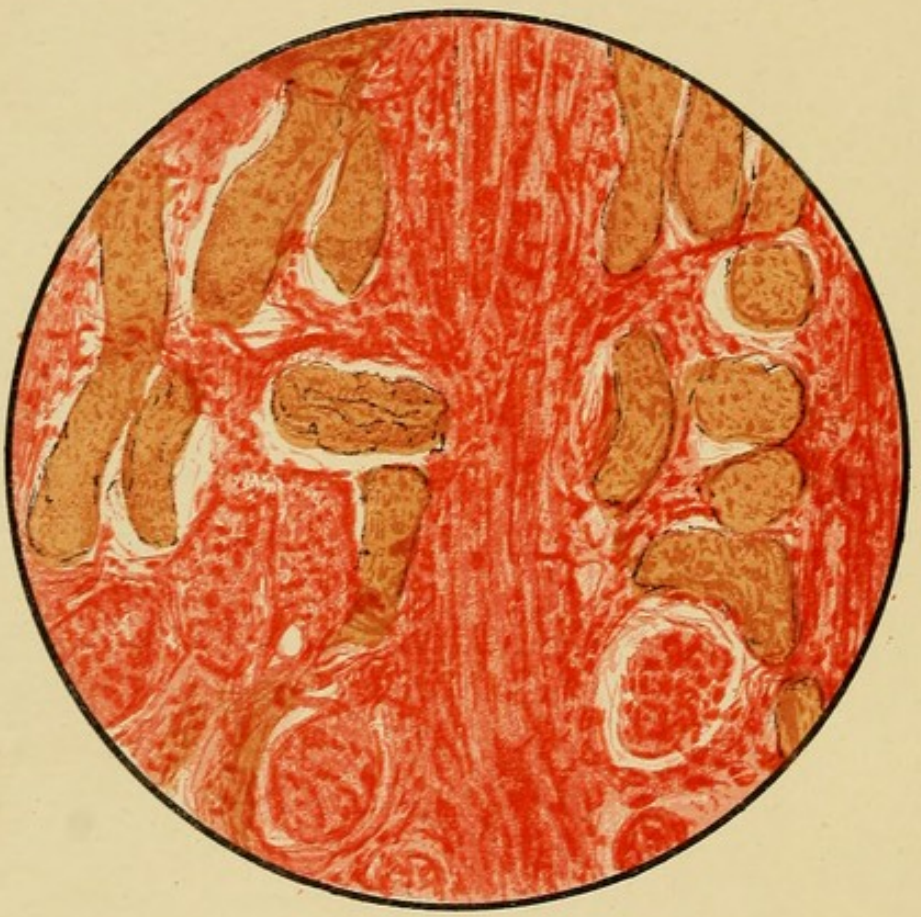


FIGURE X.

FIG. 10.—Kidney showing increase of interstitial tissue and atrophy of secreting structures. Case of E. T., died five weeks after birth; both parents are lead workers, and have suffered from plumbism.

FIGURE VIII.

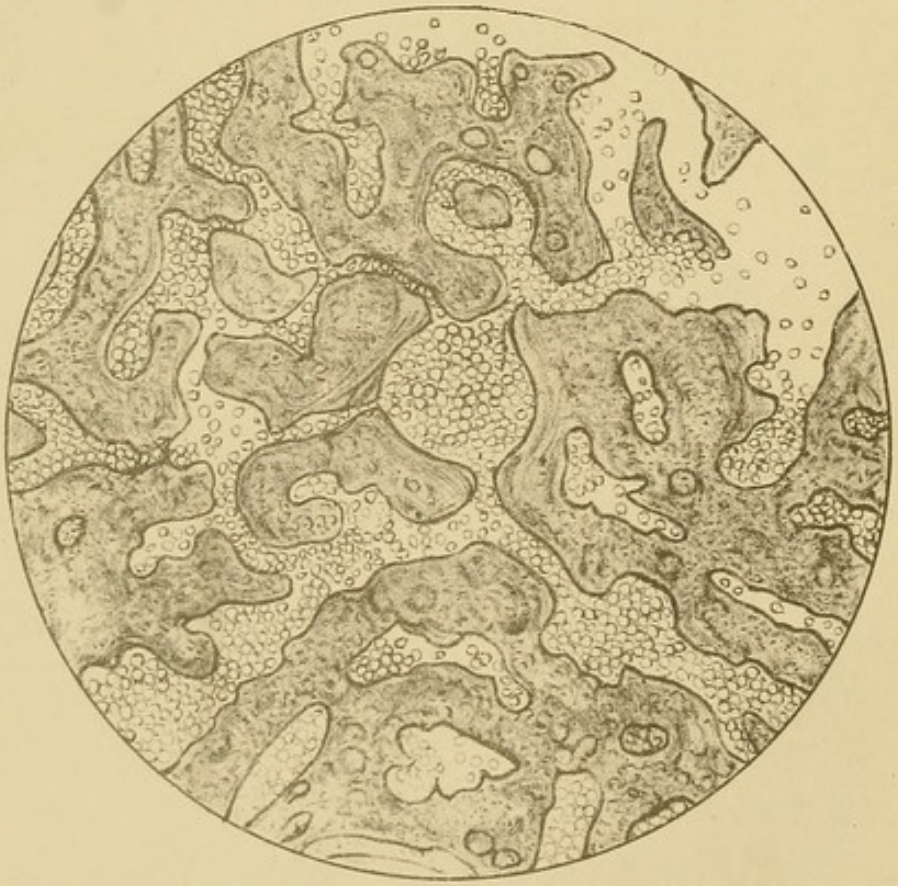


FIGURE IX.

FIG. 8.—Liver of lead worker's child E. T. Cirrhotic. Shows marked increase, of fibro-connective tissue around portal vessels; also shrunken hepatic cells, giving appearance of increased size of blood spaces between the cells. (Crouch, one-sixth inch.)

FIG. 9.—Liver, lead worker's child, E. T. Shows altered hepatic cells and very large spaces filled with blood. (Crouch, one-sixth inch.)

death took place in November, the weather being then extremely cold. The internal organs of this infant resembled in size those that are taken from an ordinary wild rabbit—the liver, kidneys, and heart, were not any larger than what are found in one of these animals. Under the microscope, the liver presents the following appearances. The liver cells are atrophied, and are irregular. In those parts of the section where the blood has remained, the capillaries are seen to occupy a much greater share of the space of the lobule than they ought to. The interlobular tissue is increased, absolutely as well as relatively. Delicate cells are observed lying on and around the capillaries. In a word, we have in this liver taken from an infant whose parents were both lead workers, an exaggeration of the capillary spaces between the hepatic cells, atrophy of liver cells, and an intercellular cirrhosis. In the kidney, we found thickening of the capsule, a peculiar atrophied condition of the secreting portion of the kidney, the Malpighian bodies shrunken and extremely irregular, convoluted tubules also shrunken, epithelium granular. Briefly, there is distinct atrophy of the cortex of nearly every element, except the connective tissue, and running down from capsule into cortex, there are strands of connective tissue; the epithelium is granular throughout.

The nervous system suffers severely in lead poisioning; ill-defined pains and cramp-like sensations in the limbs, particularly in the calves of the legs, and dull aching pains in the joints, which are regarded as rheumatic, are complained of. The muscles are tender to the grasp; there is a sensation of tingling or numbness in the fingers, and soon afterwards certain muscles become suddenly or slowly paralysed and undergo atrophy, or cerebral symptoms supervene, including convulsions and coma. Boerhaave was amongst the first to associate paralysis with Saturnine poisoning, and following him Slockhusen, Van Swieten, and de Haen; but it is to Tanquerel des Planches, and to Duchenne de Boulogne, that we turn for the earliest and most complete clinical description of paralysis in lead poisoning.

Duchenne was the first to utilise electricity in the investigation of this form of paralysis, a method which had already furnished him with wonderful results in his researches upon infantile spinal paralysis and progressive muscular atrophy. To Duchenne also belongs the honour of having localised the paralysis, and of describing the order in which the various muscles are affected. He observed the immunity of certain muscles, particularly the supinator longus. Regarded in the light of a peripheral lesion by Duchenne, Vulpian and Raymond reported cases of lead poisoning in which the paralysis was general, and thus they threw upon Duchenne the responsibility of establishing a differential diagnosis between it and sub-acute general anterior poliomyelitis.

As a matter of experience, we may say that paralysis, as a sign of lead poisoning, is met with almost always in the chronic, seldom in the acute form. It occupies in Tanquerel's collection of cases the third place in the order of succession of the symptoms met with in plumbism. Before paralysis arises, there has generally been experienced one or more attacks of colic, headache, vomiting, or the patient has had lead encephalopathy, but in some of my cases paralysis of the extensor muscles of the arms occurred without the patient ever having suffered from colic. Gowers[1] divides the local muscular paralysis with wasting, met with in lead poisoning, into two classes, one, the more frequent, where the loss of power precedes wasting. The muscles, whilst presenting "the degenerative reaction," namely, loss of Faradac irritability, still preserve their voltaic irritability. The common seat of this form is the extensor muscles of the wrist and fingers. Wrist-drop is the consequence of it. In the other form of Saturnine atrophic paralysis, the weakness and wasting come on simultaneously and proceed *pari passu*. Faradic and voltaic irritability are both lessened in proportion to the wasting, just as in progressive muscular atrophy. This form may affect any part, but is especially common in the small muscles of the hand.

[1] "Diseases of the Nervous System," vol. ii., page 870.

We are all familiar with the classical form of paralysis met with in lead poisoning, the ordinary double "wrist-drop" —double, for it is generally symmetrical, although as a rule it is more pronounced on one side than the other. It also develops on one side earlier than the other. The patient at first complains of weakness in the hand—the right hand as a rule—and it

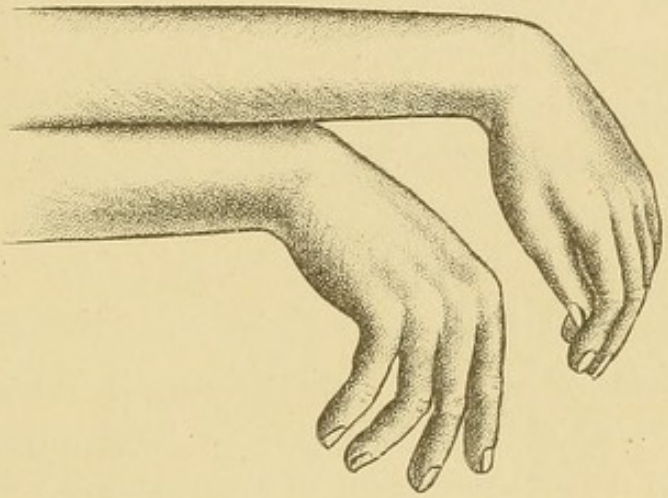


Fig. 11. Lead poisoning, double wrist-drop, shows attitude of hands.

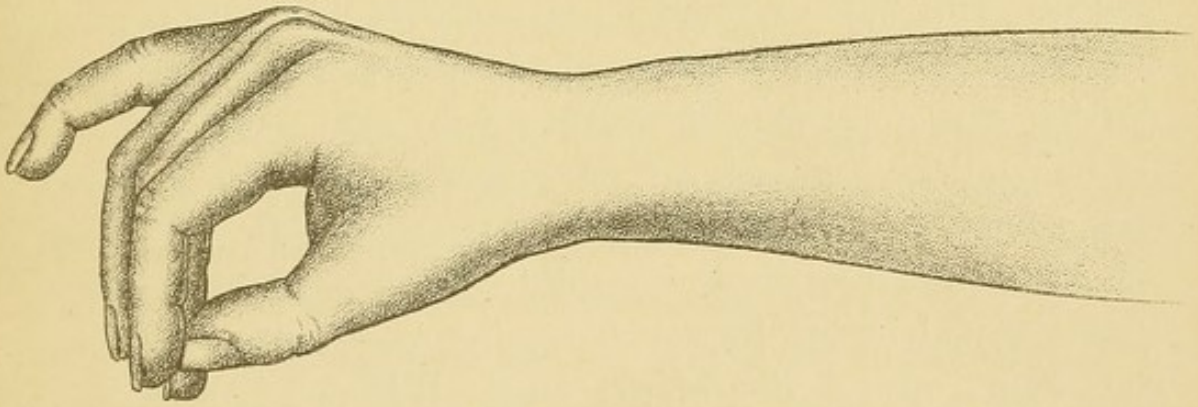


Fig. 12. Lead poisoning in F. G., age 33, a house painter, paralysis of extensors of the fingers; the little finger has however escaped (Dr Philipson's case).

is noticed that his grasp is not so firm as it used to be. The disease may proceed no further. Generally the weakness becomes more intense; suddenly, or it may be gradually, it deepens into a paralysis, which affects principally the extensors of the fingers, usually those of the middle fingers. The patient is unable to extend the fingers in question at the metacarpo-phalangeal joints.

At this stage, extension of the index and of the little finger is still possible, owing to the special extensor muscles of these fingers having thus far escaped, and beyond this the paralysis may not proceed. The tendency, ho ever, is for it to spread so as to involve these special muscles, and thus is developed a paralytic condition of the extensors of the basal phalanges of the four outer fingers. By degrees it is observed, that the phalanges of the thumb cannot be extended, the patient retains, however, the power of abducting the metacarpal bone. With a preference for the ulnar rather than the radial side of the hand, this loss of power gradually involves the extensors of the wrist, inducing wrist-drop, in which the hand hangs more or less powerless from the forearm, forming a right angle with it, the fingers slightly bent, and the thumb generally drawn towards the palm of the hand. The patient is now unable to raise his hand, but in certain positions, as for example, when he rests his arm on his elbow, or extends the elbow, he is then frequently able to raise the hand that has dropped and afterwards extend the fingers. This action is due to, and explained chiefly by, the fact that the supinator longus has escaped. Previous to the complete development of the wrist-drop, which is so characteristic of lead poisoning, there has been a feebleness of the grasp of the hand accompanied by increased flexion of the wrist. In wrist-drop, the flexors also seem feeble, but this is a weakness more apparent than real, and is due to the shortening of the flexor muscles consequent upon flexion of the wrist. It is owing to the fact that power is still retained by the flexors, that patients are able to execute a certain number of movements, such as seizing objects. To accomplish this, a certain attitude has to be assumed. The right hand is placed beside the object, and by means of the fingers of the left hand, those of the right are passively extended, and then, with rapid flexion of the fingers of the right hand, the object is seized. Even these limited movements, however, are not accomplished without great fatigue of the flexors ; if repeated too often, and the attention of the patient is not concentrated

upon the act, the object falls from his fingers. The muscles which in this form of lead paralysis are involved, are supplied by the musculo-spiral nerve, and are the common extensors of the fingers, the extensor indicis, those of the phalanges of the thumb, and the extensors of the wrist; and, yet, whilst in these muscles the paralysis may be complete, the supinator longus, which is supplied by the same nerve, escapes, as does also the extensor of the metacarpal of the thumb. The forearm presents an appearance of flattening on its posterior aspect, in which the outline of the supinator longus may be detected. The nerve is observed to have undergone degeneration; it has lost its irritability. The muscles still retain, even in an exaggerated form, their voltaic irritability, yet by degrees they lose it as they lost their Faradic. This, the commonest form of paralysis, is the antibrachial type described by Remak, and, as an illustration, I give you the following:—

Mary A., aged 39, a widow, was admitted into the Newcastle-upon-Tyne Infirmary, under my care, complaining of loss of power in both wrists. She has been a lead worker for twenty years, and only once, seventeen years ago, has she had colic. She has had eight children, all of whom died in the earliest days of infancy in convulsions. A month previous to her admission into the infirmary, she experienced a sensation of pins and needles in the palms of both hands, accompanied by pain in the muscles of the thumb running up into the shoulder, slight sickness, headache, and constipation. One morning whilst combing her hair, she suddenly lost the power in her wrists, a weakness which has increased. Her pupils are unequal; the left is the more dilated; both react to light and accommodation. There is neither anæsthesia nor hyperæsthesia. The left knee-jerk is the more exaggerated. There is complete paralysis of the extensor muscles of both wrists—absolute "wrist-drop" in fact. The extensor muscles scarcely exhibit any reaction to either kind of electrical stimulation. A few weeks afterwards this patient lost the power in the abductor muscles of the thumbs.

Whilst the poison has a special predilection for the muscles already described, these may escape, and the muscles of the upper arm may be involved—those which constitute the Duchenne-Erb group, namely, the deltoid, biceps, brachialis anticus, and the supinator longus, and to these is generally superadded, paralysis of the supra and infra spinatus. This form of paralysis, usually bilateral, occurs in the more inveterate types of lead poisoning. Of the muscles enumerated, the deltoid is generally the first to become affected, and in not a few cases, it is the only muscle of the Duchenne-Erb group that is paralysed,—as in a case reported by Buzzard. In other instances, as in the cases reported by Gaucher,[1] and also by Pidra,[2] the supinator longus is the only muscle of the group affected. When all the muscles of the Duchenne-Erb group are paralysed, the patient presents these appearances—the arm hangs powerless, the humerus is rotated inwards, and the forearm is semi-prone. The patient is unable to raise his arm, owing to paralysis of the deltoid, nor can he bend his forearm upon the arm, owing to paralysis of the biceps, and yet extension of the forearm is easy, for the triceps remains free in most cases. In this type the disturbances of electrical contractility and atrophy are much less pronounced than in the more classical. It is rare, according to Dejerine-Klumpke,[3] that there is noticed a complete abolition of the Faradic or voltaic irritability of the muscles. In other cases only the small muscles of the hand are affected, the thenar and hypothenar prominences, and the interossei. The localised nature of the paralysis, and the external aspect of the hands, recall the appearance presented by progressive muscular atrophy. It is distinguished therefrom by its electrical reactions, and by the fact that the atrophy accompanies muscular paralysis. The atrophy is always very pronounced, and is almost contemporaneous with the paralysis. Gowers, whilst regarding the wasting as slow, also says it accompanies, rather than succeeds, the loss of

[1] France médicale, 1882, vol. ii., page 244. [2] Thésis de la paralysie saturnine.
[3] Des polynévrites, p. 74.

power. Fibrillary twitchings are frequent, just as in progressive muscular atrophy. This may be the primary mischief in lead poisoning, and it is usually a very obstinate form of paralysis. It may precede or accompany that form of paralysis in which the common extensors are affected. Occasionally certain muscles of the legs are paralysed; this occurs, according to Tanquerel, in 13 per cent. of all cases of lead paralysis as a complication of the other forms. If it is part of a general paralysis, the muscles which emerge from the pelvis, for example the psoas, may be affected. The paralysis under these conditions is slight, and lasts no length of time. In the leg, as in the arm, there is the same predilection for certain muscles—those that are homologous with the muscles of the forearm, particularly the peroneal muscles, and the long extensors of the toes, which are supplied by the peroneal nerve,—the tibialis anticus, supplied by the same nerve, escaping. I have only met with this peroneal paralysis in cases where there was wrist drop; it is generally preceded by such prodromata as pains in the legs, a sense of numbness or tingling, and there is occasionally associated with it a degree of hyperæsthesia. The patient complains of feeling weak in his limbs, and walks on the outer border of his feet, feeling a difficulty mostly when going up or coming down stairs. It is owing to the peroneal paralysis, and to the fact that the common extensors of the toes, and the special extensors of the great toe, are also paralysed, that walking and descent of a stair are rendered difficult, for the tibialis anticus has now to bear the weight of the body during the descent. The electrical reactions of the muscles are altered, but the atrophy is not very conspicuous. The tibialis anticus plays a similar part to the supinator longus: whilst, as a rule, escaping, it too may suffer. When the tibialis anticus is paralysed, it is generally associated with a similar state of a special group of muscles,—the triceps and the muscles of the tendo Achillis,—as in the unique case of Remak and Oppenheim, where the peroneal and extensor muscles of the toes were un-

affected, a condition which, whilst rare in Saturnine paralysis, is frequent in anterior poliomyelitis, for example, infantile spinal paralysis.

Tanquerel alludes to laryngeal paralysis, and Sir Morell Mackenzie points to lead as a cause of paralysis of the adductors of the vocal cords.

There is a form of generalised paralysis of rapid development occasionally met with in lead poisoning. The muscles are paralysed in their whole length, and *en bloc;* the muscles of any region, sometimes within a day, becoming absolutely powerless. Those who are the subjects of other forms of paralysis may thus suffer, and in them the malady may invade rapidly, and in succession, muscles that have hitherto escaped. Sometimes, indeed, a generalised paralysis is induced, either in an ascending or descending form, which extends rapidly day by day, always invading the whole length of the muscles of the limbs, the trunk, abdomen, and thorax. The patient occupies the dorsal decubitus, unable to move a limb, and is even incapable of eating; the intercostals, the diaphragm and muscles of the larynx are involved. The patient suffers from dyspnœa and loss of voice. The noteworthy feature in these cases is, that the muscles in the head and neck are respected. Rapid as is the development of this form of paralysis, it just as rapidly begins to amend, and it is exceptional for death to come about by asphyxia, as occurs now and then in acute ascending paralysis. Whilst recovery is the rule, death has occurred by respiratory paralysis, as in the case reported by Strauss and Heugas.[1]

The following case, which was under the care of my colleague Professor Philipson, is an illustration of this type of paralysis. J. F. A., aged twenty-nine, single, a white lead worker, was admitted October 30th, 1884, complaining of symptoms of lead poisoning of seven weeks' duration. Patient has followed his occupation for the last three and a half years, has been engaged

[1] Heugas, "Contributions à l'étude de la paralysie saturnine," page 54.

principally in the drying department. This work consists of drying powdered white lead in an oven. His face and hands were continually covered with the powder, and the lead dust produced a sweetish taste in his mouth. At the end of a year and a half, he had lost all colour; he became pale and sallow, his appetite failed. He took his meals in the works but always washed his face and hands before eating. It was not until the end of two and a half years that he experienced his first attack of lead poisoning, namely, colic and constipation. After fourteen days, he returned to work quite well, but on working for three months he had a second attack of colic, from which he recovered in five weeks. Subsequent to this second attack of colic, patient drank large quantities of beer, and water acidulated with sulphuric acid, and was extremely careful as to cleanliness. Seven weeks before admission, patient went to work in his usual health, and whilst in the factory, was suddenly seized with griping pain in the umbilical region. In a short time, it became so severe that he had to sit down and press his hands into the abdomen for ease. He was conveyed home. Three days subsequently the patient had epileptiform convulsions, he became unconscious, and remained thus for twelve days. On regaining sensibility, the bowels were opened for the first time since the illness, and the paroxysmal pains decreased in severity, ultimately ceasing on the following day, that is, sixteen days after the commencement of the attack. Paralysis now suddenly appeared, attacking first the right leg. At six o'clock in the morning, patient was out of bed, and could walk unsupported, but on rising five hours later, he was surprised to find that his limbs gave way under him, they helplessly crossed, and he fell to the ground. The right leg was most affected, but before morning right and left were equally paralysed. The next day the arms became paralysed with the same suddenness, the right again being the worse. Other muscles of the body were similarly affected, but the patient was unable to state the order in which they were attacked. He never suffered pain in the limbs or trunk. On admission, the

temperature, pulse, and respirations were found to be normal; face was pale; well marked blue line along the gums. Patient lay in bed quite powerless. The only movement he could make with freedom, was elevation of the head from the pillow, and its movement from side to side. The pupils were widely dilated, but reacted slightly to light. Both discs were swollen, red, very irregular around the margin, with small hæmorrhages at places. The veins were large and full, and the retina surrounding the disc looked cloudy, in places obscuring the vessels; a few hæmorrhages were seen in it.

