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POISONING BY CHROME YELLOW USED AS A CAKE DYE.

The Subsequent Clinical History of the Cases, Including a Case of Paralysis Agitans and of Chronic Endocarditis.

BY

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FROM

THE MEDICAL NEWS.

January 26, 1889.

[Reprinted from THE MEDICAL NEWS, January 26, 1889.]

POISONING BY CHROME YELLOW USED AS A CAKE DYE.

The Subsequent Clinical History of the Cases, Including a Case of Paralysis Agitans and of Chronic Endocarditis.[1]

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I REPORTED, in September, 1887, a clinical analysis[2] of 64 cases[3] of poisoning by chrome yellow

[1] Read before the Philadelphia County Medical Society, December 12, 1888.

[2] This paper was published in THE MEDICAL NEWS, December 31, 1887.

[3] In addition to these, I have notes of 15 others, making a total of 79 undoubted cases traced to 2 bakers. The 15 include 4 cases of lead convulsions, which in 3 ended fatally. It is unfortunate that I was unable to obtain the histories of very few of the large number of early patrons of Palmer, the baker, who used the dye freely for years in another section of the city than that in which he was apprehended. No doubt there were many interesting cases of plumbism among these.

It is to the point, in this connection, to state that I believe I can trace to lead-poisoning from dyed cakes many cases of disease of seemingly obscure or supposed idiopathic origin, seen in the past year and a half of my service in the out-patient medical department of the Jefferson College Hospital. Among these are cases of pronounced anæmia, renal fibrosis, peripheral neuritis, spastic paralysis, neurasthenia, obstinate headache, and, at least, one case of long-standing delusional mania.

used as a cake dye. The present paper is a further history of such of these as I have had under observation to the present, a period of fifteen months.

8 of the 64 died in convulsions. The later history of 12 of the 56 survivors I have been unable to ascertain. Absolutely nothing could be learned of 6 of the 12; they are, however, believed to be living. I regret I am unable to report their condition. They were severely poisoned, after prolonged exposure. Nor could sufficient be learned of the second 6 to justify their consideration here. Several of these were personally questioned but with unsatisfactory results.

At the time the data for the analysis of the 64 cases were being collected, all of the 44 now reconsidered exhibited some unmistakable and pronounced symptoms of plumbism, such as cachexia, colic, arthralgia, or encephalopathy, and the blue line was present in the gums of all save 1. A minority only of the 44 received any continuous treatment, and many of these were in the hands of homœopathists, who, it is believed, did not give eliminants. The health of 31 of the 44 is yet decidedly impaired, and several of the remaining 13 ail slightly.

The lead cachexia is still present markedly in 12. In these the skin has been of a decided earthy-yellow hue, continuously since the poisoning. The gums of 8 of the 12 show a fading though distinct blue line, and are retracted from the teeth. In 3 of the 12 there is a purplish line, and in 1 no trace of a

line exists. Considerable anæmia[1] is present in those in which cachexia is yet marked, and in 1 in which cachexia is absent. The mucous membranes and conjunctivæ are pale. In several a venous hum can be heard in the neck; and, in 1, a basic (pulmonary) murmur.[2] Most of the 12 have breathlessness and palpitation on slight exertion. Several speak of præcordial pain. 8 of the 12 are very dyspeptic; 5 have yet occasional attacks of severe colic which occur unprovoked by food or other known cause. All of the 12 have headache, which in several is severe and constant.

Evidences of functional derangement and of organic disease of the heart have appeared in 7 of the 44 since the poisoning. In 6 of the 7 there are present symptoms of cardiac irritability, such as præcordial pain, increased by exertion and then accompanied by palpitation and shortness of breath. In 2 of these the palpitation and shortness of breath occur often when at rest and in recumbency, as at night. All of the 6 have increased frequency of pulse, and the heart of each is overacting, with accentuation of the second sound at the apex and some increase in the area and force of the impulse. In 3 of the 6 this increase is greater than can be accounted for by mere functional overaction, and other accompanying physical signs and symptoms indicate slight but unmistakable hypertrophy. The

[1] Through an utter lack of coöperation and interest on the part of these cases, none of which were my patients, I was unable to obtain a blood examination.

