affect specially and directly the second sound, but, in proportion as this condition of the ventricles impairs the propulsive energy of the systole the second sound will be enfeebled and the aortic second sound is usually weak as compared with the left ventricle first sound. The relative loudness of the sounds therefore enters into the considerations from which may be calculated the efficiency of the ventricle.

Very important information is often supplied by observation of the intervals between the first and second and the second and first sounds, the short and long pauses respectively. When, with dilatation of the left ventricle there is resistance in the arterioles and capillaries, which is very commonly the case, the interval between the first and second sound may be prolonged, and as this marks the duration of the systole it shows that the ventricle is endeavouring to cope with the difficulty and to complete its contraction. The short or systolic pause may be so prolonged as to equal the diastolic, or long pause, and the sounds thus become equidistant, the first also having become short and sharp, the only difference between the sounds is one of emphasis or of pitch, and it is often difficult to say which is which. The sounds may be compared when the heart is acting slowly to the ticking of a clock, when rapidly to the puffing of a distant locomotive.

On the other hand, the first and second sounds may be approximated, and as we cannot suppose that a dilated and enfeebled ventricle completes its systole in a shorter time than normal the only possible explanation is that it is quickly brought up short by the resistance in the arterial system and expels but a small proportion of its contents. This abbreviation of the systolic pause is therefore a serious indication of failure of the ventricles, and when carried to an extreme, so that the second sound follows the first immediately and almost seems to overtake it, it is significant of immediate danger.

In two successive attacks of symptoms due to dilatation of the heart when the degree of dilatation appears to be exactly the same, a difference in the length of the systolic pause is sometimes the chief, if not the only, point which makes a difference in the prognosis. The first and second sounds are spaced in the first attack, which is survived and approximated in the second, which proves fatal.

GOULSTONIAN LECTURES ON LEAD POISONING IN ITS ACUTE AND CHRONIC MANIFESTATIONS.

Delivered at the Royal College of Physicians of London.

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[With Coloured Lithographic Plate.]

LECTURE II.

Analysis, Symptoms, and Physical Signs of Plumbism.—Anæmia: Blue Lines on Gums: Lines Caused by other Substances.—Colic: Nature and Cause of: Pulse during Attacks of Colic and in Intervals.—Difference in Radial Pulses.—Difference in Pupils.—Disordered Menstruation.—Liability of Female Lead Workers to Miscarry.—Forms of Lead Paralysis.—Is the Lesion Peripheral or Central?—Sensory Disturbances.—Lead Poisoning and Optic Neuritis: Two Forms met with.—Transient Amblyopia.—Loss of Eyesight for Twenty-one Weeks: Recovery.—Plumbism and Insanity.

Of all the 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 development of anæmia, which becomes gradually confirmed, and is spoken of as saturnine cachexia. Almost from the first day of exposure to lead, hæmatisis is interfered with. I have never found in any lead workers the normal number of red blood cells. The average range varies from 2,500,000 to 4,000,000 in one centimetre. 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 to disturb 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. When present it is a valuable sign, but it may be absent; it generally is so 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 rinsing the mouth and cleansing the teeth; it is simply a deposit of lead sulphide on, and not in, the gum. The other and characteristic line (see coloured lithograph, Fig. iv) 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 are 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 in 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 as the two are generally associated it is 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 most 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 the 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 malnutrition, 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; in some of my own cases it has disappeared very quickly—in one within eight days—but usually from two to three weeks in its minor forms, and three months or longer in the severer cases. This is the limit which I expressed at a meeting of the Royal Medical and Chirurgical Society two years ago, in answer to a question of Dr. 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 disappeared, whilst ulceration might still remain.

In addition to the blue line on the gum there are occasionally to be noticed discoloured patches of mucous membrane (see coloured lithograph, Fig. v), varying in size from a split pea to a shilling, inside the cheek—most frequently met with oppo-

site 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 develop 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 Professor Philipson (see coloured lithograph, Fig. VI). 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 Cumberland, 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, and 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 coloration is noticed depending upon a deposit in the depth of the ulcer.

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 occasional diarrhoea. Colic occurs early after exposure to lead; in one of my cases seven days after. The pain is frequently so very acute and severe, 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 different 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 diarrhoea. 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, which is the true lead colic, and which is generally relieved by pressure; and the other is the dull continuous pain which follows it in the intervals. It is this pain which is usually aggravated by pressure. 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 whipcord. 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 constric-

tion of the small intestine 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 unstriated muscular fibre—more particularly that of the intestine, no matter the channel by which it has been introduced into the system—as, for instance, 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. Blue patches are occasionally noticed in the intestine.