On examining the muscles, the deltoid, triceps and extensors of the wrists and fingers in both arms were found to be completely paralysed. The other muscles, with the exception of the long supinator, were much weakened, almost amounting to paralysis. In the legs and thighs, all the muscles, both flexors and extensors, were powerless. The muscles of the back, with the exception of the trapezius, were also paralysed, but those of the abdomen, chest, head, neck, face, and eyes, appeared to be unaffected. On lifting the patient into the sitting position, and asking him to abduct the arms, the deltoids remained perfectly flaccid. The trapezius and muscles of the neck stood out boldly, the shoulders were elevated, but the arms hung powerless, as seen in the accompanying diagram. When the arms were held out horizontally, the usual drop wrist deformity was present; the feet were extended at the ankles, and all tendon-jerks and reflexes in the extremities were absent, abdominal reflex slightly marked, organic reflexes normal. The limbs were perfectly flaccid, all the muscles were soft and flabby, and the paralysed muscles were very much wasted. Reaction of degeneration was present in the paralysed muscles, and the weakened muscles shared in the partial degenerative reaction. Sensation was normal. No pain was complained of; his memory was bad, and his speech slow. His blood corpuscles numbered 3,950,000 per cmm.

Beyond having had gonorrhœa eleven years ago, and syphilis

FIG. 13.—Lead poisoning. Extensive paralysis; muscles of arms, shoulders, back, and legs affected. (Prof. Philipson's case.)

FIG. 14.—Lead poisoning. Extensive generalised paralysis; muscles of arms, shoulders, back and legs affected. Prof. Philipson's case. Patient recovered.

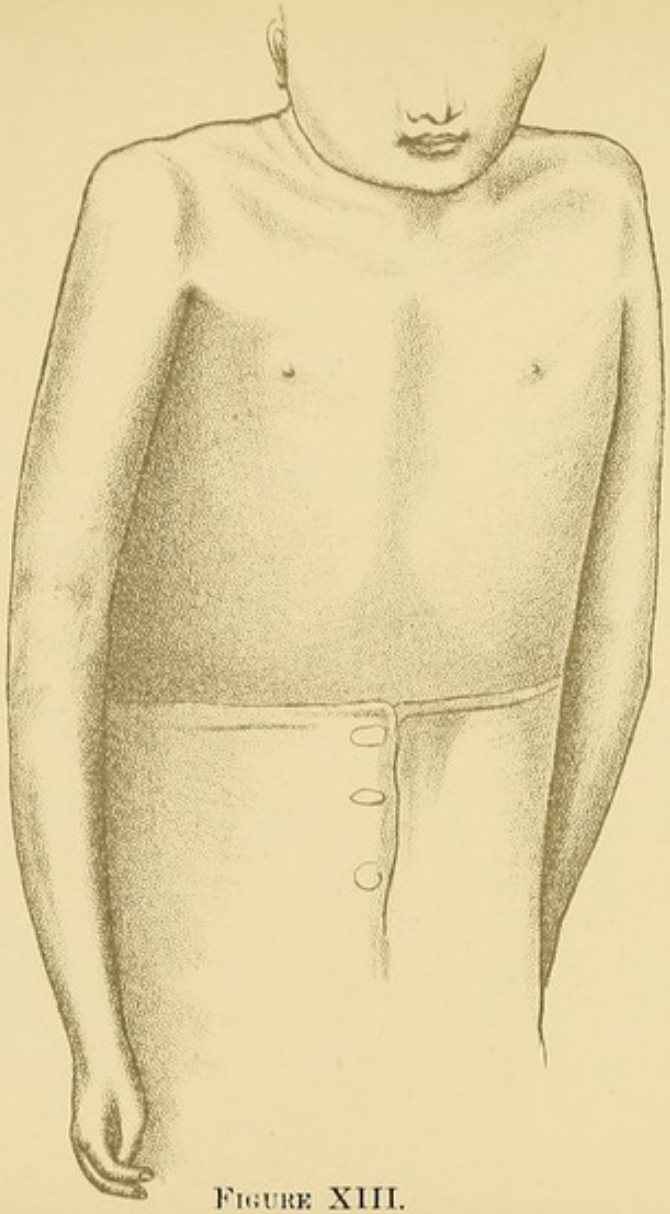


FIGURE XIII.

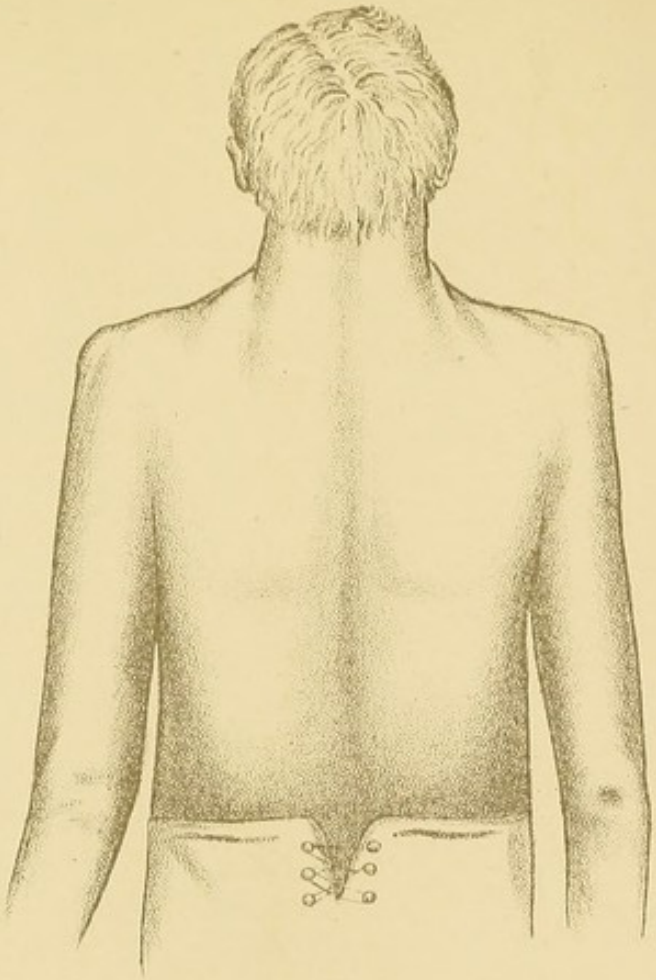


FIGURE XIV.

two years later, and a history of taking alcohol somewhat to excess, there is nothing special to remark, except that a cousin of his, a lead worker, has also suffered twice from plumbism. Patient steadily improved under treatment, and when discharged, three months subsequently to his admission, power had returned to all the muscles, excepting the extensors of the wrists and fingers. Dr Baigent saw the patient five years after the onset of the illness, and found at that time all the muscles strong and well developed, and the sight good.

Minor degrees of this general form of paralysis are also met with. Sir Dyce Duckworth[1] reports the case of a woman, who, after using a cosmetic for five years, exhibited the anæmia and blue line of lead poisoning, and suffered from paralysis of arms and legs; she also had subcutaneous nodules and gout. The muscles of the upper parts of arms and scapulæ, but particularly the deltoids, were affected; in the legs, the muscles were much wasted and flabby, and were the seat of tremors.

Sensory symptoms. Pain frequently precedes the various forms of paralysis, but seldom continues after the muscles are affected. In my notes, I find it a very frequent symptom preceding paralysis of the legs. Sensation as a rule is unaffected, but in a very large number of cases, patches of analgesia and anæsthesia[2] are met with, over the inner and posterior parts of the forearm in wrist-drop, and are also met with on the leg. A few authors are inclined to regard these as illustrations of hysterical anæsthesia, but they differ very distinctly from it, if in nothing else, in the very marked readiness with which the skin bleeds when pricked. In a girl, aged twenty-one, the analgesia was very marked over the extensor surface of the right forearm, wrist, and hand; also over the flexor surfaces of the same parts, and extended to within a few inches of the elbow. There was no analgesia of the arm; the prick of a pin over the right arm was followed by pretty

[1] "Clinical Society's Transactions," vol. xx., p. 266.

[2] Oliver, *Brit. Med. Journ.*, 1885, vol. ii., p. 732.

profuse bleeding. Guinon[1] draws attention to two types of Saturnine anæsthesia: one where it is limited to those parts, generally the fingers, which have been brought into direct contact with the lead; and the other, where there is a larger area of anæsthesia irregularly distributed over the whole of the cutaneous surface, or limited to one half of the body under the form of hemianæsthesia. Hyperæsthesia, amounting to actual pain, has been noticed by Baigent,[2] and described by him in his thesis on peripheral neuritis.

Tremor is a frequent symptom of Saturnine poisoning in a large number of people, who are not suffering from any form of paralysis, for example, in lead workers, who are still following their occupation. I have observed, in seven or eight per cent. well-marked fibrillary twitching of the naso-labial muscles and of the tongue. The tremors strongly resemble those noticed in general paralysis.

In one of my cases, a male, kindly sent to me by Dr Beatley, there is a peculiar shaking of the left arm and leg. The man has worked for twenty-eight years in a pottery; his duty being to dip the jars into a solution of lead, and thereafter to place them in the oven. He admits that he has drunk beer very freely, and states that until two years ago his health was good. The tremor is very pronounced. It begins in the left thumb; gradually extending up the arm, the limb is thrown into a state of violent agitation, over which patient has no control. It is not a painful shaking, nor does it altogether prevent him following his occupation, although he says it is very detrimental. Within the last six months, the tremor has extended to the left leg, but here it is not so bad as in the arm. Strange, too, the shaking never begins in the two limbs at the same time, never in the leg first. It is always conveyed from the arm to the leg. There is no marked paralysis. The grasp of the left hand is feebler than the right. The shaking of the arm can be stopped by

[1] "Les agents provocateurs de l'Hystérie," page 137.

[2] Baigent, Peripheral Neuritis; Thesis, M.D., Durham.

forcibly extending the thumb. Knee-jerk and ankle clonus are absent, but foot-trepidation can be induced by causing patient to bear his weight upon his foot. There is a well marked blue line on gums; urine is not albuminous.

Of the rarer forms of lead paralysis, I would mention paralysis of the third nerve, as in a case successfully treated and reported by Buzzard in *Brain*, 1890, where there was ptosis of right eyelid, with abolition of movement of the upper, lower and internal recti; the pupil was dilated and did not react to light. In many of my own patients, there has been diplopia—diplopia without any apparent muscular defect; in others, slight internal strabismus, and in two cases very marked nystagmus in the acute stage of neuro-retinitis.

In a small percentage of cases of plumbism, there are disturbances of bladder function. A few women who have come under my observation, have not been able to retain their urine; they have, in the absence of any local cause, suffered from incontinence; others are unable to empty the bladder. Mr Hurry Fenwick has reported to me almost a similar case of atony of the bladder, occurring in a man who was a painter, and in whom there was residual urine to the extent of six ounces. There was no blue line on the gums, nor was any form of paralysis detected. Without positively asserting that this was lead paralysis, the fact is of some importance, taken in conjunction with my own observations and the trade of the patient.

How far are we to regard the various forms of paralysis in lead poisoning, as due to a peripheral or a central cause? We have seen that the characteristic features of lead paralysis are, that it attacks certain groups of muscles which are functionally related to each other,—that the nerve loses its response to both kinds of electrical stimulation, whilst the muscles retain their voltaic or galvanic irritability, having lost their Faradic. Any theory, therefore, proposed to account for these characteristics, must be a comprehensive theory. Is lead paralysis primarily a nervous or a muscular disorder? Changes are, undoubtedly,

detected both in muscle and nerve. In which of these did the change first begin? Charcot, Leyden, and Westphal support the neuropathic theory, whilst the myopaths, amongst whom may be mentioned Gusserow, Henle, and Hitzig, maintain that the affected muscles are the primary seat of the alteration. The predominating idea in Gusserow's mind was, that in whatever organ the largest quantity of lead was found *post mortem*, that organ must have had the greatest affinity for lead; and on the basis of this observation, Gusserow arrived at the conclusion that the muscles, bones, liver, and spleen contained the greatest quantity of lead, whilst the brain and spinal marrow contained the least. To him, therefore, lead paralysis depended upon a diseased state of the muscle substance. Admitting for the moment that most lead is found in muscle, are we at liberty to reckon upon absolute values, or are relative values only, to be conclusive? Equal weights of tissue only can be compared. Friedländer also pleads for the myopathic theory on the ground that in one case of lead poisoning which lasted over four years, the spinal cord was healthy, whilst the substance, not only of the extensor muscles, but of all the muscles of the body, was affected. Influenced by the results of Harnack's experiments on rabbits and frogs, Friedländer concluded that lead causes primarily a functional disturbance of muscle, which is soon followed by a failure of nutrition: this is succeeded by exuberant growth of connective tissue, which reduces the size of the neuro-muscular fibres, and is followed by a degeneration of the nerve. Paralysis would thus ensue, possessing all the characteristics of the peripheral variety, and close upon paralysis would supervene atrophy. Is the pathological connection between muscle and nerve so close? Remak[1] has questioned the validity of Harnack's opinions drawn from the injection of ethylate of lead into rabbits, in which an acute and generalised paralysis had been developed. After all, this acute Saturnine paralysis can scarcely contrast with the partial and more chronic form that

[1] "Archiv für Psychiatrie," vol. ix., part 3, page 510.

occurs in man, and in nearly every instance affects the extensor muscles. The predilection of these muscles for lead is, after all, only relative. How are we to dispose of the careful examinations made by Bernhardt, who showed conclusively that the flexors and the supinator longus, which usually escape, contained exactly the same quantity of lead as the extensors, the amount in each instance being very small. The myopathic theory, however, need no longer detain us, for Heubel has also demonstrated that, with equal weights of material, the muscles are, of all the tissues of the body, those which contain the smallest quantity of lead in saturnine poisoning. Recent opinion has veered partly in favour of the neuropathic theory, which assumes, that in lead paralysis the intra-muscular terminations of nerves become affected, and this, as the primary lesion, is followed by an ascending atrophy of the nerve trunk. The nerve trunk is composed of both sensory and motor fibres. But why should motor fibres, which are descending nerves, suffer from an ascending lesion, whilst the sensory nearly always escape? This part of the neuropathic theory, which claims the intra-muscular terminations of nerves as the primary seat of the lesion, must share the same fate as that which regarded the muscle substance itself as the first to be affected.

If, therefore, peripheral conditions do not adequately explain lead paralysis, does a study of the central nervous system throw any light upon it? Oeller, Romberg, and Erb, claim the spinal cord as the primary seat of the lesion in lead poisoning, and in many respects, it is difficult to distinguish lead paralysis from subacute anterior poliomyelitis. There are two things which we must remember, both of which were forcibly driven home to me by a study of cases of acute infantile spinal paralysis, made by my colleague, Dr Drummond. I employ these to support my argument, and they are these:—In the early stages of lead paralysis, the pathological changes must be slight; time has not yet been given for destructive processes to be developed. And the second point is, that very many victims of lead paralysis

recover, particularly those of the generalised type. Many writers state that no pathological change is detected in the spinal cord. That is true; but when we know that functionally related groups of muscles are paralysed, and atrophied, and that, in the cord, only a limited area is allotted to these groups, it is an easy matter for the investigator not to discover precisely that part of the spinal marrow where the lesion is located. In a case reported by Monakow[1] (that of a painter, aged 56, who had suffered from chronic lead poisoning for thirty-five years, during ten of which he had paralysis of the extensors of the right side, and for some months before death, a right-sided hemiplegia, due to cerebral hæmorrhage), there was found in that part of the spinal cord, which extends from the sixth to the eighth cervical nerves, very distinct atrophy of the central ganglion cells of the anterior cornua, and sclerosis of the posterior commissure. Oeller,[2] in his monograph, gives, at considerable length, the details of a case of lead poisoning in a man, aged 43, who had worked amongst white lead for only fifteen months. Shortly after exposure to lead, he suffered from colic. In nine months, the extensor muscles of the left hand were paralysed, but this quickly and completely disappeared. Three months afterwards, he was found with total and complete paralysis of the extensors, first on the left side, and then on the right. As the disease progressed, œdema of the feet appeared, and patient became anasarcous; the urine was albuminous. Death supervened during an attack of severe dyspnœa. I pass over details of the *post-mortem* examination, and will briefly mention that in the spinal cord, from medulla to cervical region, there were numerous capillary apoplexies irregularly distributed over the grey substance of both halves of the cord. These hæmorrhages increased both in size and number as the cervical enlargement was reached, where they exhibited a distinct anatomical distribution—occupying the central parts of the anterior cornua, and

[1] Monakow, Arch. für Psychiat, vol. x., part 2, page 495.

[2] Oeller zur Pathol. Anat. der Bleilähmung.

their extension into the posterior. In nearly every instance the blood had extravasated itself into the perivascular spaces, or the hæmorrhage had diffused through the grey substance. Along with these apoplexies, were foci of softening in the anterior horns of grey matter—all remnants of previous hæmorrhages. The ganglion cells were in a state of advanced atrophy: in many of them the nucleus was absent, and the prolongations of many of the cells had disappeared: some of these cells too, were pigmented, particularly in the neighbourhood of the nucleus, whilst others were vacuolated. The white matter of the spinal cord scarcely suffered in proportion to the grey. It is to be noted that the changes I have described, did not extend below the cervical enlargement of the spinal cord. In the nerves of the extensor muscles, healthy as well as degenerated axis-cylinders were met with. The medullary sheath was either healthy, or in a state of granular degeneration. In places, the interstitial tissue was increased. The affected muscles exhibited well marked nuclear proliferation, loss of transverse and longitudinal striation, and there was also noticed a granular condition of the muscle substance. In Oeller's case, what strikes us most is the fact, that the lesion of the spinal cord was far in advance of that in the peripheral organs. This, whilst suggesting, by no means proves that the central lesion was the first to be developed. Changes in the central nervous system may be consequent upon alterations at the periphery, or *vice versa*, but what absolutely proves that the central lesion in Oeller's case was primary, and dominated these alterations of structure noticed in the periphery, is the nature of the changes met with in the cord. Partial degeneration of ganglion cells, with hypertrophy of the surrounding connective tissue, might, doubtless, be explained by an ascending degeneration, but the vascular changes, the hæmorrhages and softening would still remain unaccounted for. Vulpian found in a man who had died from lead poisoning, and in a dog in whom paralysis of hind legs, followed by weakness of the fore limbs, had been induced by injection of carbonate of lead,

very distinct evidence of subacute myelitis. I advocate, therefore, a central origin for the primary lesion in lead paralysis, but am forced to admit, from the vulnerability of all parts of the nervous system, that the lesion might be peripheral under certain circumstances, and I base that remark upon this experience. In the course of fevers and certain constitutional states, a limited paralysis may develop, for example in phthisis, diabetes, alcoholic-poisoning, and so on. Whilst in Bergen last summer, I had the opportunity of seeing some cases of leprosy in the leper hospital, under the care of Drs Hansen and Lofte. Two of them suffered from wrist-drop—a paralytic condition which was regarded as peripheral. A transverse and longitudinal section of a nerve, removed from a paralytic patient who had died of leprosy, was placed under the microscope, and in it could be plainly seen numerous bacilli lying between the nerve fibrils, and which had produced complete destruction of the nerve fibres. Here was disease with a distinct peripheral lesion. If micro-organisms are capable of producing peripheral lesions of nerve and muscle, I see nothing to prevent the same being induced by lead, the only point of difference between the two being this, that in infectious diseases the flexors as well as the extensors suffer, whereas in lead poisoning the extensors only. My colleague, Dr Drummond, has observed paralysis of the flexors, with atrophy, in a case of lead poisoning. Among recent writers, Dejerine-Klumpke is one of the strongest advocates for the peripheral seat of the lesion, but whether we regard it as primarily central or peripheral, the answer is not yet forthcoming as to why the extensors are specially affected in lead poisoning, to the exclusion of other muscles supplied by the same nerves. The examination of sections of the spinal cord in Oeller's case and in Drummond's are of interest on this point.[1] The anterior nerve root divides

[1] I am supported, in regard to the opinion here expressed, as to the nerve lesion in lead poisoning being at least in many cases primarily central by the following:—Alcoholic neuritis is generally admitted to be peripheral. Lately this has been doubted. In the *Lancet*, May 9th, 1891, p. 1058, is a brief abstract

FIG. 15.—Portion of grey matter of anterior cornu of spinal cord from Dr. Drummond's case of lead poisoning; shows extreme hyperæmia of small blood-vessels. (One-sixth inch.)

FIG. 16.—Transverse section of posterior interosseous nerve. Dr. Drummond's case of lead poisoning showing increase of connective tissue. (Crouch, one-sixth inch.)