[2] This was not examined for in all.

seventh case illustrates one of the insidious effects of chronic lead poisoning, a tendency toward slow degenerative changes in the vascular system. There was at first irritable heart, which was succeeded by hypertrophy, and that by a chronic mitral valvulitis with insufficiency. Dilated hypertrophy of the left ventricle then followed.[1]

The case is that of Mrs. H. H., aged forty-seven. There is absolutely no history of rheumatism, gout, syphilis, or alcoholism. Her health had always been good until the spring or summer of 1886. Attacks of typical lead colic then developed with the usual attending phenomena. These continued, at intervals, until the summer of 1887. There were also present during this period severe headache and constant mental and physical exhaustion. Slight pains in the ankles, unaccompanied by any inflammatory condition, but with sensations of numbness and burning in the soles of the feet, were occasionally felt. When first seen in June, 1887, she had been a patron of Palmer for fifteen months, and had eaten almost daily of the dyed cakes. Her condition then was much as has been stated. In addition, it was noted that she was emaciated and dyspeptic, had palpitation and shortness of breath on exertion, and præcordial pain. There was heightened arterial tension, a much overacting, irritable heart, but no murmur. Lead was found in her urine by Dr. Leffmann at this time, and an examination of the eye-ground by Dr. Hansell showed "œdema of both

[1] Professor Da Costa, to whom I showed this case somewhat more than a year ago, when the valve changes were not perceptible, and, later, when they were markedly present, fully agrees with me as to the pathological condition, and as to its plumbic origin.

retinæ, with effusion into the right retina below the fovea; pulsation of larger and tortuosity of both large and small veins."

Because of her absence from the city during the summer of 1887 I saw her but seldom. Ailing severely in September following, she presented herself for regular treatment, and has been under constant observation to the present. The mental and physical prostration had persisted, and, in August, coarse tremor, involving the head and limbs, had appeared. The shaking began each morning on rising, and continued constantly through the day. It was especially aggravated by physical exertion. Decided weakness of grasp accompanied it, but no paralysis. She had continuous headache, and palpitation was so troublesome at night that she was unable to rest in recumbency. Early in September there was found extreme overaction of the heart, with undoubted hypertrophy of the left ventricle. No murmur, however, could be detected until about the 14th of October following. There had then developed a faint systolic mitral bruit, which was accompanied by distinct accentuation of the pulmonary second sound. Palpitation was now more distressing than formerly, and was associated with attacks of shortness of breath and of dyspnœa by day, but oftener by night. Prior to this, believing renal fibrosis was in progress, the urine, which had been of a low gravity and profuse in amount, had been frequently examined for albumin and casts, but always with negative results. Shortly subsequent to the development of the murmuralbumin appeared, detectable at first only with picric acid, but soon with nitric. The amount never exceeded a gramme to the litre, and was usually less. Coincident with the appearance of the albumin the urine diminished in quan-

tity, and hyaline and granular casts were very often found, sometimes in large amount.

The remaining facts in the case are concisely as follows: The mitral murmur became louder, and the area over which it could be heard greater. Evidences of stretching of the left ventricle soon appeared, and, with these, signs of overloading of the pulmonary and general venous circulation assumed prominence. The attacks of dyspnœa at night deepened into orthopnœa. There were cough and frequent blood-spitting. The ankles and feet were at first puffy, and then swollen. Purpuric spots appeared on the legs. The portal circle was constantly congested, causing an icteroid hue of skin and pronounced gastro-intestinal catarrh.

This condition of things continued pretty constantly until the spring of 1888. She then improved very steadily, and the more pronounced signs of "back-working" were only occasionally present. Since April last the daily quantity of urine passed has been normal or above, and albumin and casts have been absent. All the usual symptoms of plumbism had disappeared at this time, The tremor persisted intermittently for months, but grew less and less frequent as her condition ameliorated.