We cannot quite easily dismiss the 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 Guttman have shown, by reflex inhibition through the mesenteric and coeliac plexuses of the abdominal sympathetic; the fibres which convey the impression being sensory and running, 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.

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 aneurysm, where there was no positive sign of pressure upon the thoracic sympathetic; and the other interesting fact is that where there have been repeated attacks of colic, or where there has 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 than in the

Sphygmograms showing heightened Arterial Tension during Lead Colic; also Inequality of Pulses in and after Attacks of Lead Colic.

A. K. aged 27, lead worker, taken at factory, October 2nd, 1890, shows normal tension.

A. K., right radial, has recently had colic, is still occasionally pained, December 24th, 1890.

A. K., left radial, December 24th, 1890.

C. McG., aged 19, October 2nd, 1890, taken at factory, never had colic.

C. McG., admitted into Infirmary October 28th, 1890, is suffering from lead colic, right radial.

C. McG., left radial, October 28th, 1890.

H. E., taken when cramp-like pains passing off.

H. E., colic had quite disappeared.

M. C., aged 17, is experiencing her first attack of lead colic, right radial, shows difference between the two radial pulses noticed in colic, November 21st, 1890.

M. C., left radial, November 21st, 1890.

W. McF., a paint mixer, severe colic, left radial, December 29th, 1890.

W. McF., right radial, December 29th, 1890.

E. B., lead poisoning, October 31st, 1886.

E. B., lead poisoning, shows increased tension, since last tracing; November 7th, 1886.

C. B., aged 34, ship painter, right radial, colicky pain in left abdomen only, December 20th, 1890.

C. B., left radial, pulse of higher tension on same side as abdominal pain.

E. H. (Mr. Dodd and Dr. Bowlan's case and series of tracings), lead optic atrophy, right radial, August 1st, 1890.

E. H., left radial pulse, has profuse palmar perspiration left hand, shows lower arterial tension, August 1st, 1890.

E. H., right radial, August 22nd, 1890.

E. H., left radial, August 22nd, 1890.

E. H., right radial, October 6th, 1890.

E. H., left radial, October 6th, 1890.

E. M., aged 21, lead colic, right radial, October 25th, 1890.

E. M., left radial, shows pulses to be unequal in the radials, October 25th, 1890.

C. R., aged 23, severe headache, lead poisoning, June 28th, 1889, made good recovery, returned to lead works, followed her employment (vide next tracing).

C. R., aged 24, completely blind, marked optic atrophy, no albumen in urine, July 14th, 1890.

B. R., aged 23, acute lead encephalopathy, convulsions, tracing taken few hours before death, patient comatose, urine contained albumen, December 5th, 1890.

T. L., a white lead worker, aged 25, taken a few days after convulsions, had lost all memory, shows high tension, no albumen in urine, February 22nd, 1890.

M. W., aged 39, lead encephalopathy, optic neuritis, April, 1889.

other. Admitting that even in health there is a degree of difference in the fulness and tension of the two 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 the 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.

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 17 to 24—I find nearly 50 per cent. of those engaged at the works; 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 percentage than other women. Dr. Barnes¹ alludes to the subject of abortion and lead poisoning. He quotes the experience of M. Paul as to the 15 pregnancies of four women who had worked in a type foundry; 10 of these ended in abortion, 2 in premature labour, 1 in stillbirth, and 1 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 36 pregnancies; of these, 25 ended in abortion, 1 in premature labour, 2 in stillbirths, and 5 children died, 4 in the first year. A woman had 5 pregnancies, all ending in abortion; she left the factory and bore a living child. Another, having ceased work at the factory, had 2 children; she returned, had 2 abortions. This is also my experience: In one of my cases (E. C., aged 27) 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 39, lead worker for eighteen years, has had 12 children, of whom 4 are now alive; the remaining 8 died at ages varying from 5 days to 4, 6, and 14 months from convulsions. She suckled her children whilst following her employment at the lead works. She has had in addition 5 miscarriages; 3 of these were in succession. There is no specific history, no albuminuria, but she is anæmic. In another case, M. A., aged 40, whose mother, too, had been a lead worker, we have a history of 8 children, all of whom died of convulsions. M. C., aged 24, 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. M. A. T., aged 25, had one living child at full 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 cause abortion, or, if a child is born, it is ill-nourished, and dies almost immediately after its birth, generally from convulsions.