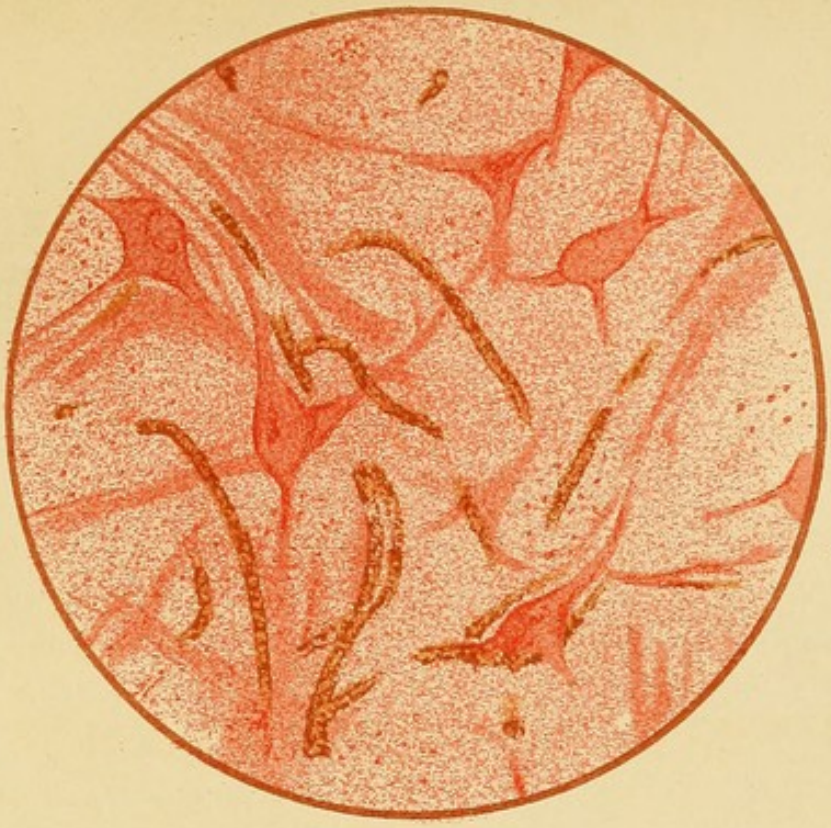


FIGURE XV.

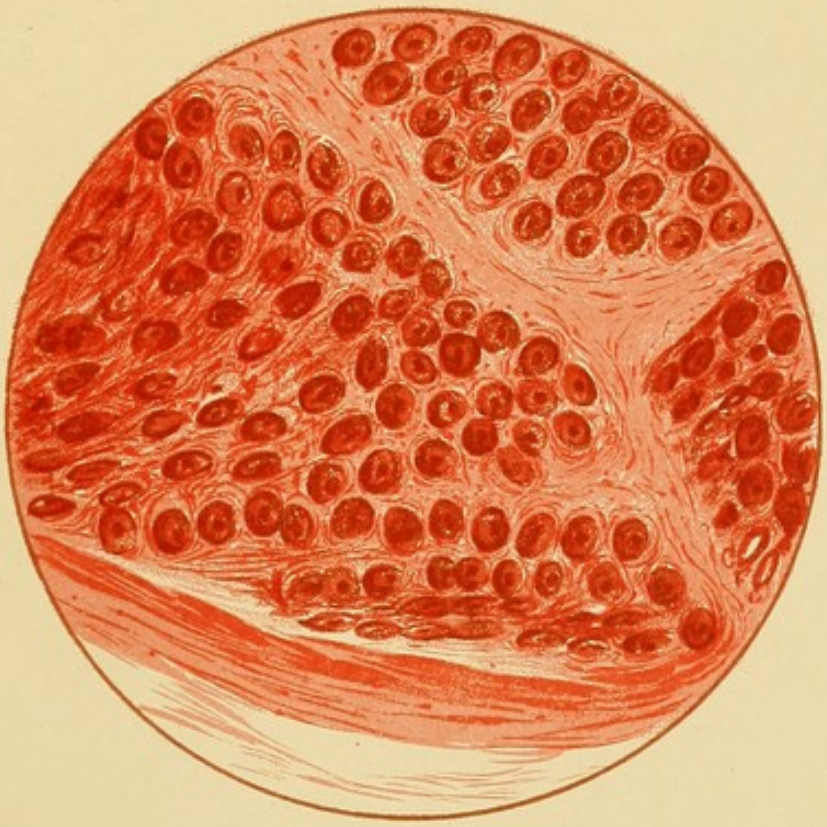


FIGURE XVI.

the ganglion cells in the anterior cornua into distinct groups—particularly into two groups, a median and a lateral, in the greatest length of the cord, but in the cervical enlargement this grouping is not so distinct, or so simple. The two large groups are here divided into smaller groups—four to eight. This anatomical sub-division must serve some physiological end. It possibly means that this latter grouping of ganglion cells is the expression of an arrangement, according to a fixed law, of the motor nuclei of the cord, whereby separate groups represent various motor departments; but what kind of motion is governed by one set of cells, and what by another, it is impossible to say. Both in Monakow's case and in Oeller's the morbid process had mainly located itself in the central portions of the anterior cornua. In both cases there was marked paralysis of the extensor muscles. Can we, from these facts, assume that the central ganglionic cells in the anterior cornua of the grey matter in the cervical enlargement of the spinal cord, represent the motor centres of the extensors of the upper extremities? We may, but this still leaves unsettled the question, why these cells in par-

of a paper, "Trophic Centres in the Cord," by Brissaud. The paper deals primarily with the subject of Alcoholic Neuritis, and the subject discussed is, whether, associated with the changes which are acknowledged to be present in the peripheral nerves, there may not co-exist in the spinal cord some change in the cells of the anterior horns of grey matter. It is not denied that the chief force of the poison seems to expend itself on the peripheral nerves in alcoholic paralysis, as in other forms of toxæmia, in which weakness and muscular wasting are observed, such as lead palsy; but the author contends that, as in some of these cases, there certainly are changes in the grey matter of cord, so, it may be, similar or analogous changes may at times be recognisable in cases of multiple neuritis from alcohol. . . . And even if changes in the cord are not recognised, it does not follow that they are not present, for it may be that changes do exist, which by our present methods are unrecognisable. A curious fact in this connection is that mentioned by M. Raymond, who produced artificially a slight degree of myelitis in animals, the symptoms of which soon disappeared. When the animals were killed a year or eighteen months later, examination could reveal no trace of the original myelitis, but the nerves in connection with the injured segment of the cord were not unfrequently found to have undergone profound alteration."

ticular should be so specially affected in lead poisoning to the exclusion of others.

Before leaving the nervous system, I should like to say a word or two upon the effect of lead upon the *optic nerve and retina*. Two kinds of neuro-retinitis are met with in lead poisoning, one the acute, in which the borders of the disc are swollen, ill-defined, and irregular; the disc itself hyperæmic and mottled, or generally reddened; the vessels obscured, or if observable, they are seen to be narrowed, and have delicate white lines running along their borders; the veins are distended; occasionally hæmorrhages are seen just at the border of the disc, or in the retina. This is the form of neuritis most frequently met with in cases of acute encephalopathy, and may either precede or follow the attack. It is accompanied by loss of sight, often absolute, and is followed by partial or complete restoration of sight. Optic atrophy is a frequent consequence. Gowers, in his "Medical Ophthalmoscopy," page 233, says, "these ocular changes commonly occur in chronic cases of lead poisoning, which have presented toxic symptoms for some time, often several years, and they coincide with an increase of the other symptoms." Such is not altogether my experience. This is, in short, a form of optic neuritis, which may be rapidly developed; it occurs in people who have only worked a few weeks or months in the lead factory, and after suffering from acute headache and vomiting, they suddenly lose their sight, and become convulsed. Sight may be regained, or it may be permanently destroyed. The drawings represent the conditions in question.

Optic atrophy frequently follows. The disc, with its slightly irregular borders, is then seen to be extremely pale, small, and the vessels contracted. The changes in the fundus oculi frequently occur in cases in which there is no albumen present in the urine, and where no special changes are detected in the brain or meninges after death.

The second form of neuro-retinitis met with in lead poisoning, is in old cases where the kidneys have become affected and there

Fig. 17.—Lead poisoning, acute neuro-retinitis. John F. A., æt. 29. White lead worker. October 30, 1884, under care of Prof. Philipson, was admitted for very extensive paralysis ; the muscles of arms, shoulders, back, and legs affected.

Fig. 18.—Lead poisoning (late stage) of the albuminuric retinitis.

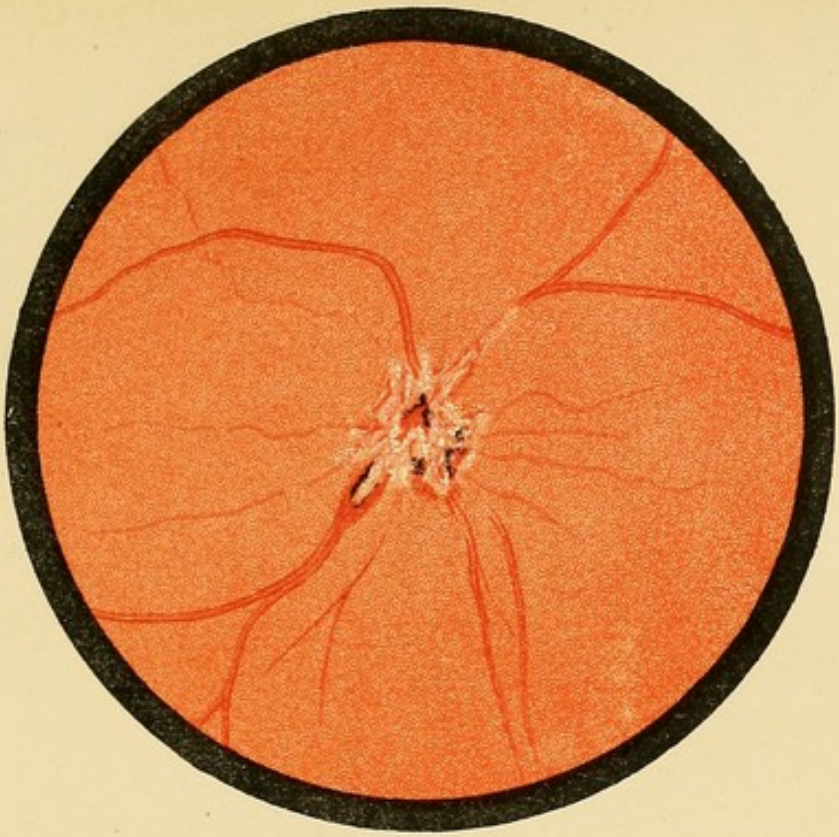


FIGURE XVII.

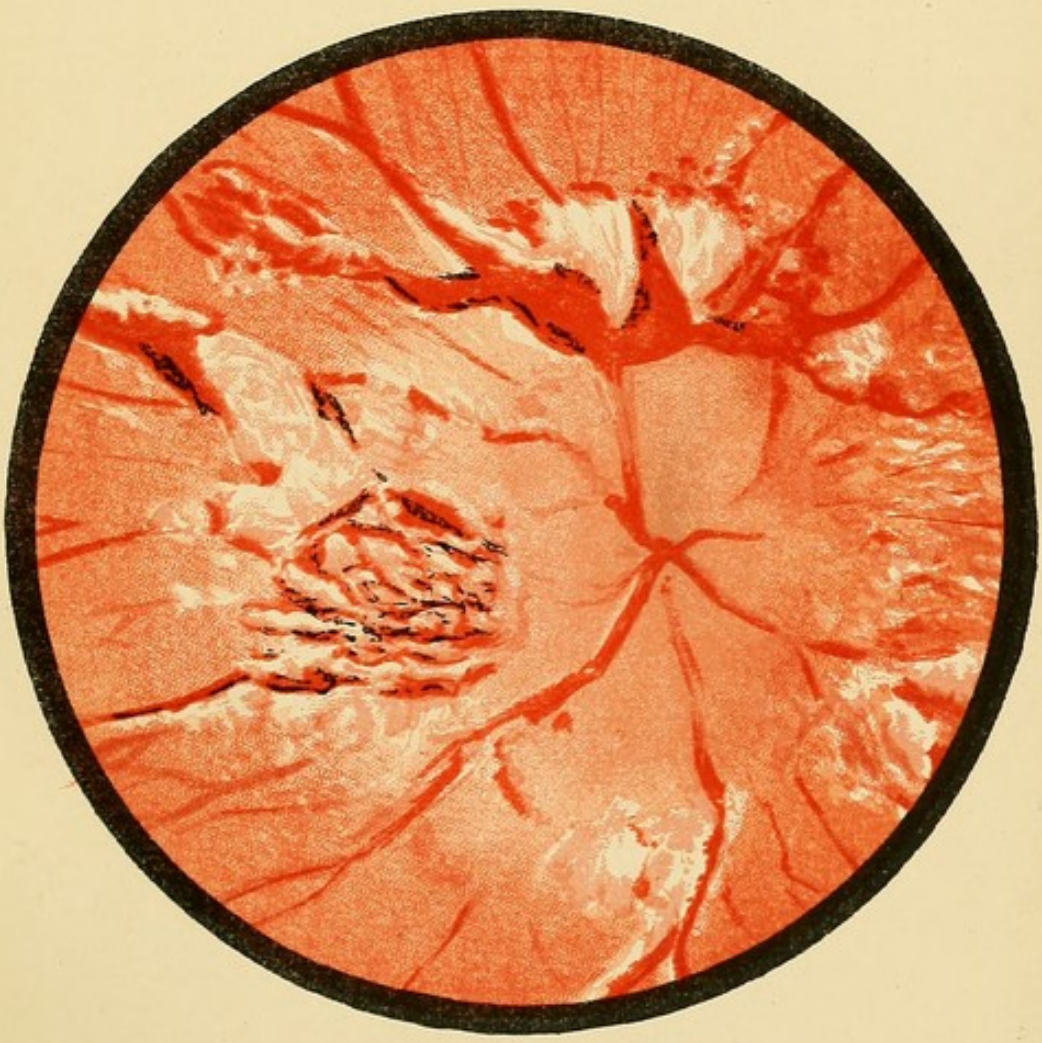


FIGURE XVIII.

FIG. 19.—Optic nerve and retina ; case of acute lead poisoning, encephalopathy. Barbara R.

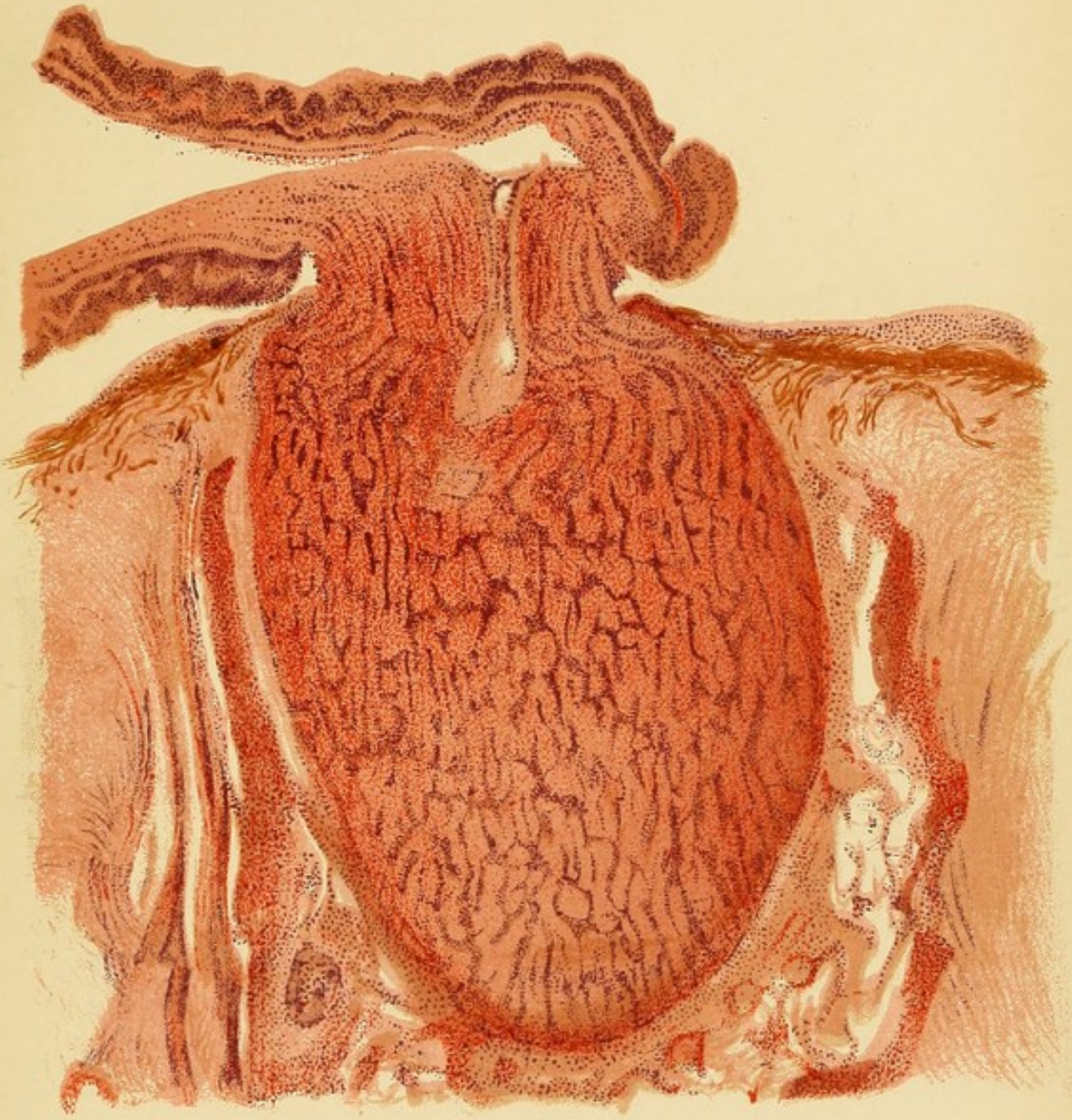


FIGURE XIX.

has been albuminuria. Here the disc is swollen, and there are extensive patches of exudation around the vessels. The appearances presented are simply those of albuminuric retinitis, and have nothing to do, in appearance at least, with the simple form of acute optic neuritis first alluded to.

In not a few cases, on the other hand, transient loss of sight is complained of, and in this no retinal changes are observed, capable of explaining the transient amblyopia. Here the poison has in all probability acted more upon the nerve centres than the peripheral expansions, very much as in the temporary blindness in other forms of poisoning. The patient soon recovers sight, although the blindness may have been complete whilst it lasted.

The subject of lead optic neuritis is extremely interesting, on account of the discussion which has gathered round it. Some observers, notably Jeaffreson,[1] do not regard the optic neuritis which arises in the course of saturnine poisoning, as in any way specially related to lead. Lead only plays, it is said, a secondary part. Optic neuritis may develop, but that is due, we are told, to lead acting mechanically, and inducing sudden and rapid effusion into the ventricles and sub-arachnoid spaces, and the nerve sheaths become distended. Disordered menstruation, particularly amenorrhœa, is by Jeaffreson regarded as a strong predisposing cause of the neuro-retinitis. Other observers, again, maintain that the neuro-retinal changes are uræmic and due to albuminuria. My argument is, that long before any changes are developed in the kidney sufficient to give rise to albuminuria, optic neuritis is present, and that it may pass through all its stages: the acute, in which blindness is complained of, to death; it may end in complete recovery, or proceed to optic atrophy, followed by permanent blindness, without the least trace of albumen having appeared in the urine.

Case I. The case of Barbara R——, aged 33, married, may be mentioned here in connection with the accompanying draw-

[1] "Clinical Lecture, Optic Neuritis," *Lancet*, May 3rd, 1890.

ing of the retina. She was admitted into the Newcastle Infirmary suffering from convulsions and coma, on the 4th December 1890, and on the following day she died. She was a well nourished woman, but extremely pale. She had on several occasions worked in the lead factory during the last few years, but never for more than a few days at a time. Domestic circumstances obliged her to return to the factory, where she followed her employment for a few weeks. She was seized with convulsions on November 19th, which kept recurring at intervals until December 1st, when she was seen by Mr Baumgartner. On that date she was "extremely light-headed, wanted to be out of bed, kept talking incoherently." I admitted her into the Infirmary on the 4th December, where, as we have seen, she died next day. At the *post mortem* pupils were noticed to be dilated and equal. Blue line on gums well marked. Heart healthy, $9\frac{1}{4}$ oz. Lungs healthy. Liver weighs $44\frac{3}{4}$ oz. A cicatrix-like scar is noticed on the upper surface of the right lobe; it is star shaped; liver tissue presents nothing abnormal—the scar does not extend into the liver substance. Kidneys together weigh 10 oz.; capsule slightly adherent at places. Kidney tissue presents nothing abnormal. Spleen weighs 4 oz., is soft and pulpy. Stomach contains several ounces of a gruel-like fluid. Cerebrum, cerebellum, and pons weigh together $42\frac{1}{2}$ oz. The surface of the brain is extremely pale and dry. Convolutions are flattened. On section the brain tissue is noticed to be very pale and dry; is firm to the touch; very few puncta hæmorrhagica are noticed, and those are extremely small; only a few drops of serum are met with in the lateral ventricles. Cerebellum is pale, but healthy. Abdominal aorta shows signs of commencing atheroma. Ovaries healthy; the uterus contains a small fibroma. The wall of the small intestine, which is practically empty, is extremely thin; large intestine almost empty; walls thin, slightly discoloured in places dark. The posterior half of one of the eyes was sent to Mr Treacher Collins of the Moorfields Hospital for examination, and he has kindly furnished me with the following report

FIG. 20.—Lead poisoning. Acute optic neuritis. Cath. R. June 20 1890—is perfectly blind.

FIG. 21.—Lead poisoning. Optic atrophy. Cath. R. September 20, 1890. Recovered very considerably her sight.

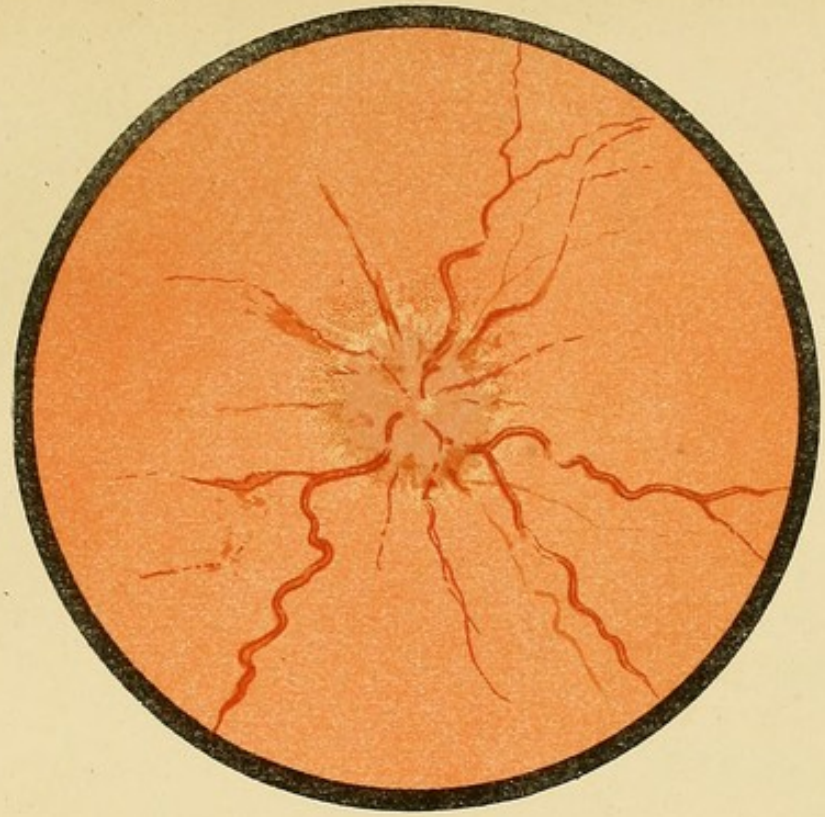


FIGURE XX.