The physical signs referable to the heart are now those of mitral regurgitation and dilated hypertrophy of the left ventricle, with hypertrophy in excess of the dilatation. The murmur is very distinct, and has the usual characteristics of that of mitral insufficiency. Though there are yet almost constantly present some indications of mechanical derangement of the circulation, such as œdema of the ankles and legs, congestion of the portal viscera, and, at times, of the lungs with blood-streaked sputum or actual bloodspitting, and attacks of bronchial

catarrh, freedom from excessive palpitation in the past few months could be obtained only by the persistent use of aconite. Digitalis and strophanthus, which, in moderate doses, were of service a year ago, now aggravate the palpitation, and do not materially lessen the tendency toward venous congestion. The attacks of dyspnœa, which occur once or twice weekly, are relieved, as before, by free dry cupping of the chest. Restorative agents, such as arsenic, iron, and the vegetable tonics, are employed to improve nutrition. Frequent resort to purgatives is necessary to relieve the portal stasis.

I have no doubt that some cases of chronic endocarditis, the origin of which we are at a loss to account satisfactorily, a history of acute articular rheumatism or of strain not being obtainable, are of lead origin.[1] Lead is, I believe, a more frequent

[1] Chronic valvulitis, as a result of lead poisoning, is not uncommon. Duroziez reported (L'Union Médicale, December 15, 1885) eleven cases of mitral stenosis and one of aortic regurgitation, occurring in plumbic subjects in whom there was absolutely no other assignable cause for the valve affection.

A typical case of lead endocarditis, originating aortic stenosis and mitral regurgitation, is that of B. F. K., a painter for twenty-eight years, who has been under constant observation in the out-patient medical department of the Jefferson College Hospital since 1881. He first presented himself for treatment because of obstinate colic and an ileocolitis which had arisen in consequence of lead poisoning. There was a well-marked blue line, typical lead cachexia, constant headache and vertigo, and distressing arthro- and generalized neuralgia with cramps in the muscles of the extremities, and jerking of the limbs. The urine was free from albumin. His heart was examined on several occasions by Prof. Da Costa between 1881 and 1885. On the first exploration there were found considerable overaction, and a soft systolic murmur limited to the apex. In the autumn of 1884 the

cause of chronic endocardial inflammation than gout, syphilis, or alcoholism. Even where a doubtful history of rheumatism exists, we should not be deterred from an inspection of the gums and an inquiry as to the possibility of lead poisoning in the past. In all cases of valvulitis of seemingly obscure source this should be done as a matter of routine. A caution is necessary that a mere history of pains in and about the joints must not be accepted as undoubted evidence of rheumatism, without a careful investigation as to signs of accompanying arthritic inflammation; for it is undoubtedly a fact, of which I have seen striking examples, and the non-recognition of which by practitioners has caused many lead cases to be overlooked, that arthralgic pains are among the most common of the protean manifestations of plumbism; and that they may persist for lengthy periods entirely without other noticeable symptoms, save, perhaps, cachexia; and that even the latter may be absent in undoubted cases.

An abnormally forcible cardiac impulse, with accentuation of the aortic second sound and increased tension in the peripheral arteries, is constantly

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area of cardiac dulness was found to have considerably increased. The apical murmur was very distinct, rather harsh in quality, and was well transmitted toward the inferior angle of the left scapula. There was now present a systolic right basic murmur, distinct in quality and pitch from the apical bruit, and heard in the carotid artery. There had been frequent cardiac pain, attacks of palpitation, and signs of arterial ischæmia. At the present writing the mitral and aortic murmurs are very distinct. Compensation is good.

present in many cases of lead cachexia. In these a tendency toward insidious valvular changes must be great, and this is no doubt favored by the state of faulty nutrition and anæmia always existing in such cases. Simple anæmia is supposed sometimes to originate chronic valvulitis, through the augmented arterial tension present in its earlier stages causing increased backward stress on the valve curtains, and especially on the mitral leaflets, which, with each ventricular systole, are subject to a force equalling the resistance of the whole arterial circuit. Since the conditions favorable for the production of chronic valvulitis are more constant and decided in plumbism than in ordinary anæmia, it is not unlikely that chronic lead poisoning is oftener a cause of slow valve changes than mere poverty of blood. Having in mind the wide prevalence of lead poisoning and the frequency in the past year and a half with which I have encountered cases of somewhat obscure type, which no doubt I would have previously overlooked, because not on the watch for them, and seeing repeatedly in my service in the out-patient medical department of the Jefferson College Hospital cases which have passed through the hands of other practitioners unrecognized, I believe it probable that mitral stenosis, often met with as an insidious affection in young anæmic women free from rheumatic history, not infrequently owes its origin to unsuspected lead poisoning.