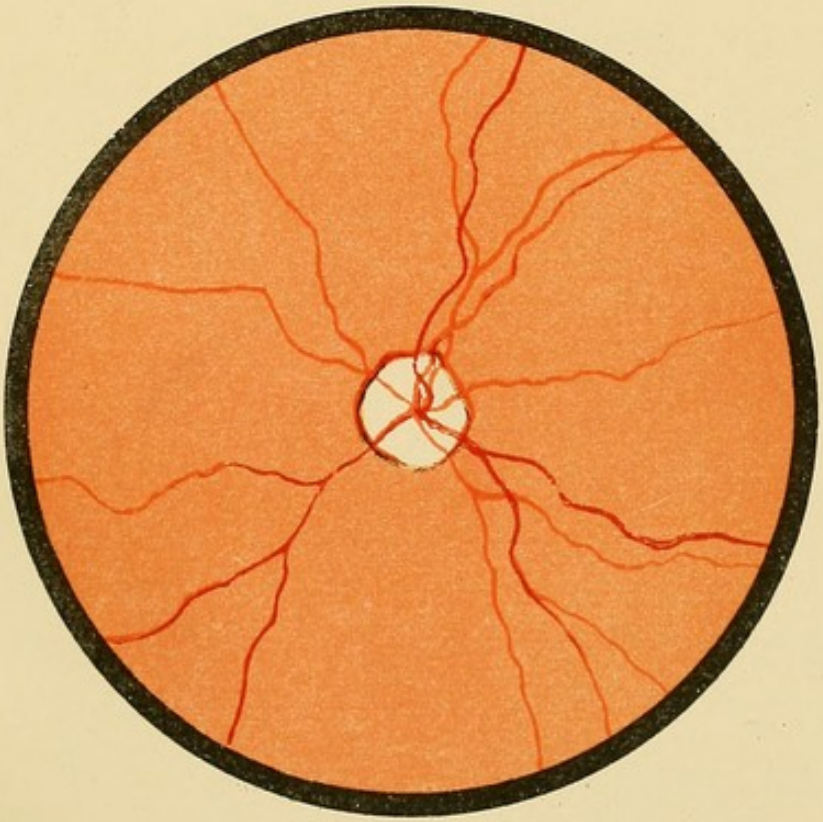


FIGURE XXI.

bearing upon the microscopical appearances. "Sections have been cut of the posterior part of optic nerve transversely, and of the optic disc and anterior part of the optic nerve antero-posteriorly. The former show scarcely any morbid changes, only slight hypernucleation. The central vessels appear healthy. The latter show considerable round cell infiltration of the fibrous trabeculæ of the nerve between its bundles of nerve fibres. The most marked infiltration is at the posterior surface, and for some little distance behind the lamina cribrosa. The papilla is somewhat swollen. There is a layer of cell infiltration between the pial and dural sheaths. The blood vessels appear healthy. The retina shows no changes.

Case II. *Rapid death in lead poisoning.* Elizabeth T——, æt. 22, single, admitted into the Newcastle Infirmary, July 18th, 1889, worked two and a half years at the "white lead." After the first three months, she was obliged to leave off work for three weeks, owing to colic. She returned and worked for seven weeks, when she was again obliged to leave on account of colic. In August 1888, she had severe pain in the head, which was followed by partial blindness. She did not return to the factory for two months. Gradually she regained her eyesight, and has since then worked off and on at the lead works. She began to menstruate at the age of fifteen; her menses, which have been regular, have been scanty since she went to the lead factory. At present the patient is menstruating. For the last few days, she has complained of pain in her joints and loss of eyesight. Urine normal, free from albumen. The patient died in a convulsion early on the morning following the day on which she was admitted.

Post mortem. Body that of a well-developed female. Blue line on gums. No œdema. Pupils semi-dilated. Lungs healthy. Pericardium healthy, contains about two drachms of serum. Heart healthy, weighs 10½ oz. Right ventricle—walls flaccid, cavity empty. Left ventricle—wall fairly thick, chamber empty, aortic valve competent. Endocardium healthy, valves all healthy.

Liver smooth, healthy, weighs 60½ oz. Gall-bladder contains fluid bile in small quantity. Liver-tissue on section is seen to be pale. Spleen tears easily, is soft and pulpy, weighs 6½ oz. Left kidney 5¼ oz. Capsule is removed with ease. On section the veins in cortex and medulla are seen to be injected, otherwise nothing abnormal is detected. Right kidney 5¼ oz. Capsule removed with ease. A small quantity of pus is seen exuding from pelvis of kidney, but the lining membrane is not noticed to be injected. Kidney substance rather injected but healthy. Vagina—hymen absent. Uterus—cervix eroded and granular. Interior of uterus covered with a red slimy material, which may be menstrual. Ovaries—right ovary enlarged, contains two or three corpora lutea, one yellowish, the others rather red, but evidently not very recent; left ovary smaller and somewhat cystic. Stomach healthy, small ecchymoses near pylorus in upper wall. Large intestine—longitudinal and circular muscular fibres well developed, mucous membrane distinctly injected. Brain—dura mater slightly adherent at vertex; subarachnoid fluid has accumulated to excess in the interpeduncular space; pons and cerebellum extremely pale compared with rest of brain, the pallor being particularly noticeable in the pons. The surface of the brain is healthy; vessels not unduly injected. Corpus callosum very pale, as also brain-tissue generally; very few puncta hæmorrhagica. Each lateral ventricle contains two or three drachms of serum. Membranes of brain other than stated above are healthy, and there is no effusion. Spinal cord feels extremely hard, and is pale.

Case III. *Acute lead poisoning, blindness, recovery, fresh exposure to lead, further loss of eyesight for twenty-one weeks, recovery of sight, atrophy of discs.* Catherine R., aged 23, single, a white-lead worker, was seen by me along with Dr Bowlan, at the Newcastle Workhouse, in July 1890, suffering from blindness of eight weeks' duration. In the previous year, she had been under my care in the Royal Infirmary, suffering from acute lead poisoning (colic, headache, and vomiting,

FIG. 22.—Optic atrophy in lead poisoning. Elizabeth H. Permanent blindness. June 28, 1890.

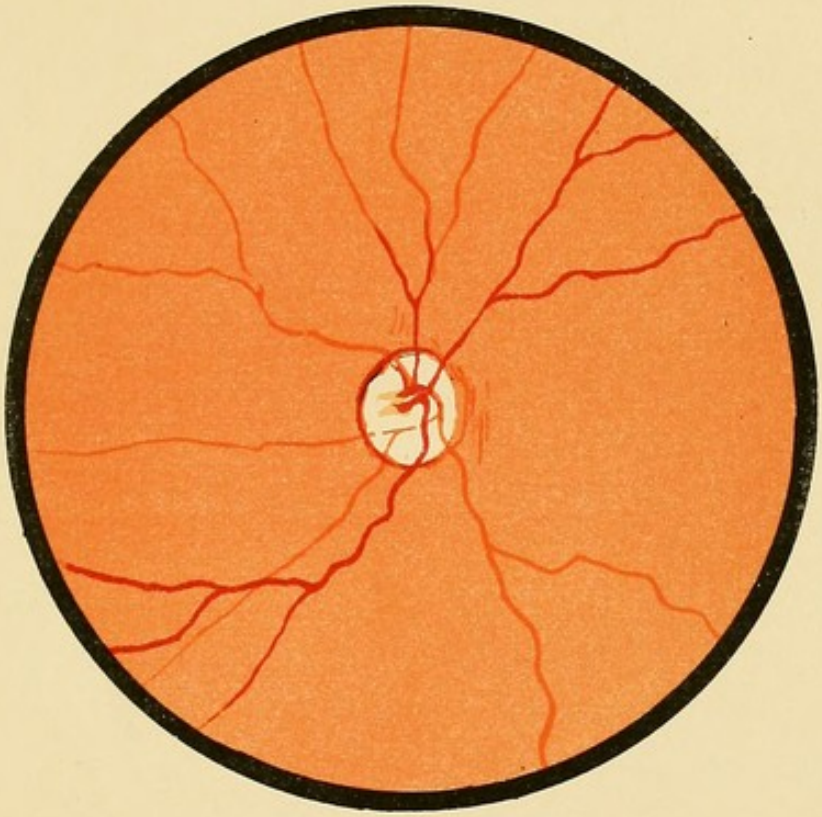


FIGURE XXII.

with slight blindness), and after three weeks she was discharged feeling quite well. She returned to the lead factory, where she had worked with more or less regularity for six years. After following her employment for several months, she began to experience great weakness, headache, and pain in the bowels and right wrists, with slight wrist drop, but at this time her vision was quite good. Acting upon medical advice, she went into the workhouse hospital, where two days after her admission her eyesight suddenly left her, not completely, but the partial blindness gradually deepened, so that in a day or two she was totally blind. Dr Bowlan found the urine to be quite free from albumen on the date of her admission, 14th of May 1890. She had not menstruated, however, for two months. She complained of severe headache. Two days after admission, she began to menstruate, and on the third day, she found she had lost her eyesight, she could not see the medicine when it was handed to her. The headache had not only continued but increased in severity. Her pupils were widely dilated, and the discs appeared woolly; right knee jerk was absent, the left was exaggerated. On the fourth day, when I saw her with Dr Bowlan, nystagmus was well marked, the movements were chiefly vertical. There was still the partial wrist drop, also hyperæsthesia of arms and legs. Before blindness came on she had diplopia. On examining the discs, which were hazy, the veins were seen to be enormously engorged, and there were retinal hæmorrhages. By degrees the pains in her limbs subsided, and headache disappeared, but she remained quite blind, the nystagmus still being as pronounced as formerly. The left pupil was, on May 30th, noticed to be very much larger than the right. On the 30th July, I brought her into the Newcastle Infimary. This was eight weeks after the onset of her illness, and she was still totally blind. Otherwise she was feeling well; the urine did not contain albumen. The discs were now observed to be passing into a state of atrophy. Potassium iodide was prescribed. After a week or two she was able to see, but very indistinctly, the nurses. At the end of

August she was able to see a window at the end of the ward, and could see the medicine-glass when held out to her. All this time the blue line on her gums was well marked. On the 10th October, that is, exactly twenty-one weeks to the day on which she became blind, I made the following note:—"This patient was totally blind on admission; the condition continued for some little time afterwards; for weeks past her sight has been gradually returning, and she is now able to enumerate correctly the number of clerks and nurses collected round her bed, and to discriminate between the various nurses." Here then is a case of complete restoration of eyesight, which had been lost for more than four months. The discs as seen in the drawing are atrophied. In one of Mr Baumgartner's cases, sight was restored after a period of five weeks' blindness.

Case IV. The next case is one where *colic and partial wrist drop were followed by a slight fit*, this again by grave *epileptiform eclampsia*, the actual onset of which was possibly *accelerated by grief*. The illness almost terminated in death from coma. *Total blindness has been the result.* For the opportunity of seeing this case, I am indebted to Dr Bowlan.

Eliza H., aged 25, previously a white lead worker. In her family history there is nothing special to relate, save that she lost a good many brothers and sisters in infancy, also a sister from phthisis aged eight, and it is her death which plays a *rôle* in our patient's illness. When an infant, E. H. was thought to have had "water on the head;" there are remains of a scar behind the ramus of lower jaw on the left side. She was healthy from six to fifteen years of age, when menstruation appeared; she lost very little and had much pain, was regular for the first three months, then saw nothing until she was twenty. Five years ago she went to the lead factory, and worked in the stacks, carrying white lead in boxes to the mills to be ground. After five months' service, she was seized with colic, and was ill for seven weeks; she recovered, and on presenting herself at the lead works, was refused employment.

Her menstruation, which had ceased between the ages of sixteen and twenty, reappeared soon after she entered the lead works; it came regularly at first; it was scanty and painful. Some months after the refusal to allow her to prosecute her usual toil, she obtained employment in another factory, where she worked with interruptions for the next two years in the "stoves." During this period she had another attack of colic, accompanied by headache, and her wrists became weak. She soon recovered and again returned to work, but had not been thus engaged for three weeks, when she was seized with a "fit" on her way to the factory at six a.m.; unconsciousness lasted for fifteen minutes; her comrades helped her into the factory, where, as this attack was unknown to the employers, she worked all day, feeling as she said very shaky. For the next two months she followed her employment, having neither fits, colic, nor double vision, but with weakened wrists. A few days prior to the 23rd of July 1888, her sister, to whom she was much attached, died of consumption, and she fretted after this child's death. Patient, however, still followed her employment, but at this date, and when at work, she was seized with a convulsion and became unconscious, in which condition she was admitted into the Workhouse hospital under the care of Mr Dodd. A few days afterwards, she had a succession of fits, during which she had to be closely watched, for although unconscious she would spring out of bed. Urine and fæces were passed involuntarily. Dr Bowlan's note two months after is briefly this: A well developed woman; pupils widely dilated, patient is totally blind, and is very anæmic; bowels regular, no trace of blue line on gums; pulse normal; urine specific gravity 1012, daily average 60 ounces. No albumen. Urea 7.5 per cent.; has incontinence of urine. Menses, which have been absent for several months, have twice appeared at intervals of one month. She has occasional headache over the frontal region. The grasp of the left hand is feeble compared to the right; the left hand is always bedewed with perspiration—there being almost constantly large

beads of perspiration upon the palm, whilst absent from the right hand. There is slight anæsthesia over parts of the left forearm, which measures a quarter of an inch less than the right. The anæsthesia was subsequently noticed to be more widely distributed. It was confined to the left half of the whole body, including the face. The knee jerk was absent. Ophthalmoscopic examination shows atrophy of both discs.

It is difficult to say, positively, what is the cause of optic neuritis in acute lead poisoning. Occurring without any albumen being present in the urine, it has no resemblance to the albuminuric retinitis met with in the chronic form of plumbism, with advanced kidney lesion. It may be a descending neuritis, or it may possibly depend upon a distension of the sheath of the nerve, but in no case has the sheath of the nerve appeared to me distended. In nearly all cases with acute brain symptoms, arising in the course of lead poisoning, there is increased intracranial pressure. The convolutions are flattened, and this may not be without its influence, although, possibly, lead itself may have some special effect upon the disc and retina. Leber and Deutschmann have propounded the theory that optic neuritis may be caused by irritating or infective particles, carried by means of the sub-arachnoid fluid from the cavity of the brain to the vaginal sheaths surrounding the nerve. This is a likely cause, but as there are many conditions in operation at one and the same time in lead poisoning, it is impossible to fix definitely upon one thing to the exclusion of others. Meningitis, however, I do exclude. Lead salts, or the products consequent upon their presence in the system, have a special irritant action upon the retina, optic nerve, and brain, and with this, there is well-marked intracranial pressure, which cannot but play an important part in the development of neuro-retinitis. On examining the brain in cases of acute lead poisoning, I have been struck by the dryness, firmness, and pallor present, and the decidedly contracted state of the arteries. Therein may lie an explanation of the causes of optic neuritis. As bearing upon this, the observation

of Schreiber[1] is interesting. A patient lay for several days in a state of profound coma, with retracted abdomen, in lead poisoning. On ophthalmoscopic examination, the retinal arteries were seen to be extremely narrowed. After inhalation of nitrite of amyl, this contraction of the arteries disappeared, as also some of the other symptoms. Each time the nitrite of amyl was inhaled, this change in the calibre of the vessels occurred.

Occasionally lead encephalopathy is ushered in by hysteria. When this occurs, the medical attendant may be entirely thrown off his guard. I have seen a young woman, a lead worker, who was the subject, apparently, of ordinary hysterical convulsions, die from acute lead encephalopathy within two days. It was Debove and Achard who first employed the term toxic hysteria to indicate cases in which the neurosis was developed under the influence of intoxication. To them, hysteria was only a symptom of saturnine poisoning. Letulle, admitting the identity of saturnine and ordinary hysteria, maintains that lead only prepares the soil for the development of the neurosis in subjects already predisposed. That predisposition, according to Guinon, is heredity. Mr Baumgartner, who has medical charge of lead factories in Newcastle, tells me that he has observed this on a few occasions. It was Jaccoud who introduced the comprehensive term of cerebro-spinal saturnism, so as to include the great variety of nervous symptoms met with in lead poisoning. The presence of anæsthesia has occasionally rendered the diagnosis of lead poisoning difficult. Guinon has collected several cases of hysteria occurring in saturnine poisoning. In these, there have been the combination of symptoms of organic and functional disease. Manouvrier[2] found loss of muscular sense, sensorial anæsthesia of sight and hearing, of odour and taste. Wherever the following characteristics are met with—anæsthesia limited to one half of the body, concentric narrowing of the field of vision, dyschromatopsia, the

[1] Deutsches Archiv für klin. Med., 1878, page 99.

[2] Manouvrier, Archives de physiol., 1870.

attacks accompanied by general convulsions lasting for some considerable time, absence of aura, absence of any injury such as would have occurred had the malady been truly saturnine epilepsy,—the attack is hysterical. Hysteria may be the only symptom, therefore, of saturnine poisoning. Charcot has drawn attention to the hysterical nature of a large number of the so called anæsthesias, even when they occur in the male.

Of the mental symptoms produced by lead poisoning, I would mention delirium, met with during the course of acute encephalopathy, sometimes so violent as to amount to acute mania, and noticed too in the late stage of the disease, when the kidneys are affected. This delirium, as Dr Alexander Robertson noticed,[1] is not attended by rise of temperature, and is, therefore, not due to meningitis. In one of my own cases, there were the symptoms and delusions of general paralysis, and distinct meningitis with sub-arachnoid effusion, was found, *post-mortem*, in the neighbourhood of the fissure of Rolando. Dr Hale White has reported similar cases. I may safely say that saturnine poisoning sends few patients to the asylums. In the Waterford Asylum Report of 1871, Dr Maccabe[2] recounts a case of monomania and depression, arising from lead poisoning, in which the symptoms of mental derangement disappeared as the patient recovered from the effects of the poison. The patient was a woman, aged 50, who was a white lead worker. She exhibited the blue line on her gums; the extensors of her wrists were weak. Admitted into the asylum suffering from monomania, her insanity was disregarded, and she was put upon treatment for lead poisoning by iodide of potassium. The blue line began to fade, and her hallucinations disappeared. She made a good recovery. In this case, it is impossible to say whether any special predisposition to insanity or other neurosis existed, but the fact that treatment was successful, shows the imperative necessity of attending to the bodily conditions that underlie insanity. Dr Campbell

[1] *Journal of Mental Science*, July 1886.

[2] *Journal of Mental Science*, 1872-73, page 233.

Clark, in the *Journal of Mental Science,* October 1883, reports a similar case, but with an alcoholic as well as with a saturnine history, in which partial recovery took place. In one of my own cases, there was a striking loss of memory, a circumstance which has also been noticed by Dr Campbell Clark in one of his cases. My patient, a man aged 25, was admitted after having colic and a few epileptiform convulsions; there was no albumen in the urine; he had well marked blue line on his gums, but there was neither paralysis, anæsthesia, nor hyperæsthesia: his knee jerks were diminished. Several days after he had recovered from the convulsions, he was unable to tell me what his occupation was, nor could he name the factory in which he was employed. On mentioning to him, however, a list of the factories in Newcastle, he was able to fix upon his own. As regards the name of the street he lived in, and the part of the town in which the street lay, he was equally unable to express himself. He was quite conscious of this defect in his memory, and always asked for time to consider the questions before replying to them. He regained his memory.

LECTURE III.

We have dealt somewhat exhaustively with the symptomatology of lead poisoning. It now devolves upon me to draw your attention to the channels by which lead in its various forms is introduced into the system. It may at once be said that lead gains entrance into the human body by the skin, by the lungs, or by the digestive organs. The mere handling of lead in its metallic state develops a peculiar odour—indicating the escape of metallic particles in a state of fine division. It is thus that we seek to explain the wrist-drop met with in leather-cutters and type-setters. Tanquerel does not admit that any absorption of lead occurs through the skin. His opinion upon this point is very decided. He says that colic has never been known, in the practice of the surgeons of the Paris hospitals, to follow the local application of lead. Actors who use cosmetics in which white lead is an ingredient, are never affected with colic. Our experience is the opposite of this. I regard the skin as a very suitable surface for the absorption of lead. I have seen lead amaurosis in an actress from the use of cosmetics, and the reason why actors and actresses do not suffer more frequently is, in all probability, owing to the fat in the paste blocking up the pores of the skin. The symptoms which follow the use of hair dyes is an illustration of the fact that only through the skin could the lead have passed into the system. In my own experiments I found that the application of lead oleate, mixed with lanoline, to the skin of rabbits was more quickly followed by symptoms than when lead salts were ingested by the mouth. From these facts, and the

experiments of others, we say that the epidermis is not an obstacle to the absorption of lead, as Tanquerel maintained. In two of my patients, lead was found by Professor Bedson in the perspiration. If lead is capable of being eliminated by the skin, it must be capable of entering the system by the same channel.

As regards the entrance of lead by the respiratory organs there is no doubt. People who have slept a few nights in newly painted rooms have suffered from colic. Colic has been experienced by naval officers whose sleeping cabins had been but recently painted. To this circumstance, namely, lead dissolved in a volatile agent, such as turpentine, we attributed part of the colique sèche met with on board French men-of-war. Lead dust, disseminated through the atmosphere whilst entering by the digestive organs, may also pass into the system by the respiratory mucous membrane. The insoluble lead carbonate is carried into the trachea and bronchi, and probably into the air cells of the lungs, but in whatever part of the respiratory passages it is deposited, it comes under the influence of heat and moisture—it is moistened by alkaline fluids, rich in carbonic acid coming from the lungs, and the lead carbonate is converted into a bicarbonate which is fairly soluble. A similar chemical change in all probability occurs with the fumes of the molten metal. Carried into the respiratory passages, the suspended particles of lead would first be deposited, then acted upon by the carbonic acid during expiration, and converted first into carbonate, and subsequently, into bicarbonate.