Disorders of digestion are prominent in a number of the 44, independent of colic or colicky pains. One (Mrs. G. A.), who was so prostrated by

long-continued colic and arthralgia, that she was confined to bed for two months, and lost thirty pounds in flesh, has, in addition to attacks of severe generalized headache, constant gastric irritability. A trifling dietetic error which in health would not affect her, now readily provokes vomiting.

Obstinate constipation is yet present in 7. In these the bowels do not move without purgatives.

A chronic catarrhal enteritis has existed in 1 since his first attack of colic fifteen months ago. There are five or six loose passages for three or four days, succeeded by two or three days of constipation. Abdominal tenderness and pain are felt, in addition to colic which is yet frequently present though in a milder form. He had arthralgia affecting the knees and ankles for months, so severe that he was compelled constantly to maintain the recumbent posture. He also had wrist-drop.

Apart from slight colicky pains, which have been frequent in many since the abatement of pronounced symptoms of plumbism, and which, in a few, may be traced to dietetic errors, paroxysms of abdominal cramp having all the intensity and character of true lead colic, yet occur in 5. The seizures appear at longer intervals, as time passes, and are lessening in intensity. At first the interlapse averaged from ten to fourteen days, and the duration of the attack was three to seven days, but the former gradually lengthened and the latter diminished, until recently, in each of the 5, a severe seizure occurred about every four to eight weeks, and lasted from one to three days. None of these cases received regular

treatment. In all there is reason to believe lead is yet present in the system.

Pains about the joints and in the muscles in their vicinity yet occur in 15.[1] In some the pains have been constant, with exacerbations and remissions; in others they have occurred intermittently every five to thirty days, in attacks lasting three to ten days. The ankles and knees, and the neighboring muscles are most frequently affected in 9; 3 of these have in addition sharp stabbing pains in the wrists, and 1 of the latter, pain in the hip. In 2 others the ankles, knees, and hips are equally affected; in 1 solely the ankles and wrists, and in another the lumbar and sacral regions. Dull and aching, burning or darting pains in the muscles adjacent to the affected joints accompany the arthralgia in all, and these muscles are yet the seat of frequent painful cramps. 2 have, beside the joint pain, very sharp darting pains in the forearms and wrists, in the course of the nerve trunks, associated with some loss of power and weakness of grasp. In 2 others[2] severe burning in the soles of the feet, alternating with numbness and tingling in the feet and hands, is sometimes felt simultaneously with the arthralgia.

Very painful and persistent inframaxillary neuralgia is present in 2 of the 15. Neither has carious

[1] 13 of these received no treatment, save from homœopathists; the remaining 2 were under a regular physician's care for two or three weeks when first affected.

[2] The plantar surfaces in these cases were somewhat anæsthetic.

teeth, and each has had several sound teeth extracted without the pain ameliorating. Aching in the right clavicle frequently accompanies the arthralgia in 1. General muscular aching and loin pains persist in 3. It is believed that inflammatory conditions of the joints, such as redness, heat, or swelling, at no time occurred in these cases. They were frequently searched for. The pains in nearly all are aggravated by night. Damp and cold weather increase them in 5.

I was recently able to ascertain the state of cutaneous sensibility in 8. Some anæsthesia was present in the area supplied by the peroneal nerve and its terminal branches in 2; there was plantar anæsthesia in 2 others; in 4, sensation was normal or hyperæsthetic in the painful extremities. No tendinous swellings about the wrists, or elsewhere, were noticed in any.