Alderson says that statistical observations have shown that absorption by respiration is more productive of deleterious consequences than any other method of receiving the metal into the system. On this point I offer no opinion. Sir Joseph Fayrer, remarking upon my paper "on Lead Poisoning," two years ago, at the Royal Medico-Chirurgical Society, alluded to the case of a gentleman in Calcutta who was anæmic, and was supposed to be suffering from one of the Protean results of malarial fever, or climatic disturbance. He was sent home, and on his symptoms

becoming worse Sir Alfred Garrod, under whose care he had been placed, again investigated the case, and finding that the patient, who was suffering from plumbism, was an inveterate snuff-taker, he discovered that the source of the poison was the leaden envelope in which the snuff was encased. When the source was detected, suitable remedies were employed, and the patient soon recovered.

The channel, par excellence, by which lead is passed into the system is the digestive canal. Under any circumstances, the amount of lead which is absorbed is small. As lead is never present in the blood or tissues to any great extent, we must admit that very little of the poison is absorbed, and that when absorbed it is eliminated immediately by the kidneys and skin, and that the insoluble lead is thrown out in the fæces. That it is eliminated freely by the kidneys is shown by its presence in the urine of lead workers, and those who are suffering from colic. Most of it, however, is unabsorbed, as is shown by the infrequency of symptoms in those who are exposed to the metal or its compounds. That the fæces carry off the unaltered lead compounds, is confirmed by an observation like the following:—

J. K., aged 40, a healthy man, formerly engaged in agricultural labour in Devonshire, came to Tyneside and got employment in a red lead factory in Newcastle. He had not worked more than three or four days when part of the machinery broke down, and the atmosphere of the factory, which he had previously noticed to be quite clear, now became so thick that the workmen could scarcely see each other across the room. In this dusty atmosphere, he followed his occupation for twelve days, when he was seized with severe colic, which obliged him to come to the Infirmary. When I saw him he was anæmic, and had a deep blue line on his gums. His hair, which was naturally greyish, had become distinctly of a light carmine colour, and in spite of baths, this colour persisted; his night-dress, owing to perspiration, was also coloured red. His bowels were constipated. For several days after admission the stools were composed of hard, red lumps, in which the red oxide of lead was plainly

discernible. Two or three weeks after his admission, I had some of his hair analysed, and it was found to contain lead, a striking illustration of how such an insoluble substance as red lead, in spite of all bathing, had passed into, and become intimately incorporated with the structure of the hair.

With the object of determining the chemical conditions under which lead enters the system through the digestive canal, I had a series of experiments carried out for me by Mr Best, under the direction of my colleague Prof. Bedson, of the College of Science, Newcastle-upon-Tyne.

How, for example, do lead compounds behave in contact with the various secretions which are poured out during digestion? In the experiments which follow, basic lead carbonate, or "lead white," was used, as lead poisoning is more frequently due to this compound than to any other. Throughout all the experiments, the same quantities of lead carbonate and animal juices were as nearly as possible employed.

Human saliva has a slight solvent action upon white lead. Its influence is greater when no other ingredient is present. During the digestion of starch by saliva in the presence of lead carbonate, we found that less of the metal was dissolved than where saliva operated upon lead alone. The minced tonsils of a dog and carbonate of lead were digested together, but no lead was dissolved, either when these two substances were alone, or during the additional digestion of starch. We conclude, therefore, that whilst human salivary secretion has a slight solvent influence upon lead carbonate, that of the tonsils of the dog has none at all.

I. Digestion Experiments.

CARBONATE OF LEAD.

(*a*) By means of Saliva.

Amount of Carb. Lead.	Amount of Saliva used.	Amount of Carb. Lead taken up by Saliva.
(1) 15·4 grains.	154 grains.	·01488 grains per oz.
(2) 15·4 ,,	154 ,,	·01688 ,, ,,

(*b*) By Saliva when digesting starch (7·7 grains).

(1) 15·4 grains.	154 grains.	·00816 grains per oz.
(2) 15·4 ,,	154 ,,	·00816 ,, ,,

Gastric Juice.

Two double sets of experiments were performed with *gastric juice, artificial and natural.* The natural juice was obtained through the gastric fistula of a dog, the experiments being performed in ordinary test tubes, and also in parchment tubes, as recommended for digestive experiments by Sheridan Lea of Cambridge.

When lead carbonate was simply digested by the gastric juice for three hours at blood heat, we always found that more or less of the lead was dissolved.

(A) Gastric Juice (*a*) Natural, obtained from fistula in stomach of dog. 1 part of filtered gastric secretion mixed with 2 parts of water.

	Amount of Carb. Lead.	Condition of Digestion.	Amount of Lead dissolved.
	(1) 15·4 grains.	154 grains of diluted G.J.	·00312 grains per oz. of G.J.
Nov. 2.	(2) 15·4 ,,	154 ,, ,,	·00206 ,, ,,
Nov. 5.	(3) 15·4 ,,	154 ,, ,,	·00298 ,, ,,

(*b*) Gastric Juice (artificial—Merck's).

Lead Carb.	Amount of Artificial Juice.	Amount of Lead dissolved.
15·4 grains.	154 grains.	·001916 grains per oz.

(B) To show that the active agent in effecting the solution of Lead Carbonate in Gastric Digestion is Hydrochloric acid.

Experiments in parchment membrane.

Amount of Lead. Carb.	Amount of Pepsin.	Amount of HCl. (in terms of solution).	Lead in grains per oz. Diffused.	Lead in grains per oz. Non-diffused.	Total dissolved. grs. per oz. of solution.
15·4	15·4	·3 per cent.	4·22	1·25	5·47
15·4	7·7	·3 ,,	3·28	2·05	5·33
15·4	3·85	·3 ,,	3·32	2·16	5·48
15·4	nil.	·3 ,,	3·56	2·31	5·87
15·4	15·4	nil.	nil.	nil.	nil.

The results obtained are not intended to be compared with the experiments made with Artificial and Natural Gastric Juice.

II. Digestion Experiments.

(C) Action of Natural Gastric Juice upon Lead, when Albumen is being digested.

	Amount of Lead. Carb.	Condition of Digestion.	Amount of Lead dissolved.
Nov. 1.	15·4 grains.	154 grs. of dil. Gastric Juice, 200 grs. of diluted Albumen.	·00048 grains per oz. G.J.
Nov. 2.	15·4 ,,	,,	·00057 ,, ,,
Nov. 5.	15·4 ,,	,,	·00048 ,, ,,
Artificial Gastric Juice.	15·4 grains.	,,	·00048 ,, ,,

(B) These experiments were repeated in parchment membrane with varying amounts of pepsin, but with the same percentage of hydrochloric acid, and the results indicate that the active agent throughout is the hydrochloric acid contained in the gastric juice, converting the lead carbonate into lead chloride. They also illustrate the easy diffusibility of lead chloride. The advantage of Lea's digestive tubes over the ordinary chemical test tubes is that the dissolved products pass out of the original liquid into the surrounding fluid. Looking at these results, it will be noticed that the greatest proportion of lead carbonate dissolved, is when there is hydrochloric acid alone. When pepsin is alone present with lead carbonate, not any of the metal is dissolved. The active agent, therefore, is the acid, and the influence of pepsin is rather to diminish than increase the amount of lead dissolved.

(C) Having settled so far that the acid of the gastric juice is, in all probability, the active agent in dissolving lead in the stomach, our next question was to determine the influence of the digestion of proteids upon the amount of lead carbonate acted upon. Here, as in the case of salivary digestion of starch and lead, the presence of proteid in gastric digestion was found to reduce, considerably, the amount of lead dissolved, a circumstance to be explained by either the acid of the gastric juice acting upon the lead carbonate, converting some of it into lead chloride, which, as a fairly soluble compound of lead, would unite with the albumens to form an insoluble albuminate; or the acid is first used up in the conversion of peptone, leaving, therefore, the lead carbonate unacted upon, and which, being insoluble, would pass out of the stomach with the chyme.

One or two points of interest arise out of these observations The first is, that in all probability it is in the form of lead chloride, which is a soluble and diffusible salt, that lead chiefly passes into the system from the stomach, and the reason why herbivorous animals, such as the rabbit, are not so quickly

poisoned by lead as carnivorous animals, is due to the deficiency of hydrochloric acid in their gastric juice.

It must be as chloride and not as albuminate of lead that the metal passes into man. Chloride, or basic acetate of lead, if mixed with albumen, forms at once a dense coagulum, just as mercuric chloride does. Albuminate of lead is a very insoluble and indiffusible compound. In this coagulum, we either have a chemical compound of albumen and chloride of lead, or the albumen simply retains, mechanically, the chloride of lead. If, however, this coagulum were exposed to fluids containing salines, *e.g.*, such a fluid as blood, which contains chlorides, the lead would be gradually removed in a soluble form as chloride—a compound known to be more soluble in water containing chloride of sodium, than in water alone. In this soluble form, it would circulate in the blood and pass out by the kidneys. This is, in my opinion, the manner in which it is thrown out. Peptone, just like albumen, is precipitated by lead. It cannot be, therefore, either as lead albuminate or peptonate, that lead is absorbed from the stomach. These are insoluble compounds, and must pass on with the chyme into the upper part of the intestine.

When lead is taken into the system by means of drinking water, it is probably in the form of the bicarbonate. This is a soluble salt, and it might pass at once into the system as such from the stomach, or be converted first into chloride by the gastric juice. In whatever form lead reaches the stomach, what these experiments have shown is, that during proteid digestion, less lead is dissolved than when the stomach is empty. We find here an explanation of the readiness with which saturnine poisoning is induced by the drinking of lead-contaminated liquids, before food has been taken. These experiments offer a scientific explanation of what has long been known as simple fact. Employers have frequently noticed that it is the ill-fed who suffer, and that if the workers have breakfasted before beginning their toil, they practically run no risk whatever. The giving of a free breakfast, or the allowance of a reasonable

quantity of milk to the workers, is likely to postpone or prevent lead poisoning. This circumstance, simple as it appears to be, is most valuable—for it is no longer a fact simply resting upon ordinary observation, but is one, the truth of which can be scientifically proved. A few hours work at a time in a lead factory, and frequent meals, are the corollaries to be drawn from these experiments, and that no worker should be allowed to enter the lead factory without having first had a substantial meal.

Having satisfied ourselves that the acid of the gastric juice is the principal agent in dissolving lead in the stomach, we proceeded in our investigation to examine the action of the various juices that normally assist in duodenal digestion, viz., the *bile and pancreatic juice.* When lead carbonate was alone digested with bile, a relatively large quantity of lead was dissolved compared with the gastric juice. Bedson and Best

(D) Effect of Bile on Lead Carb. (ox bile used).

(*a*) 15·4 grains. 156 grains of Bile. ·0152 grains per oz.
(*b*) 15·4 ,, 156 ,, ,, ·0139 ,, ,,
(*d*) 10 cc of Albumen and 1 gramme of Lead were digested in presence of 10 cc. of natural gastric juice; after 3 hours at 98° F., 10 cc. of Bile were added, and digestion at 98° F. was continued for another hour.

Lead dissolved = ·00625 grains per oz. of fluid.
,, ,, = ·0125 ,, ,, bile.

With gastric juice alone it was ·00312 ; it is now greater = ·00625.
With bile alone it was..... ·0152 ; it is now less.... = ·00625.
or ·0125 for bile present.

found three times the amount of lead dissolved. When bile was allowed to act upon fat at the same time as upon lead, less of the metal passed into solution.

In order to illustrate what would occur in normal digestion when the acid chyme passes from the stomach into the duodenum and is brought into contact with the bile and pancreatic juice, we first of all digested lead carbonate with albumen in gastric juice for three hours, then bile was added and digested for another hour, the result being that the amount of dissolved lead was greater than in ordinary gastric digestion, but was less than

in biliary digestion alone. The diffusibility of lead, under these circumstances, was investigated, the experiment being made in a parchment tube. Lead carbonate was digested for three hours with excess of bile—no lead diffused; fat was now added—still none of the metallic compounds passed through the membrane.

During pancreatic digestion, either when artificial or natural pancreatic juice was employed, no lead carbonate was found to be dissolved, and this applies to the action of this juice upon lead alone, or when, in the process of digestion, there were also present peptone, fat, or starch. When combined with bile, it was noticed that there was an amount of lead dissolved, which practically agrees with the effects of bile alone. The experiment also confirmed what has already been shown, viz., that during the digestion of food, the amount of lead dissolved is always lessened.

III. Series of Pancreatic Digestions.

A	B
1. 1 gramme of $PbCO_3$: 5 cc. of Benger's Liq. Panc. digested 4 hours at 98° F. No Lead.	1 gramme $PbCO_3$: 10 cc. of Panc. extract* in alkaline medium digested for 3 hours at 98° F. No Lead.
	* Made from pancreas of dog recently killed.
2. 1 gramme of $PbCO_3$: 2 grammes of Merck's peptone : 10 cc. of Benger's Liq. Panc. : digested 4 hours at 98° F. A doubtful trace.	1 gramme of $PbCO_3$: 2 grammes of Merck's Peptone : 10 cc. of Panc. Ext. as before, digested 3 hours at 98° F. No Lead.
3. 1 gramme $PbCO_3$: 1 gramme of Starch : 10 cc. of Benger's Liq. Panc. digested 4 hours 98° F. No Lead.	1 gramme $PbCO_3$: 1 gramme of Starch : 10 cc. Panc. Extract, digested 3 hours 98° F. No Lead.
4. 1 gramme $PbCO_3$: 2 cc. of Olive Oil : 10 cc Benger's Liq. Panc. digested 4 hours at 98° F. A doubtful trace.	1 gramme of $PbCO_3$: 2 cc. of Olive Oil : 10 cc. Panc. Ext. digested 3 hours 98° F. No Lead taken up.
	A digestion performed in a portion of dog's intestine (animal killed immediately before the experiment) gave also negative result.

Complete gastro-duodenal digestion experiment. 50 cc. of normal gastric juice were allowed to react upon 15 cc. of dilute albumen in presence of 1 gr. $PbCO_3$. After digestion for 3 hours, ·05 grs. of Pb (in form of Chloride ?) had diffused. Into the tube 20 cc. of bile, 5 grs. of animal fat, ·5 grs. of partially digested starch, and 10 cc. of Liq. Panc. (Benger) were added. Temp. was maintained for other 2 hours. The additional amount diffused was ·012 gr., whilst the dissolved but undiffused lead was ·029 gr.

In order to lay before you as comprehensive a pathology of lead poisoning as time and circumstance permit, I have had recourse to experimental investigation, and whilst admitting that the results obtained from this source are not to be taken as applicable *in toto* to human pathology, they go very far to substantiate opinions which I have previously expressed upon the pathology of acute lead poisoning. In some respects lead poisoning in animals differs from that in men. Even though animals receive lead by the same channels as human beings, it is extremely difficult to induce in them the paralytic phenomena characteristic of plumbism. Heubel[1] says that it has hitherto been found impossible to create, in an animal, fully developed lead paralysis. Such is not my experience. It is extremely difficult, I admit, but an animal, after exhibiting symptoms of paresis, may become paralysed in all its limbs, if only time is given. It occurred in a rabbit which had first paresis, then inco-ordination, so that if the animal was laid on its side, it could not rise again; ultimately there appeared paralysis of all its limbs, particularly of the extensor muscles. The ordinary phenomena of lead poisoning are readily induced, but in some animals more quickly than others. The rat is not easily poisoned by lead. Whether this immunity has been acquired by heredity,—rats having for long been known to gnaw through leaden pipes with impunity,—I cannot say. Rats were not easily brought under the influence of lead. The plan adopted was to give rabbits and rats small quantities of lead with their food; rectal injections of a solution of acetate of lead, or of the chloride; hypodermic injections of a 1 per cent. solution of acetate of lead, and the inunction of lead oleate into the skin of the back of the neck, where the chances of the animal rubbing off the ointment by its paws, and eating it, were reduced to a minimum. In nearly every instance the first effect that was noticed was emaciation and anæmia,—both of which were progressive. If, however, the lead was withheld from the food for a day or two, the animals

[1] Pathogen. und Sympt. der chron. Bleivergiftung, p. 28.

soon showed signs of improvement; they began to put on flesh. One or two of the rabbits exhibited a distaste for food. The food consisted of bran mash, and green vegetables principally. Other animals retained their appetite almost to the end. They all drank water greedily. Some remained lively for a length of time; others early lost this natural characteristic, and became dull and apathetic. One or two of the rabbits became extremely nervous, and seemed to be afraid of being handled, but did not resent it, whilst another would not allow herself to be caught. The emaciation was general, but as a rule it appeared most in the muscles, and subcutaneous fat of the back, and the hind legs. Two or three of the rabbits weighed one-fourth less at the time of their death than on the day they first began the eating of lead. The length of time required to develop symptoms of lead poisoning ending in death, varied. A white rat, treated by hypodermic injections of lead, lived from July 1890 until the following January. The shortest period was nineteen days, and this was in a rabbit which had a limited patch of its skin anointed with lead oleate. The longest period during which a rabbit took lead in its food was one hundred and thirty-one days, when we found it blind, and paralysed in its extremities. A guinea pig was found to be less susceptible to lead than the rabbits. It is only when lead is given in very small quantities in the food for an extended period that there is the opportunity for the development of the symptoms of lead poisoning identical with those found in man. It was in the rabbit that lived the longest that there occurred blindness, depending upon neuro-retinitis—paralysis of the extensor muscles of all the limbs, and that albumen was found in large quantity in the urine. Some of the animals suffered from lead colic,—a condition which was noticed by Heubel to be present in a very severe form in dogs fed upon lead. Rabbits and guinea pigs alike miscarried when pregnant, and so violent must have been the uterine contractions that in one case the uterus ruptured, causing death of the rabbit. The others died evidently

in convulsions. How far the emaciation in these animals depended upon disorder of digestion, it is impossible to say. Those that received the lead through the skin, and by hypodermic injection, also rapidly emaciated. Emaciation and anæmia, with deficiency of hæmoglobin, early appeared. The blood exhibited a very rapid tendency to coagulate. These circumstances are of interest when taken in conjunction with the examination of the blood made by Heubel and by Popp. The latter found in the blood of a man, aged 27, suffering from lead cachexia, that the quantity of fibrin was considerably increased, whilst the blood corpuscles had undergone a diminution. Heubel also noticed this tendency for the blood to coagulate rapidly. His analyses of the blood are valuable, as they comprise examinations before and after the digestion of lead. He found that the solid constituents of the blood decreased by 50·6 per 1000 :

	Per 1000.
The blood corpuscles had decreased by .	49·6
Albumen, ...	4·2
Solid constituents of serum, . . .	7·03
Albumen of serum, ...	8·07

whilst the water of the blood had increased by 50·682 per 1000.

	Per 1000.
Extractive substances of the blood by . .	1·49
Water of the serum, ...	7·03
Extractive substance of serum, . . .	1·29

Three further analyses gave substantially analogous results, with the exception that the decrease of red blood corpuscles was less than in the case just quoted. It generally amounted to 20 to 40 per 1000 only, so that briefly what occurred in Heubel's case was a decrease of solid constituents of the blood by 24 to 50 per 1000—a corresponding increase of water—a decrease of red blood cells in the ratio just stated :—a decrease of albumen by 4·5 to 7·5 per 1000, and finally, an increase of the extractive substances and soluble salts.

In the later stages of lead poisoning in animals, just as occurs in man, and particularly where optic neuritis is developed, the pupils are dilated. Constipation was present in most of the animals, but in a few there was diarrhœa. The fæces were generally dark, due to the unabsorbed lead passing out in the form of sulphide. The temperature was, as a rule, normal throughout. In some, it rose a few degrees a day or two before death.

On *post-mortem* examination, generally made immediately, I was always struck by the peculiar odour of decomposition which escaped, and also by the physical signs of rapid decomposition. The blood was greatly diminished in each instance—scarcely more than a few drops being detected. Beyond the extreme emaciation, which affected mostly the muscles and the subcutaneous fat, nothing, as a rule, was observed by the naked eye. The small intestines were pale, generally contracted and empty—the large were discoloured, bluish-black, and contained liquid or solid dark fæces. In every instance the left ventricle was firmly contracted, as in systole, and completely empty, but the auricles and the right side of the heart were full of dark clot. The lungs were in all healthy with the exception of those of the rabbit that died from rupture of the uterus. In her lungs were several discrete hæmorrhages. Beyond the softness of the walls of the stomach, and the presence of a thick layer of tough mucus around the food which lay in that viscus, there was nothing discovered. Beyond pallor, there was nothing noticed in the other organs by the naked eye.

The following represents the amount of lead found in the organs of the rabbits:—

RABBITS.

	Annie.	Mary.	Sambo.	Jimmy.
Muscle, .	...	...	·07	Nil
Liver, . .	·15	Nil	·05	Trace
Small Intestine,	...	...	Nil	...
Stomach, .	Nil	·04	...	...
Heart, . .	...	Nil	...	...
Brain, . .	·02	Nil	...	...
Colon, . .	Nil	...	...	...
Kidney, .	Trace	...	...	...