It is difficult to decide as to the exact influence of lead in the production of the arthralgic pains in these cases. Their character and seat, and their continuance in those that received no eliminative treatment, and that yet show evidence of plumbism, suggest they are in most of the 15 a mild form of the articular and muscle pains which were present in 47 of the 64 when these cases were thoroughly investigated over a year ago. Our knowledge of the peculiar morbid state underlying so-called lead arthralgia is too slight to permit us to assert that the same pathological conditions always exist. In most of the 47 the symptoms were originally those of aggravated neuralgia of the joints, bones, and intra-

muscular nerves. In not a few who were under observation for a considerable period and received no regular treatment, while the arthralgic pains were prominent, there were present symptoms suggestive of neuritis, such as sharp pains in the muscles and in the limbs in the course of the nerve trunks, often with decided tenderness on deep pressure; alterations in sensibility such as paræsthesia, hyperæsthesia, and anæsthesia; weakness in the limbs; rigidity, and frequent cramps in the muscles.

The electrical reactions were taken in several of these cases, but no quantitative or qualitative changes were found. The examinations, however, were made at too early a period for the negative result to be of much value in the diagnosis of the condition.

In none did the character of the symptoms suggest that the pains were of a gouty nature, nor do they now in the 15 under consideration. The smaller joints were unaffected, and no inflammatory appearances accompany the pain in the large joints. It is not unlikely that slight deposits of urates may occur in a joint, and no appreciable objective phenomena at once arise. Yet this condition could scarcely continue long without an inflammatory outbreak. There is no doubt that a special predisposition to gout exists in subjects of chronic plumbism, and it is not improbable that slight joint pains, with aching in the limbs, and muscular cramps, ephemeral neuralgias, headache, vertigo, and mental depression, occurring in such subjects, are sometimes symptoms rather of the gouty or lithæmic state induced by lead than of the direct action of that metal. Where

doubt is felt, it is of diagnostic importance to examine the urine and blood-serum, quantitatively, for uric acid. I have, as yet, been unable to do this in any of the 15 arthralgic cases. It was probably excreted in deficient amount in at least 2. The urine of these has been more or less scanty, since pronounced symptoms of plumbism were present; it often deposits urates, and fits of the gravel occur. An unsuccessful effort was made to get specimens.

At the date of the preparation of the clinical analysis of the 64 cases, complete paralysis of the extensor muscles of the forearms (typical wrist-drop) had occurred in but 2; bilateral weakness of the extensors of the fingers existed in 1 other, and in 2 others there was slight loss of power in all of the extensors of the fingers and of the wrists, without actual paralysis. In the third-mentioned of these cases (1 of the 44) complete forearm extensor paralysis afterward occurred. In the 2 last mentioned the ataxia disappeared without paralysis ensuing. So that complete wrist-drop was met with in but 3 of the 64 cases, and it was not encountered in any of the other (15) cases of the chrome yellow poisoning traced to the same source, of which I have notes. This exemption among so many cases of pronounced plumbism in which the rarer condition—encephalopathy—occurred so frequently, is quite remarkable.

In none of the 3 cases did the paralysis extend to the upper arm or involve the supinator longus. Complete recovery has taken place in 1. In 2 of the cases power has been partially regained in the

extensors of the wrist, so that the hand can be incompletely extended if the fingers are flexed into the palm, but the extensors of the fingers and thumb in both cases are yet too ataxic more than very slightly to extend these members or the hand itself if the fingers are first passively straightened at the metacarpo-phalangeal joint. The paresis is greater on the left in 1; in the other, it is equally marked on both sides. Sensation is unaffected. Considerable atrophy exists in the situation of the paretic muscles in each case. Neither would come for electrical examination. It is presumed reactions of degeneration are present. 1 case is known to have taken for some weeks potassium iodide; the other received no treatment.

In 1 of the 44 in which typical paralysis agitans has developed, presumably as a result of lead poisoning, there exists, in consequence of weakness and stiffness of the forearm muscles, lack of complete extension of the hands and fingers on the wrist, more marked on the right. This case is of especial interest and worthy of detailed report, because it is generally supposed that although lead may occasionally originate tremor resembling somewhat that of paralysis agitans, the typical "shaking palsy," of Parkinson does not arise in consequence of poisoning by that or other metals.