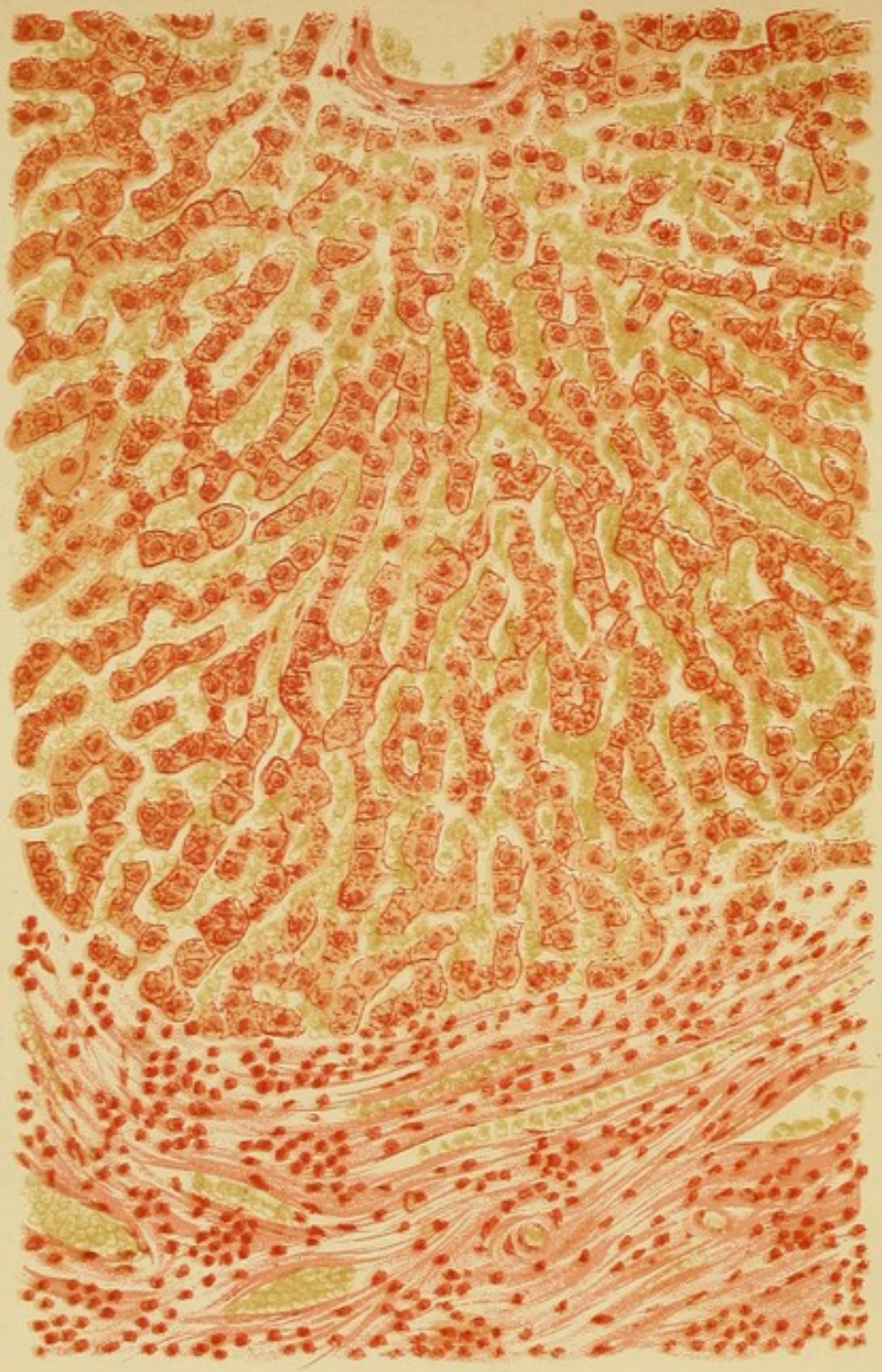


FIGURE XXIV.

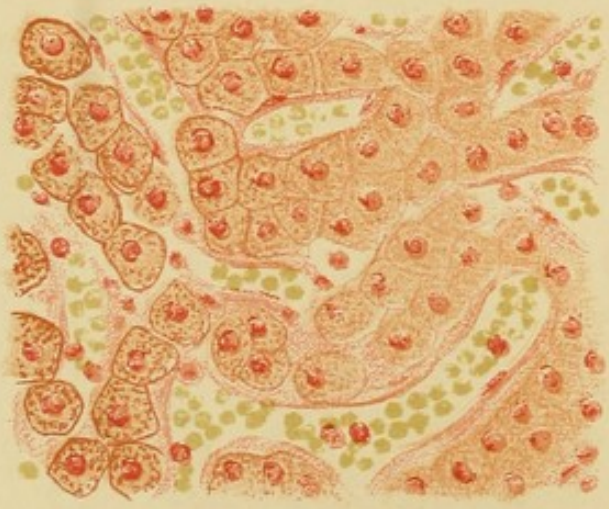


FIGURE XXV.

FIG. 24.—Liver, acute lead encephalopathy. Case of Cath. Y., aged eighteen years. Patient had worked only forty days in lead factory. Cirrhosis already well advanced, hepatic cells shrunken, large blood spaces between the hepatic cells and rows of cells. (× 250 diameters.)

FIG. 25.—Liver, acute lead poisoning. Cath. Y., æt. 18. (× 450 diameters.) Shows the large blood spaces between the hepatic cells and commencing intercellular cirrhosis.

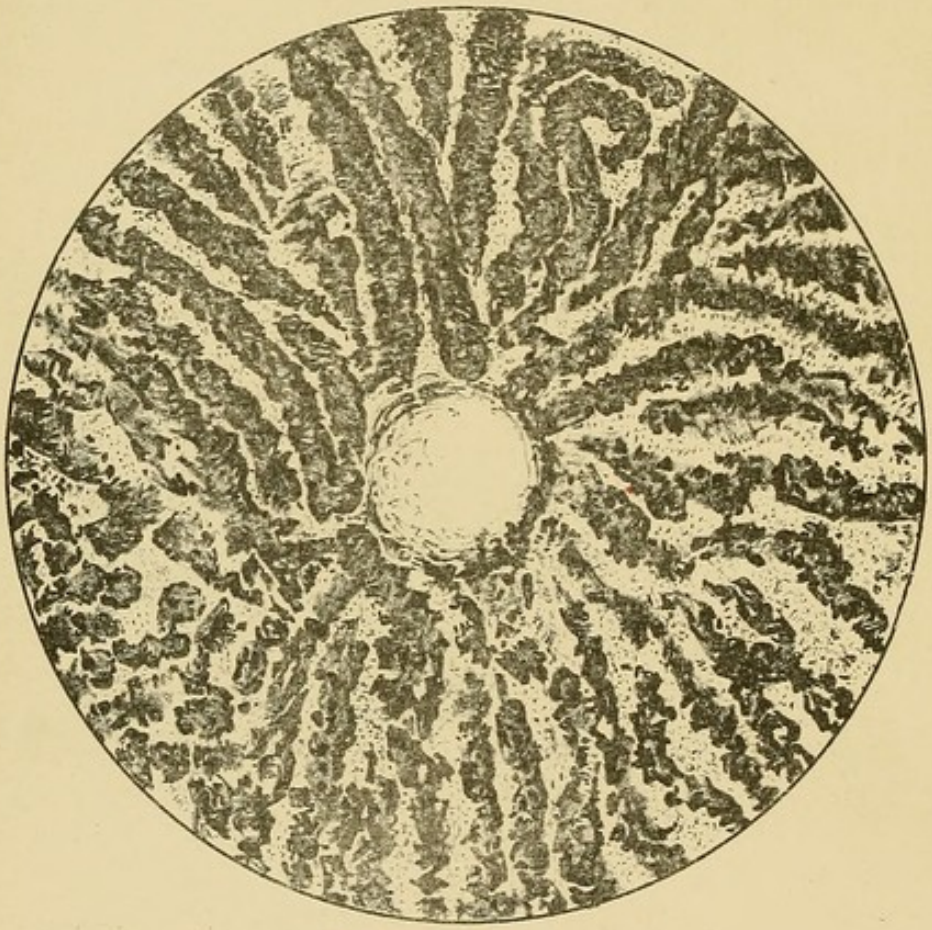


FIGURE XXIII.

FIG. 23.—Liver, rabbit ; lead poisoning shows atrophied rows of hepatic cells.

These results are the total quantities of lead in the whole quantity of each organ sent for examination. They represent milligrammes of metallic lead. It was the rabbit "Mary" that died from rupture of the uterus; she had only taken lead for twenty-three days. Her death, therefore, was not due so much to lead poisoning, as to an accidental and yet related issue of it, hence the absence of lead in her various organs. Whilst lead, however, was practically absent in her tissues, it was found to the extent of ·1 milligramme in the liquor amnii, and to the extent of ·09 in each of the five fœtuses found in her uterus. The blood, just as in man, contained only a trace of lead, and a very small quantity of urea. In the urine of one of the rabbits, urea equivalent to 23·64 grains per ounce was detected.

Setting aside for the moment microscopic examination of the stomach and intestines, the lungs and the heart, the spleen, brain and spinal cord, as these failed to reveal anything abnormal, I would invite your attention briefly to a consideration of the microscopical examination of the liver and kidneys. It is in these organs, as in man, that I believe there principally arise these changes which largely determine the events that occur in plumbism.

In the liver of animals, and in the liver of lead workers, there will be found an atrophied, cloudy, or granular condition of hepatic cells, amounting at times to a true fatty degeneration. The columns of cells become irregular and slender, and thus become separated from each other by spaces of considerable size. In nearly all the specimens taken from human beings and ruminants, these spaces between the individual hepatic cells and rows of cells were occupied by small round cells. There was an increase of connective tissue elements, derived in all probability from the capillaries or the sheath which surrounded them. In other words, there was an intercellular cirrhosis, not unlike that met with in congenital syphilis. The capillaries of the lobules were generally found to be over-distended, a condition observed

nearer the centre than the periphery. Interstitial inflammation, with degeneration of columns of the liver cells, was so generally present in all the specimens, that I cannot but regard this as a constant lesion, and as one likely to arise in the early stages of lead poisoning.

The kidneys presented alterations not less interesting. Pathological opinion in regard to the relation of kidney lesion and lead poisoning has been too stereotyped. A few years ago, I drew attention to the fact, that interstitial nephritis was not the uniform lesion met with in lead poisoning, that whilst it was the structural alteration met with in the late stages, a parenchymatous nephritis running on to interstitial was really the order of events. Recent observations based upon experimental investigation and human pathology, have only confirmed that opinion. In the early stages of lead poisoning, the renal epithelia of the convoluted tubules become enlarged, their protoplasm exhibits cloudy swelling, and becomes fatty-granular; the cells soon break down, their débris being found in the interior of the tubules. There is, however, not the rapid shedding of cells seen in ordinary cases of desquamative nephritis. It is noteworthy, that whilst the renal epithelia of the convoluted tubules thus exhibit alteration, those of the straight tubules of the medulla exhibit almost no change, from first to last. I have observed this early stage of parenchymatous nephritis in girls who have died from lead encephalopathy; it was also observed in rabbits. In addition, there is generally to be detected an increased leucocyte exudation underneath the capsule of the kidney, and the same is noticed around the afferent vessels of the Malpighian glomeruli. This accumulation of small round cells around the entering vessel of the glomerulus is very interesting, as I found it in nearly every instance, and with the alteration next to be described, seemed to me to be uniformly present in lead poisoning. Bowman's capsule becomes thickened and laminated, and the cells composing its internal lining are seen to have multiplied greatly, and to lie in irregular heaps at places

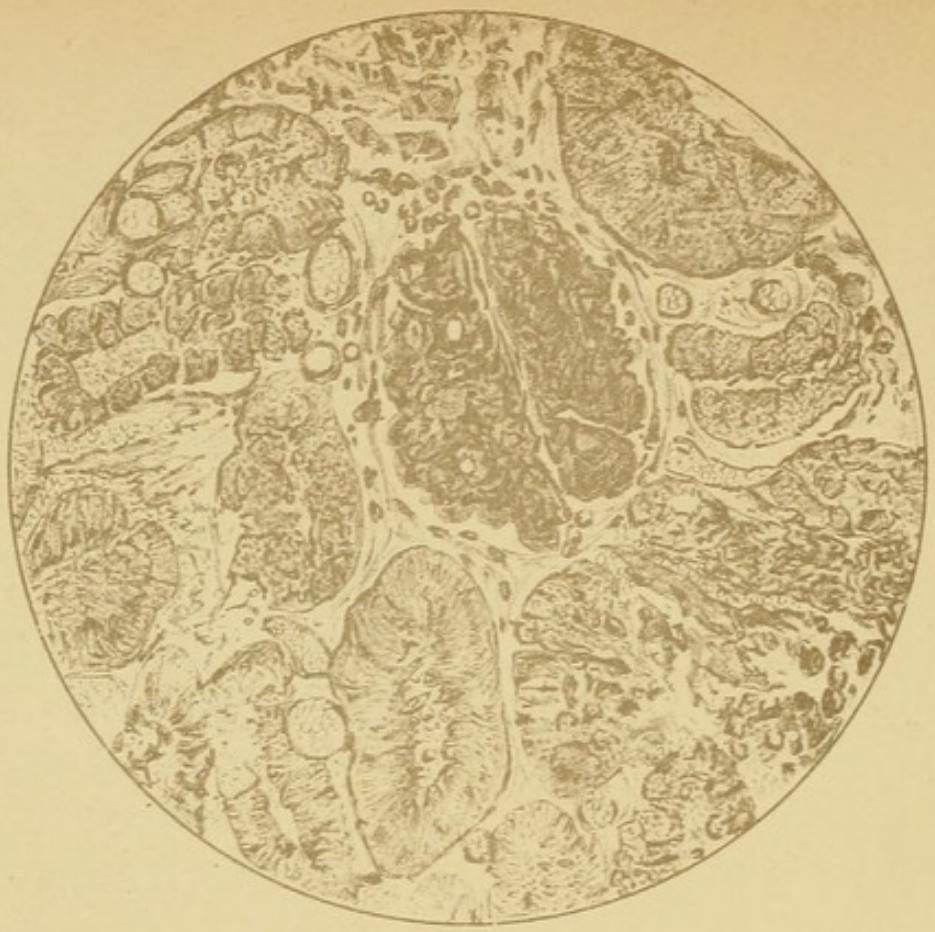


FIGURE XXVIII.

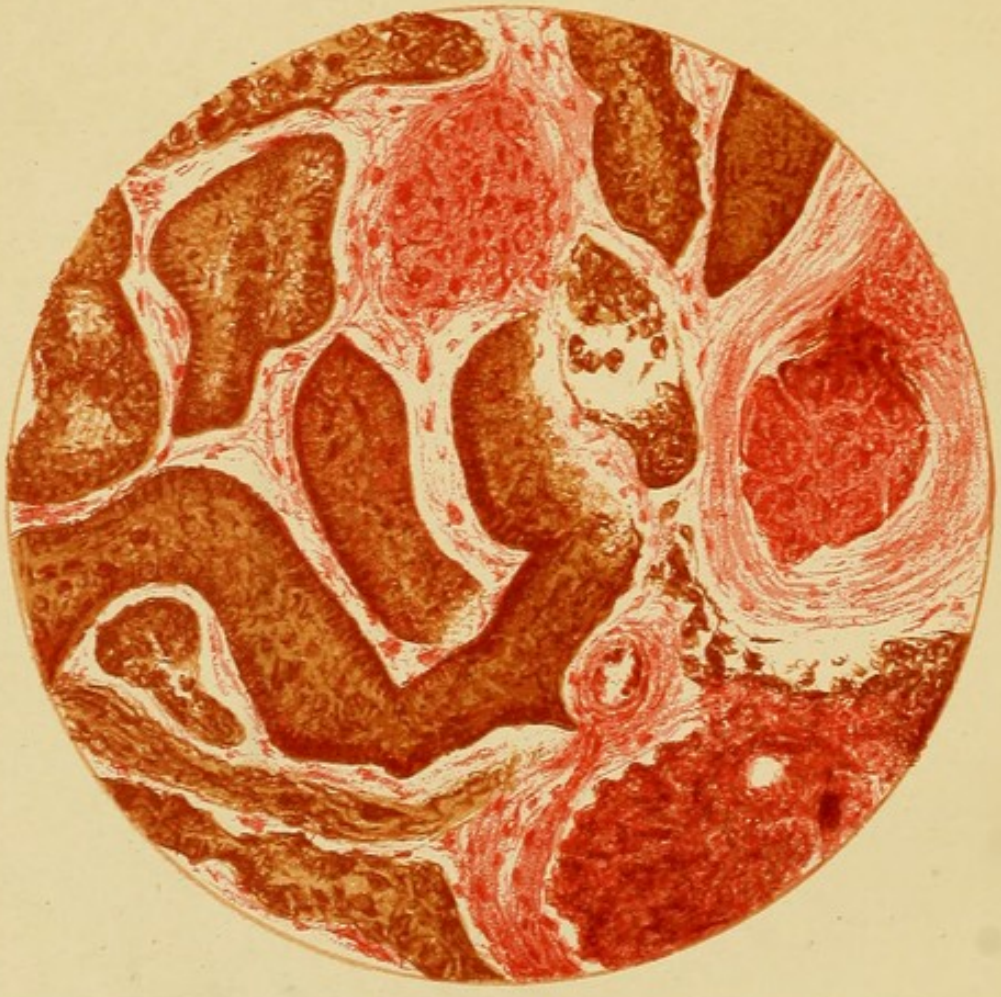


FIGURE XXIX.

Fig. 28.—Acute nephritis—tubal and interstitial—marked increase of small round cells around afferent vessel of glomeruli, also in Bowman's capsule, and glomerular tuft of vessels. Case of Eliz. H., Acute lead poisoning. (Crouch, one-sixth inch.) Early stage.

Fig 29.—Kidney in lead poisoning. Parenchymatous and interstitial nephritis ; shows thickening and the laminated appearance of Bowman's capsule, with accumulation of leucocytes around afferent vessel of glomerulus. (Crouch one-sixth inch.) Case of Barbara R.

FIGURE XXVI.

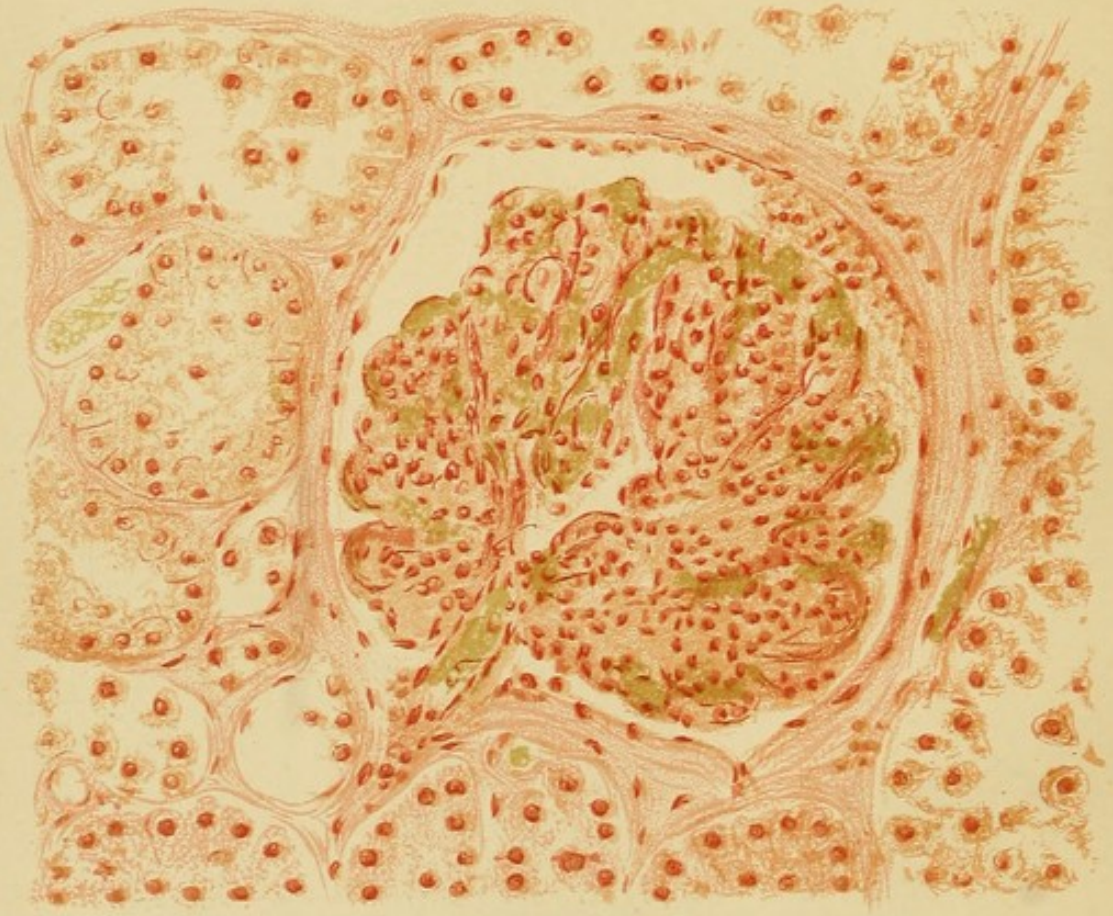


FIGURE XXVII.

FIG. 26.—Lead poisoning, experimental. Acute tubular nephritis fatty granular degeneration of epithelia, thickening of Bowman's capsule. Kidney of rabbit.

FIG. 27.—Kidney, acute lead poisoning. Cath. Y., æt. 18, had worked only forty days in lead factory. Tubal nephritis, also glomerulo-nephritis and increase of leucocytes inside Bowman's capsule, very noticeable at upper and right side of drawing. (× 250 diameters.) Very early stage.

within the capsule. The capillaries in the glomerulus are noticed to be at places distended with blood cells, and at others constricted, whilst in not a few the thickened capsule has pressed so completely against the coil of capillaries, that they appear attenuated and shrunken. In addition to the increase in the number of cells lying within the capsule of Bowman, the cells which lie between the coils of capillaries are seen to have undergone extensive proliferation. Where accumulation of leucocytes has ocurred around the afferent arteriole of the glomerulus, the cells on the outside of Bowman's capsule are also noticed to have increased in number, and from this as a focus they seem to have penetrated between the neighbouring convoluted tubules, causing a distinct increase in the connective tissue framework of this portion of the kidney. Lying in some of the tubules may be observed homogeneous and granular tube-casts. From the first, therefore, I would say that lead poisoning produces an acute parenchymatous nephritis, as observed in the cloudy and fatty granular degeneration of the renal cells of the convoluted tubules, and, concurrently with this, a proliferation of cells around the afferent vessels of the glomeruli, also of those inside Bowman's capsule, and in the inter-capillary spaces within the glomeruli. After this has existed for a time, a true interstitial nephritis is developed, in which, not only is the interstitial tissue increased, but the walls of the small blood vessels are thickened; the walls of Bowman's capsule also become thickened and laminated, and the glomeruli appear to be compressed. As these are, apparently, the primary conditions occurring in the early stages of acute lead poisoning, alike in man and animals, I regard them as characteristic of plumbism.

Upon the liver and kidneys must fall primarily the brunt of the effects of lead, absorbed from the stomach. Lead acts directly upon hepatic cells; some lead is eliminated, but very little, by the bile; part is deposited in the liver tissue, generally a good deal. Circulating in the blood, lead is being constantly eliminated by the kidneys; the urine of patients suffering from

lead poisoning always contains a quantity of lead. Thus, owing to the peculiar function which is thrown upon the protoplasm of the renal cells, the cells soon degenerate, and owing to the irritating action of the lead, as it circulates in the blood-vessels and through the glomeruli, there is hyperplasia of the surrounding connective tissue. The strain upon the kidney from the first is great, owing to the increased diuresis which is present for a period, but as the disease advances, and involves all the elements of the renal tissue, urinary secretion diminishes, frequently ceasing, as we have noticed in the terminal stages of the malady. So long as the kidneys are eliminating lead, the patient runs little risk from poisoning, but when the kidneys become defective, the individual is no longer free from all the risks of plumbism. It was Bouchard who showed that the urine eliminated by lead workers in a state of comparative health, when injected into the veins of animals, had high toxic properties, but that when such individuals become convulsed, the urine then withdrawn from the bladder was found to have largely lost its toxic power,—a circumstance clearly showing that the poison had been retained in the system, and was doubtless acting as a cause of the illness. It is difficult to say exactly how lead acts upon the normal structure of secreting cells. Potter and I found that when amœbæ were brought into contact with a weak solution of plumbic acetate, say 1 to 10 per cent. solution, that almost immediately the active movements previously exhibited by the organisms ceased. The amœbæ at once became suddenly elongated and seemed stiffened, and if carefully watched, no further movement would occur. We were able, however, to poison amœbæ for a period, for if we quickly removed, by washing, the lead solution with which the amœbæ had become surrounded, movements would return, and the organisms would be found active next day. If, on the other hand, a weak solution of lead remained in contact with amœbæ for several minutes, no amount of washing was capable of restoring lost vitality. What impressed us in these investigations, was the extreme rapidity with which

such low organisms became affected by weak solutions of lead.

Mr Potter's botanical observations respecting the effects of lead on plants, induced by a soil moistened by lead solution or by means of hypodermic injections into the stems, show that nutrition and flowering were interfered with, and that, just as in man, their reproductive force was more or less destroyed. When he injected 1 cc. of a 1 per cent. solution of lead nitrate into Datura Stramonium, he noticed that the flowers on the branches thus injected fell off, whereas the flowers on the other branches, which had not been touched, continued to bloom and reached maturity. In a paper on the investigation of the poisoning of plants by arsenic, lead, and zinc, Nobbe, Baessler, & Wille[1] mention similar interesting effects noticed as the result of lead nitrate and carbonate.

In one experiment twelve new peas of equal size and of similar appearance, growing in three-litre cylinders, received four separate doses of lead in increasing quantities, thus:—

I.	II.	III.	IV.
3.3	33.3	333.3	1,000 mg. per litre.

The experiment was begun 15th June 1881 at 4.30 P.M.

At the commencement of the experiment the plants averaged in height 160 mm., had from 5 to 6 internodes, and were well developed; the roots were white.

16*th June*, 8 A.M.

I., II., and III. are unchanged.

IV. The four bright leaves are withered; two internodes are bent.

12 Noon. Bending of internodes less.

3 P.M. All the plants are fresh.

17*th June*, 8 A.M.

Plants remain unchanged; fresh.

3 P.M. Plants remain unchanged; fresh.

[1] "Die Landwirthschaftlichen, Versuchs stationen Heft," 5 and 6, 1884.

18*th June*, 8 A.M.

I. to IV. unchanged; fresh roots.

6 P.M. Plants unchanged; a new leaf has developed on each.

21*st June*, 5 P.M.

I. and II. Decided additional growth; two new leaves; plant fresh and unchanged.

III. and IV. lag behind I. and II. as regards apparent growth; only one leaf in each has developed, the other leaves are pale and of a light yellowish green. No additional growth to roots.

26*th June*.

I. Increase in height since 15th June equals 240 mm. Three new leaves and two flowers have been produced; leaves in general are pale yellow; roots show scarcely any additional growth, are coloured yellow, but still remain fresh.

II. Increase in height 260 mm. Three new leaves; two flowers; colour of leaf unchanged; roots yellowish; no additional growth.

III. Appears to have begun to die; a crumpled new leaf has developed. Increase in height 110 mm. Leaves in general have faded, are yellowish grey or greenish yellow. Roots exhibit no increase in growth; are apparently fresh.

IV. Is dead. Lower part of leaves and stem pale yellow or yellowish brown; upper part yellowish green. The stem is bent and very weak. Additional growth 30 mm. Roots flabby but fresh.

5*th July*.

I. Total height 270 mm.; increase since 26th June, 30 mm. The two flowers have been fertilised. Another leaf has formed. Roots as on the 26th June.

II. No additional growth since 26th June. Two flowers fertilised; leaves in general green. The yellowish

roots exhibit no additional growth; growing point dead and glassy.

III. Dead; internodes dry and crumpled. General colour pale yellow; leaves are dry; roots apparently fresh; no additional growth.

11*th July.*

I. No additional growth; growing point weak. Pods measure 50 and 36 mm. respectively; contain two seeds—one well developed; roots are fresh.

II. No additional growth. Two pods, with one seed in each; colour of leaf normal; a little withered.

1*st August.*

I. Upper part of plant dead; seeds are ripe. No additional growth.

II. Upper part of plant dry and withered; pale and yellowish brown; no additional growth. Fruits have matured one seed each. Roots are evidently dead.

These observers state that, just as occurs with arsenic, there was noticed, upon the addition of a strong dose of lead, withering and bending of the internode. This in some hours disappeared. Weaker doses of lead acted only by retarding growth. In a similar series of experiments made with weaker doses of lead upon Indian corn grown in a solution of lead nitrate, it was found that where the stronger solutions of lead were employed, fewer leaves developed, and the mass of tissue was, structurally speaking, more or less degraded. That plants are capable of absorbing lead by their roots has been shown by Professor F. C. Phillips, in a paper read, in February 1890, before the Society of Engineers, West Pennsylvania. Phillips says that "lead enters the tissue of the plant, but without causing any disturbance in the growth, nutrition, or functions of the plant." With the results of other observers before us, and the effects noticed by Potter and myself upon plants and amœbæ, I cannot but regard lead as poisonous both to plant and animal life.

How plumbism is brought about in man and animals it is difficult to say. It seems to me that it primarily affects the organs of elimination such as the liver and the kidneys, and whatever consequences follow, they are due to the presence in the blood of secondary poisons arising out of the imperfection of liver and kidney function. The first alteration in structure occurs, in my opinion, in these organs. Hence, the early effect produced by lead upon nutrition and blood formation.

At this stage of our inquiry, let us consider for a moment the influence of lead upon metabolism. We have seen how very early a symptom anæmia is. It is progressive up to a certain stage. It depends rather upon a fault in the formation of blood than in disintegration thereof. Most writers have observed a deficiency of urea and uric acid in the urine of those suffering from lead poisoning. Sir Alfred Garrod's observations on this point have been repeatedly confirmed. Haig alludes to the deficiency of uric acid in lead poisoning, and nearly all writers speak of the association of lead poisoning with gout. Garrod found that 33 per cent. of those who suffered from gout had been poisoned by lead, and Duckworth observed that of 136 cases of unequivocal gout in both sexes, 18 per cent. presented signs of lead impregnation. In the London Hospitals, the administration of a few grains of acetate of lead to those who have been previously exposed to the influence of the metal has, according to Dr Lauder Brunton, been followed by pains of a distinctly gouty nature. We do not see in the north this intimate relationship between gout and saturnine poisoning. I have noticed the association, but it has generally been in subjects with a hereditary taint of lead poisoning. Workmen from the south develop it in the north of England. The natives of the north, though equally exposed, seldom become gouty even when the kidneys are affected. For the same reason, we rarely meet with the small red, gouty kidney. To prevent misunderstanding, it is necessary to say, that gout, as a disease, is not met

with in the north with anything like the frequency that it occurs in London. The absence of gout in Newcastle and Edinburgh has been ascribed to the difference in the drinking habits of the people—whisky and not malt liquor being the general drink. I cannot regard this altogether as an explanation, nor can I offer any other in its place; in some way or other, but probably as the result of external conditions rather than as depending upon the use of beverages, gout is little known in the north as a symptom of lead poisoning.

Sir Alfred Garrod draws attention to the fact that uric acid is not only present in the blood in excess in lead poisoning, but that when lead is given medicinally, it diminishes the excretion of uric acid in the urine. Further on[1] he says: "It would appear, therefore, that in an individual impregnated with lead, the blood becomes loaded with uric acid, not from its increased formation, but from its imperfect excretion." Haig[2] assumes the same position, and maintains that there is no evidence to show that there ever is an excess of uric acid in the blood, due to increased formation. To him, urea and uric acid stand in a definite relation to each other, the average being one grain of uric acid to every 33 grains of urea. Urea is a very soluble substance, and whilst it can be eliminated with almost absolute certainty, the insoluble uric acid is apt, from various causes, to lag behind, and be retained in the body. When uric acid is deficient in the urine in lead poisoning, it is not meant altogether by these observers that it is in excess in the blood. It may ultimately be present in excess in the blood, but this is due to the fact that, under the employment of alkalies, the uric acid which was retained in the tissues, owing to the action of lead, has passed in solution into the blood, and, when present there in excess, passes in excess into the urine. In other words, the amount of uric acid in the urine, is the measure of uric acid in the blood: if in the

[1] Garrod, Gout and Rheumatic Gout, p. 243.

[2] Some investigations regarding uric acid, St Barth. Hospital Reports, 1890.

blood, it will be thrown out by the urine, and the one circumstance, in particular, which determines the amount of uric acid thrown out is the alkalinity of the blood. Whatever diminishes the alkalinity of the blood and fluids generally, will favour the retention of uric acid in the tissues, for uric acid, being insoluble, generally leaves the body in the form of urate of ammonia and soda. Whenever, therefore, the administration of medicines is followed by increase of uric acid in the urine—by an increase far in excess of the normal—the explanation, in all probability is, that the insoluble uric acid, which had been deposited in the tissues, around the joints, in the liver, and in the spleen, has been rendered soluble and is being thrown out in the urine. On the other hand, lead, by diminishing the alkalinity of the blood, as Ralfe has shown, lessens the solubility of uric acid, and must therefore diminish its excretion in the urine. Haig, basing his conclusions upon the relationship of uric acid and urea as being 1 to 33, has shown that on the first day after lead has been taken, the relationship became 1 to 51.

The absence of gout amongst lead workers in the North of England is a point of great interest, physiologically as well as pathologically. Are we perfectly sure that lead *per se* determines gout? Sir Wm. Roberts has not only coined the word "uratosis," to designate the precipitation of crystalline urates in the tissues or fluids of the body, but appears to me to have raised a most important question. "It is not easy," he says,[1] "to believe that lead poisoning produces really and truly the same constitutional diathesis as that which exists in true gout. . . . The gouty diathesis and lead poisoning, while differing in all other respects, have one tendency or vice in common, viz., the tendency to uratosis." He regards uratosis therefore as a gouty tendency which may be reinforced by lead poisoning, and saturnine uratosis as one in which saturnine poisoning is reinforced by a previously existing gouty tendency. My observations go strongly to support this conclusion.

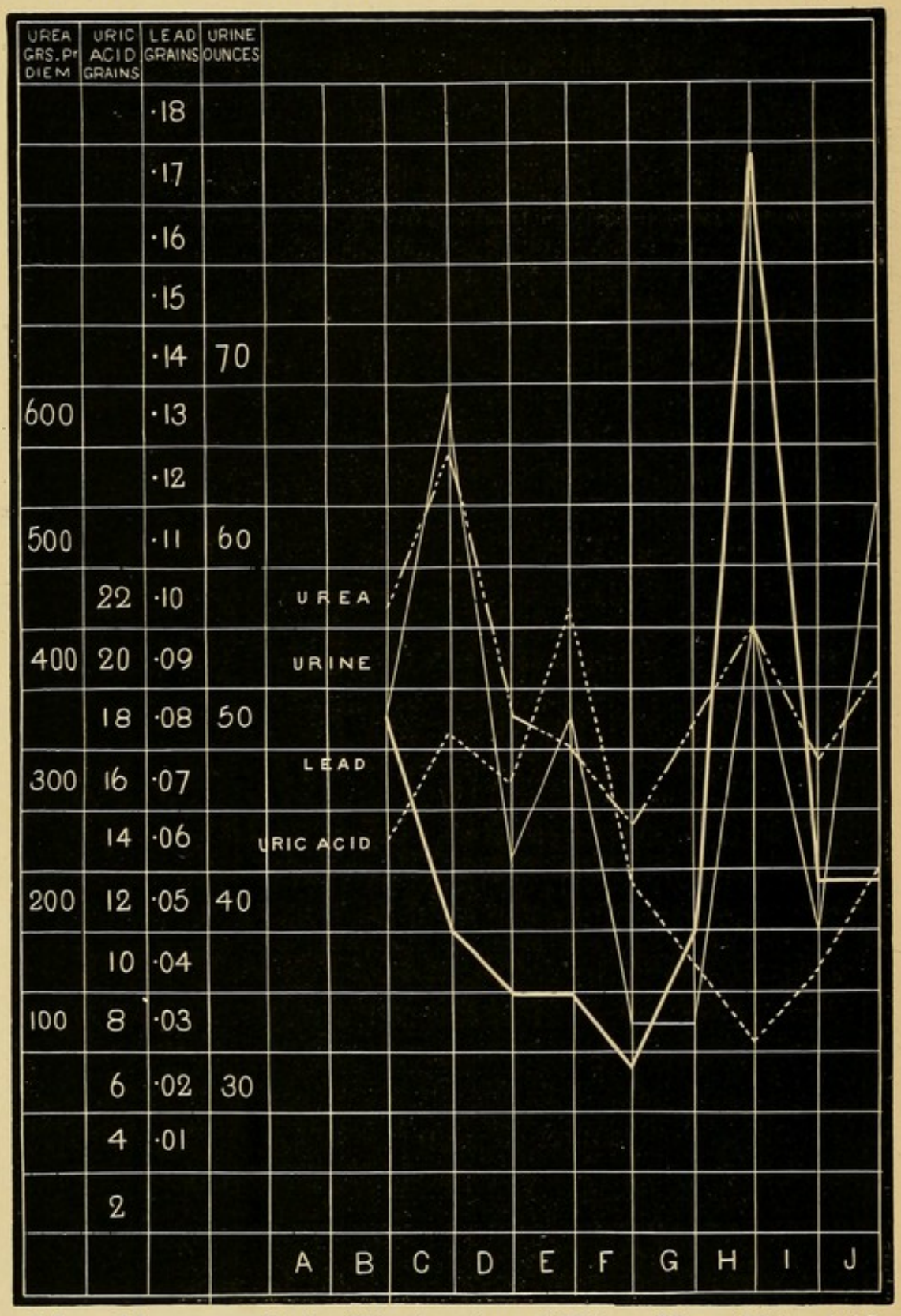
[1] British Med. Journ. 1890, vol. ii., p. 1164.

As Haig's observations extend over a period of only a few days, I should like to place before you the analysis of the urine of lead workers extending over a few weeks, and where, as the lead salt manipulated was the carbonate, there was no intestinal irritation capable of disturbing the elimination of uric acid which Haig observed after the administration of lead acetate.

The subjoined table shows the daily elimination of urea, uric acid, and lead in a lead worker, and also indicates the extreme acidity of the urine in plumbism. Sarah A. C., aged 36 years, was admitted into the Newcastle Infirmary, suffering from double "wrist drop," and pains in her knees.

1891.	Quantity of urine daily in ounces.	Sp. gr.	Daily Acidity.	Excess of Acidity over Normal.	Urea in grains.	Uric acid grains.	Lead in grains.
Jan. 4	59	1009	...	...	285.5	...	...
,, 5	59	1012	35.8	5.8	312.7	...	...
,, 6	74	1008	90	66	392.2	...	...
,, 7	60	1010	97.2	67.2	318	...	...
,, 8	68	1014	109.7	79.7	397.12	...	...
,, 9	73	1012	88.7	58.7	341.1	...	...
,, 10	54	1014	116	86	286.2	...	...
,, 11	56	1015	90.8	60.8	327	...	...
,, 12	72	1010	87.5	57.5	381.6	...	...
,, 13	78	1010	173.9	143.9	335.4	...	...
,, 14	60	1012	97.2	67.2	291	...	...
,, 15	62	1010	88.7	58.7	328	5.23	.00386
,, 16	64	1010	103.7	73.7	343	7.25	.00307
,, 17	78	1014	96	66	417	5.84	.00374
,, 18	68	1012	82.6	52.6	395.7	5.09	.00651
,, 19	69	1010	83.5	53.5	467.1	3.31	trace.
,, 20	72	1012	96.2	66.2	488.8	2.07	trace.
,, 21	100	1012	66.8	36.8	665.4	.76	...
,, 22	80	...	...	...	...	.13	nil.
,, 23	88	1012	30.2	.2	469.7	.50	.00957
,, 24	82	...	...	...	...	1.89	.00394
,, 25	105	1010	neutral.	...	438.9	2.01	.00756
,, 26	68	1012	22	dec. of 8	338.6	.26	.00153
,, 28	86	1010	45.3	15.3	582.8	...	..
,, 29	112	1010	86.3	56.3	555.5	...	...
,, 30	80	1010	97.3	67.3	418.17	...	...
,, 31	106	1010	56.1	26.1	666.7	...	...
Feb. 1	84	1010	90.2	66.2	569.1	...	...
,, 2	88	1010	58.8	28.8	502.4	...	...
,, 3	92	1010	80.2	50.2	436	...	..
,, 4	96	1012	77.6	47.6	628.8	...	...
,, 5	106	1012	34.3	4.3	704.9	...	...

The determination of the daily acidity is based upon 30 grains



Mary M——.—*Daily Elimination of Urine, Urea, Uric Acid, and Lead by the Kidneys.*

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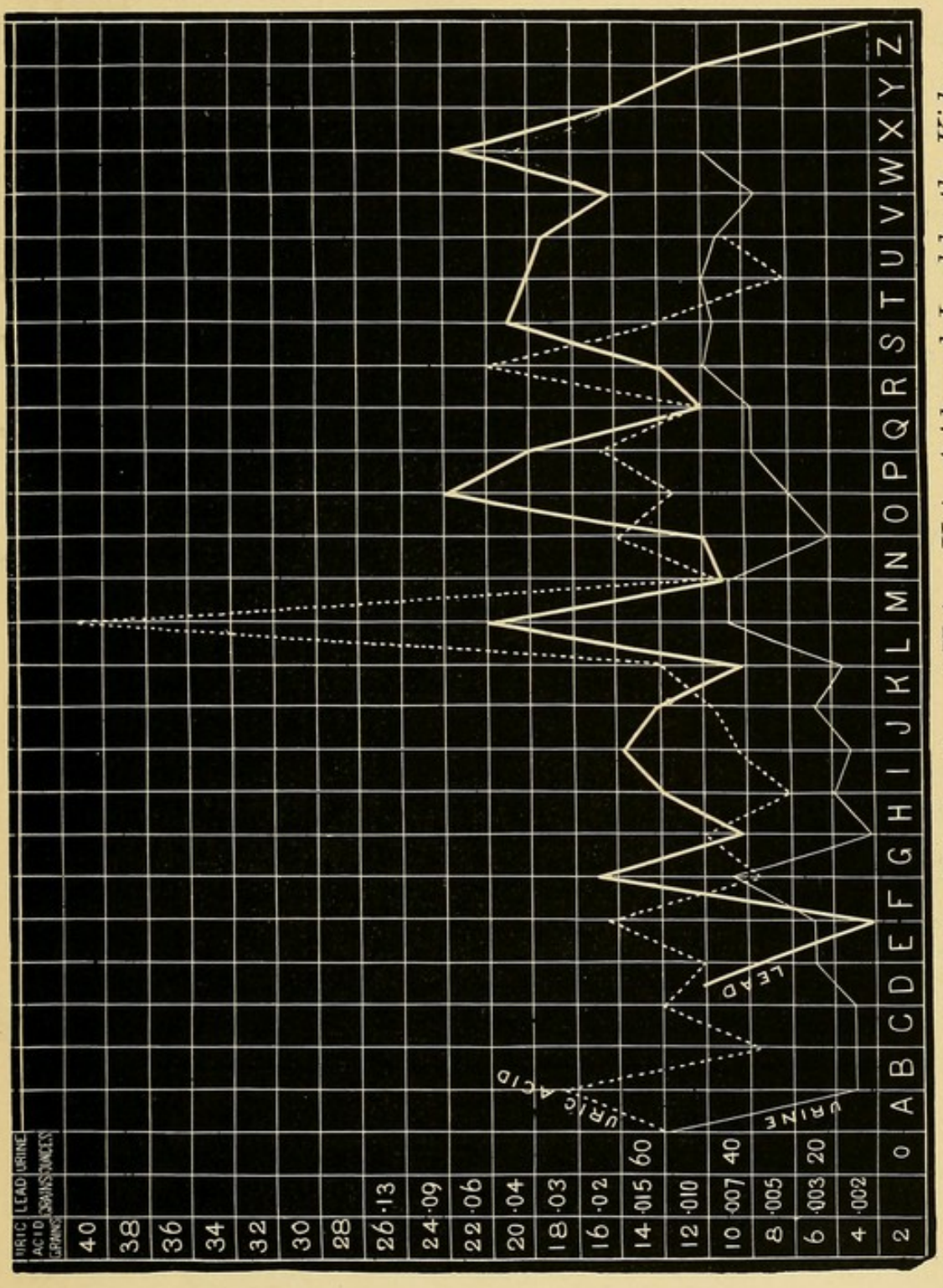
of oxalic acid,—taking that as representing the normal daily

acidity. Carbonate of lithia was given to this patient on the 13th January, and soon afterwards it is noticed that the



Elizabeth B——.—*Daily Elimination of Urine, Uric Acid, and Lead by the Kidneys.*

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daily elimination of urea increased; the proportion of uric acid all through the period of observation is small,—a point of

interest when viewed in the light of statements made by some authors, that the daily acidity of the urine may be regarded as a measure of the amount of uric acid in the urine. Another point of interest, and of some value in this patient's case, is that on the days she passed the largest quantities of urea she was quite free from headache. This relation of headache and deficient urea elimination, and the immediate disappearance of headache when the daily discharge of urea increased, were noticed so frequently in cases of lead poisoning, that it must be more than a simple coincidence. Headache is a symptom depending upon excess of uric acid in the blood, according to Haig, but in the case of Sarah A. C., whose uric acid elimination was small, the severe headache complained of was always found to be associated with a deficiency of urea.