Mrs. Mary McF.,[1] aged sixty-one years, occupa-

[1] This case recently visited me several times in the medical clinic of the Jefferson College, and was shown by Prof. Da Costa to the class. I may state he is fully in accord with me both as to the diagnosis and as to the probable origin of the disease from lead poisoning.

tion general housework. One of a family[1] of seven all of whom were severely poisoned from the same source. She had eaten the chrome yellow buns for nearly a year before symptoms other than cachexia, constipation, arthralgia, and slight colic, appeared. In June, 1886, she was seized with recurrent attacks of lead colic, lasting four to ten days, with intervals of about one week, during which slighter abdominal cramps, with nausea, vomiting, and constipation persisted. I visited her frequently in June and July, while investigating other cases of poisoning. All were studied with great care and it is absolutely certain that she was free from tremor then and before. In addition to colic, which persisted through the summer and autumn, attacks of generalized headache were now present with neuralgic pains in the joints and limbs. She lost flesh and strength, was depressed mentally, and sleepless. Tremor of the right hand and fingers was first noticed early in October, while plumbic symptoms were yet prominent. It was then slight and inconstant and appeared usually after mental or physical fatigue, but ceased on voluntary effort, by exercise of the will and during sleep.

I now lost sight of her until September, 1888. Since then she has been under constant observation. The following are the additional facts in her case from October, 1887, to the present.[2] She at no time received continuous treatment directed toward eliminating the lead. I had endeavored to persuade her to take potassium iodide. After a five days' trial she desisted, believing it aggravated colic and headache. Her health had always been robust until she suffered from plumbism. She had never had tremor

[1] 2 of the 3 cases of wrist-drop occurred in this family.

[2] November 15, 1888.

before, nor had she ever received a physical injury or mental shock. No hereditary history of any neurosis is obtainable. Her people were sturdy Irish, who died rather of old age than of any disease. Umbilical colic and severe loin pain yet occur intermittently. She is dyspeptic and constipated, and has a marked blue line, with retracted gums and discolored teeth. Her skin is earthy-yellow and conjunctivæ are icteroid. There has been quite constant headache since the autumn of 1887, but it is less pronounced now than during the preceding winter and spring. Its situation is at times frontal and occipital but it is usually severest about the vertex. It is sometimes a deep-seated heavy ache, at others it is sharp and shooting. Recumbency and the approach of night greatly aggravate it. Sleeplessness and restlessness continue and she is disturbed at night by dreams of so frightful a character that she rouses the neighbors by her shrieks.

Tremor of the right hand and fingers increased rapidly in degree, and extended to the forearm and arm soon after its appearance. By the third month the right foot and leg were noticed to shake while she was seated. Seven months later (August, 1888), fine tremor appeared in the left lower limb. The facial muscles, and those of the tongue, eyeballs, neck, and left upper extremity, are entirely free from even slight tremor. The right upper extremity, especially the fingers and hand, is in a continuous state of almost uniform, coarse movement, which ceases instantly and absolutely—though momentarily—when voluntary effort is made with it, such as buttoning or unbuttoning the dress. If the effort is continued, after a few moments the tremor returns slightly, and it starts into redoubled violence on cessation of the effort, unless it be car-

ried to complete and lengthy exhaustion of the arm muscles. Thus, having found that the constant jerking prevents sleep, she has for several months been in the habit on retiring of tossing the right arm about sometimes for hours, until, from sheer muscular weariness, tremor is no longer perceptible and sleep comes. It ceases absolutely during sleep. Mental excitement greatly augments it. All efforts of the will directed to control it are now ineffectual, and, indeed, rather increase it. If the tremulous hand is forcibly held, the movement becomes more violent after a moment, and tremor then appears in the opposite arm, and if she is sitting, that present in both legs grows coarser.

Her handwriting is suggestive. Always disliking writing, she had not before held a pen for years. This renders the zigzag character of the strokes in the specimen more interesting. For, were she used to the pen, the tremor, which ceases momentarily on voluntary motion, would perhaps have permitted her to complete the few words before it reappeared. As she is unaccustomed to writing, the will does not readily direct the hand. The movements are deliberate and the up and down strokes slowly and somewhat hesitatingly made. Time is therefore afforded the tremor to recommence.