In the case of Mary M., the uric acid elimination was always high: once, only, did it reach the normal 6.5 grs., whilst on another occasion 21 grains of uric acid were passed in the day, the urea on the same dates being 400 and 300 grains respectively. The elimination of lead seemed to stand in an inverse relation to uric acid, as if the presence of lead in the blood favoured the retention of uric acid in the tissues. In the case of Eliza B., this relation is not so noticeable, whilst in the case of John K., who was the subject of acute lead colic, the uric acid eliminations are seen to vary considerably, —occasionally they are observed to run up to 12, 13, and 16 grains per diem, without any explanation, and appear to be inversely related to the elimination of lead. In a lead worker, Mrs St G., admitted suffering from rheumatism, the daily elimination of uric acid was extremely small: it varied from .19 to 3.6 grains.

In regard to the amount of phosphate thrown out in the urine in lead poisoning, I have not observed anything worthy of more than passing mention: the estimates given are of phosphoric anhydride. Taking the daily average of phosphoric acid in the urine as 30 to 50 grains, we found in John K., the red

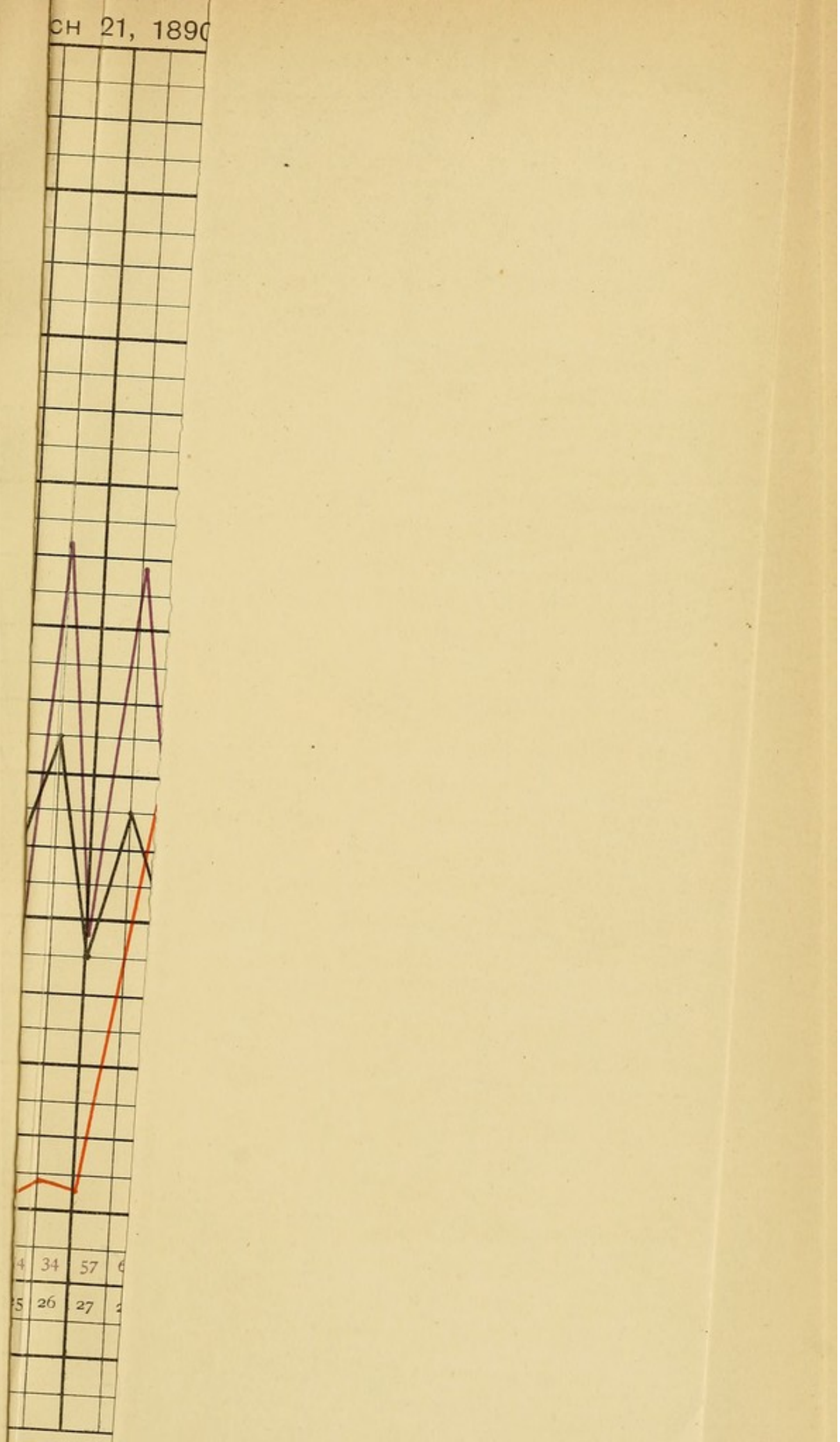
CH 21, 189
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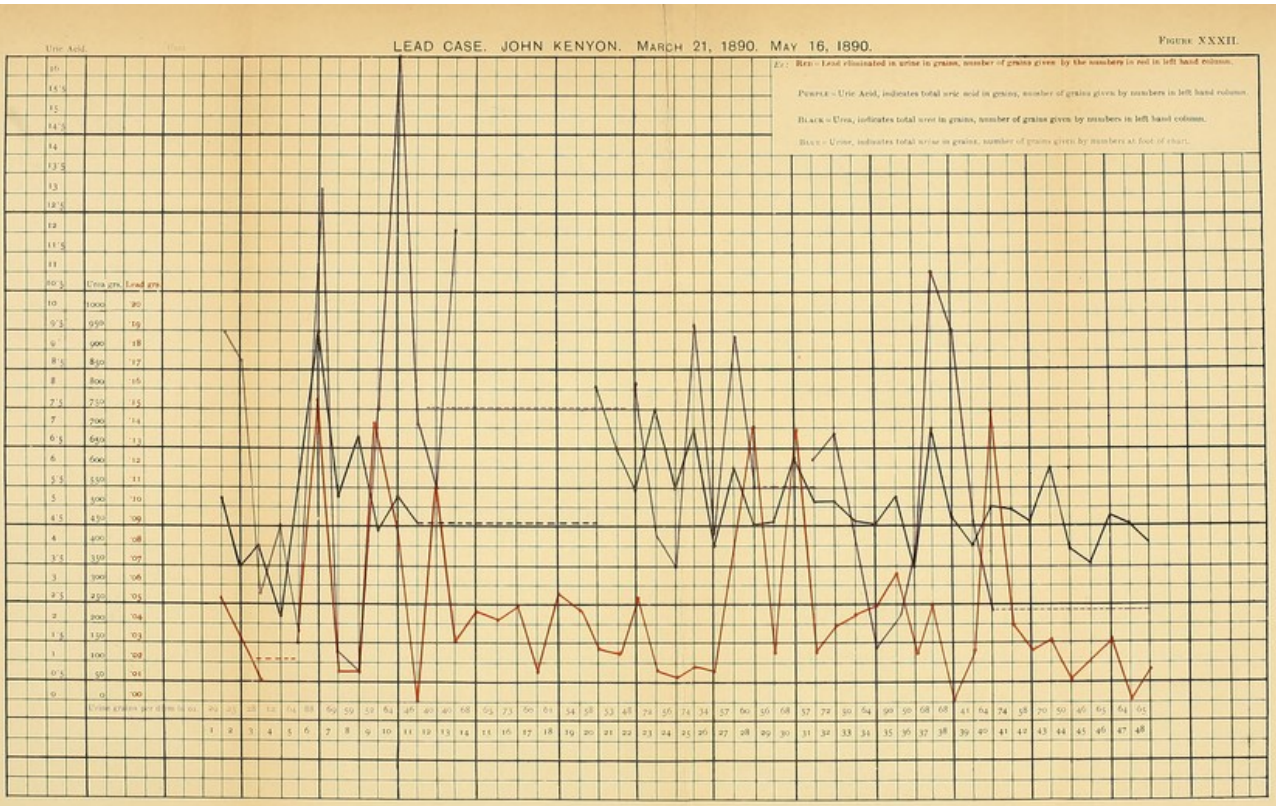
FIGURE XXXII.



lead worker whose case is reported at page 84, the following:—

Sample of urine 30 gave average of 25.84 grains as daily elimination.							
,,	,,	31	,,	,,	44.17	,,	,,
,,	,,	32	,,	,,	77.76	,,	,,
,,	,,	33	,,	,,	48.8	,,	,
,,	,,	34	,,	,,	30.7	,,	,
,,	,,	35	,,	,,	43.2	,,	,,
,,	,,	36	,,	,,	26	,,	,,

From the analysis of the urine in lead poisoning we cannot state that any absolute relationship exists between the elimination of urea, uric acid and lead. Occasionally it appeared as if uric acid and lead stood inversely related to each other. What these analyses show is that metabolism is much deranged, and as nearly all cases of lead poisoning, both in the stages of colic and encephalopathy, exhibit extremely deficient renal function, I cannot but regard many of the symptoms of plumbism as due to the retention in the system of animal poisons, depending upon imperfection of liver and renal function caused by lead.

It is a well known fact that intemperance and the use of alcohol determine attacks of plumbism. Combemale and Francois[1] have shown how very susceptible dogs are to the influence of lead, and how very rapidly nerve symptoms are developed in them. They become frightened, and run off at the approach of any one. They have hallucinations and illusions of sight, are extremely liable to become epileptiform or exhibit choreiform movements, limited to one half of the body. When the animals presented choreiform movements, they generally succumbed rapidly, even if lead was no longer given to them in their food.

The interesting point, however, in their paper is the influence shown by alcohol in precipitating epileptiform seizures. When they had fed animals up to the point at which nerve symptoms

[1] Récherches experimentales sur les phénom., &c., dans le saturnisme chronique.

were about to arise, the admixture of alcohol was always followed a few hours afterwards, either by indications of great fear or epileptiform attacks, which would be repeated for two or three days and then disappear, although alcoholic intoxication was not suspended.

Case—Eliz. T., reported page 71.

Name of organ.	Total lead in parts per million.	Weight of organ.	Grains of lead per weight of organ.
Lung, . . .	7·6	29·0 ounces	0·0964
Heart, . . .	4·12	10·5 ,,	0·0189
Liver, . . .	37·8	60·5 ,,	1·000
Spleen, . . .	12·0	6·5 ,,	0·0341
Kidneys, . .	10·0	5·25 ,,	0·0229
Cerebrum, . .	9·8	51·5 ,,	0·779
Cerebellum, . .	24·8		
Pons, . . .	22·6		
Spinal cord, . .	1·16		
Large intestine, .	37·7		

	Alcoholic extract, lead in milligrms.	Ethereal extract, lead in milligrms.	Aqueous extract, lead in milligrms.	Ash, lead in milligrms.	Total lead in milligrms.	Lead, parts per million.
Pons, . . .	0·14	0·35	0·0	6·1	0·59	22·6
Cerebellum, . .	0·25	0·4	0·0	1·15	1·80	24·8
Brain, . . .	0·3	0·0	0·0	1·35	1·65	9·8

Alcohol was not the only determining cause of these events; fear, a prospect of chastisement, and a simple catheterisation induced them—circumstances which support the opinion I expressed in a previous lecture, as to the influence of grief and hysteria in inducing saturnine epilepsy. These observers lay considerable stress upon the influence of drunkenness in causing

convulsions in plumbism. In one of my guinea pigs, the administration of small quantities of alcohol with lead in the food undoubtedly precipitated death.

If there is one class of people, therefore, to whom the results of clinical experience and experimental research forcibly appeal, it is lead workers; to them alcohol is a poison. It must in many instances have determined epileptiform seizures,

Case—Cath. H.

Name of organ.	Total lead in parts per million.	Weight of organ.	Grains of lead on total weight of organ.
Heart, . . .	0·5	7·5 ounces	0·0016
Liver, . . .	41·6	45·0 ,,	0·819
Kidneys, . .	13·3	4·5 ,,	0·0261
Spleen, . .	39·0	5·0 ,,	0·0883
Cerebrum, . .	21·6		
Cerebellum, . .	8·59		
Brain with cerebellum, .	30·19	48·0 ,	0·634

	Alcoholic extract, lead in milligrms.	Ethereal extract, lead in milligrms.	Aqueous extract, lead in milligrms.	Ash, lead in milligrms.	Total lead in milligrms.	Lead, parts per million.
Brain, . . .	0·6	0·6	0·91	1·3	3·41	21·6

terminating fatally, which complete abstinence would either have postponed or altogether prevented.

After death from plumbism, lead has been found in the brain. In one of my cases Professor Bedson found 4.04 milligrammes of metallic lead and 3.41 in another. It is impossible to say in what chemical form the lead is present in brain tissue, so I have represented the amounts in terms of metallic lead. In a case reported by Mr Wynter Blyth, 99.7 milligrammes of sulphate of

lead were found in the cerebrum and 17.4 in the cerebellum. Gowers, in his "Medical Ophthalmoscopy," mentions a case of Atkinson's in which five grains of lead were found in the brain. This is the largest quantity that I have seen reported.

The presence of lead in the brain is supposed by many writers to be the explanation of the convulsive seizures and death in saturnine poisoning. It is owing to some peculiar complex arrangement entered into between lead and the tissues of the nervous system, but particularly of the brain—a replacement of hydrogen in a molecule of kephalin by lead—that Wynter Blyth[1] attributes the convulsive seizures in lead poisoning and death. Lead-saturated cells would be incapable of high function. So far as their capability of conduction, inhibition, and volition are concerned, such cells would be practically dead. In not one of my cases was there metallic lead to the extent of one grain found in the brain. Even admitting that lead is a poisonous metal, this is a small quantity to have produced such serious results. I do not think that lead encephalopathy is to be entirely attributed to the deposition of lead in the brain—even intimately combined as it may be with the protoplasm of nerve cells. One of the worst cases that I have seen was that of Cath. Y., who was attended by Dr Inglis of Hebburn, and in the brain no lead was found on most careful chemical analysis. The absence, too, of albuminuria removes the case from simple uræmic poisoning, but not altogether from the pale of imperfect renal function. Lead early disturbs this function, and deranges alike the structure of liver as well as of kidney. These are the organs by which lead is principally eliminated from the system. Metabolism is therefore not only imperfectly carried on, but the elimination of waste products is interfered with. Add to this the fact that, before convulsions occur, there has been a gradual failure in renal activity owing to arterial spasm, and thus not only are waste products not eliminated, but even the water itself is not removed from the

[1] *Journal of Mental Science*, Jan. 1888.

system. These conditions alone are quite capable of causing convulsions. My colleague, Dr Drummond, has observed perivascular dilatation of the small cerebral arteries amounting to varicosity, and in this way by extra vascular pressure may be explained the flattening of the surface of the brain. The most careful microscopical examination of all parts of the brain, however, has hitherto failed to demonstrate the existence of any well-defined lesion. This remark does not apply to the naked eye appearances of the brain. The surface of the brain is dry and flattened, just as if it had been pressed; the brain tissue is dry, or it is hydræmic; it is firm as a rule to the touch, and always extremely pale. Many conditions, therefore, are present at the time when death occurs. A primary poisoned blood either from the presence of lead alone, or more probably from animal poisons of a much more subtle and dangerous character, and depending upon deranged liver and kidney function, may explain the severe headache which is complained of before the attack, but if the kidneys perform their work well this with other symptoms may subside. On the other hand, if the case is progressive, the urine is noticed to be daily becoming scantier —sometimes even it ceases to be secreted—and then, owing to retention of animal poisons, eclampsia occurs.

Treatment.—The treatment of lead poisoning may be considered under the two aspects—preventive and therapeutical. We are not yet fully acquainted with all the effects produced by lead upon the system. Many obscure nervous disorders, not yet thoroughly understood, may possibly be due to the entrance of the metal into the body in very minute quantities. We should see that lead enters as little as possible into the formation of receptacles for the storage of drinking water, or into pipes for conducting it. The drinking water carried into our houses should be used direct from the main, and never be stored in tanks. Wherever lead, under these circumstances, can be replaced by other metals—iron for example—this should be done. Glazed earthenware, too, might with advantage be substituted. For

the methods to be adopted, where the drinking water to a community is too soft, or where there is an excess of free vegetable acid, I must refer my hearers to special treatises upon this subject. It is sufficient for me to state that the addition of an alkaline carbonate—carbonate of lime to the extent of two grains to the gallon—will, provided that the water does not at the same time contain an excess of carbonic acid gas, allow of the formation of a protective coating on the interior of the pipe, and thus materially diminish or prevent all risk from lead poisoning. Silica possesses a similar protective influence. On this point the reports of the special commissioner of the *British Medical Journal* (B. M. J., 17th May 1890), may with advantage be consulted; also Garret's monograph on the "Action of Water on Lead." It is difficult to say to what extent the presence of micro-organisms influences the plumbo-solvent action of water. Power—medical inspector of the Local Government Board—has suggested this as an explanation of plumbism. It is not contended that the micro-organisms directly attack the lead, but that the solvent influence of water is due to chemical products caused by the multiplication of these low forms of life. Sewage-polluted water—a breeding-ground for micro-organisms—is known to have a strong solvent action upon lead from the chlorides, nitrates, and nitrites that it contains. Brown of Bacup does not consider that the epidemics of plumbism from moorland water can thus be explained. Stocking the reservoirs with fish has been found to be a cheap and efficient method of lessening the plumbo-solvent action of moorland water. Towns whose reservoirs are well stocked with fish, even though supplied with moorland water, never suffer from epidemics of plumbism.

My experience of plumbism has been gained chiefly amongst workers in lead factories. Employers are fully aware of the dangers of lead making, and take every precaution to prevent their workpeople suffering, but the workpeople themselves are not always as careful as they ought to be. The rules and regu-

lations in force at the Tyne Lead Works, Hebburn, and which formed the basis of the Act of Parliament relating to this subject, are complete in almost every detail. All the men and women employed in lead factories are examined every week by medical officers, and those who show signs of commencing plumbism, are at once suspended for three months, and are only allowed to recommence work after having been examined and certified by the doctor. Lists of those who are suspended are circulated amongst the lead manufacturers of the district, but in spite of this precaution, employers are occasionally deceived by women entering the factories under different names. Baths are provided, and there are bathwomen whose duty it is to see that frequent ablution is carried out. No person in a lead factory should take food with unwashed hands. Respirators are provided, and are worn in most of the factories, and coarse cotton over-alls are worn by many of the women, and removed before leaving the factory. An acidulated drink of the nature of lemonade, made with sulphuric acid, or a drink of sulphate of magnesia acidulated with sulphuric acid, is provided by the employers. These have a preventive influence, but the workpeople do not drink of them as freely as they ought to. There are two points relating to lead workers that I cannot too strongly draw attention to: one is, that no man or woman should be allowed to begin the toil of the day without having had a substantial meal,—experience and experimental investigation having shown that during the process of digestion little or no lead is dissolved and absorbed; and the other is, that if there is an industrial class to whom total abstinence is a physiological necessity, it is lead workers.

Colic is the most common symptom of lead poisoning; it is the one that most frequently calls for treatment. So long as lead colic was thought to be simply due to constipation, purgatives were the remedies employed, and the one which from its rapid and powerful action recommended itself was croton oil. This is a line of treatment which has considerably sunk into abeyance,

for experience has shown, that whilst amelioration of pain may follow the use of the oil, vomiting, pain and constipation recur, and oblige the physician to repeat on two or three occasions the administration of the medicine. This applies, however, to all purgatives. In this country, we are content with the use of milder purgatives, such as castor oil or sulphate of magnesia, with which if the pain is severe opium is generally combined. Occasionally the constipation is so extreme that it is only after the use of enemata and the repeated administration of purgatives that the bowels are opened. In some of my cases, there had been constipation varying from two to three weeks. Were lead colic simply due to the presence of metallic compounds in the contents of the intestine, relief would at once be got by clearing out the bowels by purgatives. But, as we have seen, this is neither the explanation of the pain, nor does relief always follow purgation. On the other hand, we are asked to transform the soluble salts of lead—present in the intestine, tissues, or blood—into soluble salts, which, if present in the intestine, would in all probability be expelled by purgatives. It was with this object that Kapeler and Brachet gave alum. They thought that an insoluble lead sulphate would thus be formed. I have not had satisfactory results from the use of alum, either administered alone or with opium and magnesia. The one drug which gives the best results in lead poisoning is undoubtedly iodide of potassium. Combined with sulphate of magnesia, lead colic will generally soon subside. Melsens first recommended iodide of potassium in 1849, and again in 1865, when a larger experience only confirmed his recommendation. He gave it with the object of causing lead compounds to become soluble, and thus reabsorbed they were rendered easy of elimination. That the iodide of potassium has a powerful eliminating action there is no doubt,—a circumstance due either to the formation of a double dialysable salt, or according to Gubler, to a heightening of the functions of disassimilation. Its efficacy is such that a certain amount of care is necessary, particularly in the presence of acute symptoms of

plumbism. Under its influence, symptoms have increased in severity, and sudden death has taken place, due in very great measure to the rapid entrance into the blood of a large amount of soluble lead salt, which had previously been insoluble and inert, although deposited in the tissues. I have had fairly good results in the treatment of lead colic by belladonna, but I have never pushed it in the manner recommended by Gauch.* Regarding it as a specific, he begins with very minute doses, gradually increased, and given every quarter of an hour, and he pushes the drug until its physiological effects are produced (erythema, dilatation of pupils, and delirium). For the treatment of Saturnine paralysis, strychnine or nux vomica, with iodide of potassium, will be found useful, but for this defect the most important agent is electricity. Although the nerves have lost their irritability, the muscles will still respond to the voltaic current, slowly interrupted. The return of power to the paralysed limb is frequently slow, but it is well to keep up the electrical treatment, for by its means muscular irritability is maintained, and nutrition of muscle secured.

For acute lead encephalopathy, where the patient is in convulsions, nitrite of amyl is the remedy *par excellence*. Under its influence the pulse regains its softness and rapidity, and consciousness may return. In other cases, where in addition to the eclampsia there is complete suppression of urine, the subcutaneous injection of pilocarpine has been followed by most satisfactory results.

* "Du traitement de la Colique de Plomb., par la Belladonna," p. 63.

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