Muscular weakness, stiffness, and some fixation of the limbs are present, but are not yet nearly so characteristic as the tremor. The grasp as measured by the dynamometer at different times recently, varied between 15 and 33 with the right hand, and between 35 and 40 with the left. Incomplete extension of the hands and fingers seems to be due largely to rigidity of the flexors, as force is required to extend passively the fingers at the metacarpo-phalangeal joints. The forearms are slightly flexed and stiffened

at the elbow. There is some rigidity of the anterior cervical muscles, and muscles of the shoulders, which slightly inclines the head forward and causes stooping. In the inferior extremities the rigidity is greatest at the knee-joints and in the adductors of the thigh; more marked on the right.

Voluntary movement is slow, but the gait is as yet not festinating, nor is there propulsion. The facial muscles are unaffected. There is no festination of speech; articulation is slow rather than rapid, but it is monotonous and not syllabic. She is said often to "mix" her words. This I have not noticed. The knee-jerk is exaggerated on both sides. There is no ankle-clonus. Muscular nutrition is poor. The mechanical irritability of the muscles is increased, and there is quantitative diminution to both galvanism and faradism, especially the latter. This is most pronounced in the extensors of the forearms and in the right deltoid, but it is throughout more marked on the right side. Sensations of heat are absent. A feeling of constant chilliness has been present for nearly a year. It was as great during the summer as in cold weather.

Tactile sensation was carefully taken on three separate occasions, and, contrary to what is the rule in this disease, it was twice found very decidedly impaired. This was manifest on all surfaces of the arms, shoulders, and legs. It was not taken on the trunk or thighs. The whole surface of the right upper extremity was especially anæsthetic. This condition was noted in September, and again in the early part of October, 1888. Six weeks later, on casually retaking sensation in the finger-tips and hands, I was surprised to find the anæsthesia had disappeared. The cause for this is not clear. It is, however, unlikely that the loss of sensation had any

direct dependence upon the paralysis agitans. Most probably it was of plumbic origin, had existed for some time, and was in process of disappearing when she came under observation this autumn. Lead was found in her urine in August, 1887. Albumin and sugar are absent.

The eye-ground was recently examined by Dr. Hansell, who reported: "R. $\frac{20}{XL}$; L. $\frac{2}{CC}$; opacities in cornea; double optic atrophy with choroidal ring and slight cup. The arteries and veins are small. Pupils normal."

Mental and physical depression is still a constant symptom in 10 cases. Headache is present in 8 of these, which before plumbic symptoms appeared were free from it. In several it is described as a diffused, deep-seated ache. In 3 it is severest in the frontal and occipital regions. In all of the 8, until quite recently, it was constant, with exacerbations and remissions. It is now of far less severity than formerly.

In 5 of the 8 the urine was examined for albumin, but with negative results. In one of these cases (Mrs. S.) in whose urine considerable lead but no albumin was found a year ago, and who had for months epileptiform convulsions of undoubted lead origin, subacute mania developed with hallucinations of sight and hearing and delusions of persecution. This condition still exists. In another (Mrs. G. W. L.), last seen in September, 1888, an eye-ground examination disclosed œdema of the temporal side of each nerve and tortuosity of the veins. Headache, worse at night, has been constant with

her for more than a year. She now is melancholic, and has delusions of conspiracy. There are restlessness and sleeplessness at night and bad dreams. Her skin is sallow. The gums and teeth are still discolored by lead. There are arthralgic pains; dyspeptic symptoms; complete anorexia; a tense, full pulse; overacting heart, with a markedly accentuated aortic second sound. Her urine, recently examined, was of low gravity, but contained no albumin. Lead was not sought for, as the quantity furnished was small, and she would not take potassium iodide.

One other of the 5, a girl, aged seventeen years, had an attack of chorea, during January, February, and March, 1888, while decided cachexia, colic, and the blue line were yet present. She had never had rheumatism, and no family history of it or of chorea can be elicited. Her heart was functionally disturbed, but there was no murmur. None of these 3 cases received any regular treatment.

Curious spells of generalized tremor, of brief duration, have occurred in 1 of the 44 (Mrs. D., aged thirty-eight years), for the past sixteen months. An attack consists of a sensation of numbness starting in the wrists, and rapidly spreading over the body. There is then a feeling of impending danger, and immediately coarse, uncontrollable tremor seizes the head and limbs and continues for a half-hour or longer. Profuse diuresis generally takes place on its cessation. There is no loss of consciousness or tendency to spasm. The fit is not hysterical, nor are there at any time indications of the hysterical

state. Motility and sensation are unaffected in the intervals. They were not tested during or immediately subsequent to one of the seizures. These spells occur about every three to five weeks irregularly; not at the menstrual period. Appropriate remedial measures directed to prevent their recurrence have the desired effect when pursued with regularity. She will not continue treatment sufficiently long to be entirely rid of them. This woman had convulsions in April, 1887.[1] Five of her children were similarly affected about the same time, and four died.[2] Another was born in May, 1887, while symptoms of lead poisoning were yet present. This infant was undersized and cachectic at birth, and, though not suckled by the mother and every effort was made to raise it, it did not thrive. When aged four months, rhachitis developed; paroxysms of laryngismus stridulus were frequent, and death took place in convulsions in the tenth month.[3]

The appearance of the gums and teeth in 24 of the 44 cases is yet indicative of plumbism. But 3 of the 24 received eliminative treatment. A decided though fading blue line is present in 10, a distinctly bluish-purple line in 5, and a purple in

[1] Her urine was slightly albuminous at that time. The albumin disappeared very soon after treatment was began to eliminate the lead, and has at no time been present since.

[2] *Vide* "Notes on Some Obscure Cases of Poisoning by Lead Chromate," THE MEDICAL NEWS, June 18, 1887.

[3] All of the five infants born of mothers exhibiting symptoms of lead poisoning during gestation had convulsions; four within two months after birth. Three of the five died in them. Another infant, born in July, 1888, of a mother who had pronounced lead poisoning during the early months of pregnancy, died in the fourth month in convulsions.

9. In 12 of the 24 there is much retraction and atrophy of the free margin of the gum, permitting considerable exposure of the neck and fang surfaces of the teeth, which, together with the crown surfaces, in the greater number of the 24, are yet discolored by lead sulphide. The free margins of the gum in many of the 24 are hemorrhagic. In these, slight friction causes venous oozing. The purple line is present in the situation previously occupied by the blue. In many of the cases I have watched this discoloration of the free gingival margin through its varied phases. It is of diagnostic interest to note that at first the so-called "line" consists of a number of fine black dots irregularly distributed, or an aggregation of them, forming a narrow bluish-black streak in the interdental portion of the gum, or in the extreme edge of the free margin, grasping one or more teeth, usually the lower bicuspids and molars, the teeth which are apt to be longest in contact with particles of food and about which decomposition of food elements is likely to be greatest.

Friction renders the pigmentation more distinct and perhaps causes slight hemorrhage. If the poisoning is slight and the absorption of lead ceases, the pigmentation slowly fades without destruction of the affected edge of the gum and no retraction of the gingival margin occurs. In several cases, three months after the lead ceased to be ingested, the narrow black streak or the fine dotting in the thin edge of the gum was distinctly visible, and at the end of a year the same thin edge was of a purple color.

Continued absorption of lead in considerable quantities produces an intense bluish discoloration of the buccal gingival margin several lines in breadth in the gums about the teeth in the upper and lower jaw. The lead sulphide, deposited in the lumen of the vessels, in their walls, and in the perivascular tissue, interferes with the nutrition of the affected portion of the gum and leads to its partial atrophy. The gums become exceedingly hemorrhagic so that the slightest friction causes venous oozing. Their dental margins in some situations become, after a time, fringed, and retracted from the neck of the teeth, exposing a part of the fang. When the blue line is decided, the crown, neck, and the denuded fang surface of the teeth are apt to contain a black deposit of lead sulphide, which persists for months after exposure to the poisoning has ceased, and then gradually fades, giving place to a yellowish-brown stain.

At a variable period following the cessation of the systemic absorption of lead, the extent depending largely upon the severity of the poisoning and whether efforts have been made to eliminate the metal, a bluish-purple discoloration succeeds the bluish in the affected portion of the gum, and it, in its turn, is succeeded by a purplish, and this by a red line entirely without any element of blueness.

In conclusion, I desire to express my indebtedness to Dr. Leffmann for examining the urine of a number of the cases for lead, and to Dr. Hansell for eye examinations.

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