The nervous system suffers severely in lead poisoning, 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, undergo atrophy, or cerebral symptoms supervene, including convulsions and coma. Boerhaave was amongst the first to associate paralysis with saturnine poisoning, and following him Stockhusen, van Swieten, and de Haen, but it is to Tanquerel des Planches and to Duchenne of 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 subacute 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. Gowers² 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.

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

Whilst the poison has a special predilection for the muscles already described, these may escape and the muscles of the other arm may be involved—those which constitute the Duchenne-Erb group, namely, the deltoid, biceps, brachialis

¹ *Obstetric Medicine and Surgery*, vol. i, p. 496.

² *Diseases of Nervous System*, vol. ii, p. 70.

anticus, and the supinator longus, and to these are generally superadded paralysis of the supra- and infraspinatus. This form of paralysis, usually bilateral, occurs in the more inveterate types of lead poisoning. Of the muscles enumerated,

Fig. VII.—Double wrist-drop.

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.

Occasionally certain muscles of the legs are paralysed; this occurs, according to Tancquerel, 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 predi-

Fig. VIII.—Paralysis of extensors of fingers; little finger has escaped.

lection 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. Tancquerel 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. 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 *en bloc* 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 incapable even of eating; the intercostals, the diaphragm, and muscles of the larynx are involved. The patient suffers from dyspnoea and loss of voice. The noteworthy feature in these cases is that the muscles of the head and neck are respected. Rapid as is the development of this form of paralysis, it just as rapidly begins to mend, 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.³

Heugas, *Contributions à l'Etude de la Paralyse saturnine*, p. 54.

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 29, single, a white lead worker, was admitted complaining of symptoms of lead poisoning of seven weeks' duration. Patient has followed his occupation for the last three years and a half; has been engaged principally in the drying department. This work consists of drying powdered white lead in an oven. It was not until the end of two years and a half that he experienced his first attack of lead poisoning, namely, colic and constipation. After fourteen days he returned to work quite well, where, after working for three months, he had a second attack of colic, from which he recovered in five weeks. After this second attack of colic the patient drank large quantities of beer and water acidulated with sulphuric acid, and was extremely careful as to cleanliness. Seven weeks before admission the patient went to work in his usual health, and whilst in the factory was suddenly seized with gripping 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.

Fig. IX.—Complete paralysis of both arms.
(Professor Philipson's case.)

Paralysis suddenly appeared. It attacked first the right leg. At 6 A.M. the patient was out of bed and could walk unsupported; but on rising five hours later he was surprised to find that his legs gave way under him; they helplessly crossed and he fell to the ground. The right leg was most affected, but before morning they were equally paralysed. The next day the arms became paralysed with the same suddenness, the right again being the worst. Other muscles of the body quickly followed, 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 perform 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.

Fig. x.—Complete paralysis of both arms and muscles of back. (Professor Philipson's case.)

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. (Figs. ix and x.)

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.

Minor degrees of this general form of paralysis are also met with. Sir Dyce Duckworth⁴ 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, also the subcutaneous nodules and gout. The muscles of the upper parts of the arms and scapulae, 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 anæsthesia⁵ 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. Hyperæsthesia amounting to actual pain has been noticed by Baigent, and described by him in his thesis on peripheral neuritis.

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*, part 11, 1890, where there was ptosis of the right eyelid, with abolition of movement of the upper, lower, and internal rectus; the pupil was dilated and did not react to light. In many of my own patients there has been 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 faradaic. 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 myopathists, 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 nerve-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? Remak⁶ 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 which 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, which 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

⁴ *Clinical Society's Transactions*, vol. xx, p. 266.

⁵ Oliver, *BRITISH MEDICAL JOURNAL*, 1885, vol. ii, p. 732.

⁶ *Archiv. für Psychiat.*, vol. ix, part iii, p. 510.

To illustrate Dr. Oliver's Goulstonian Lectures.

FIG. III.—Acute Neuro-retinitis in Lead Poisoning.
John F. A., aged 29.

FIG. II.—Optic Atrophy in Lead Poisoning.
Cath. R., Sept. 20th, 1890.

FIG. I.—Acute Optic Neuritis in Lead Poisoning.
Cath. R., June 26th, 1890.

FIG. VI.—Black line on teeth at margin of gums, probably carbon, in a miner.

FIG. V.—Blue line, ulcerated gums, and blue patch inside lower lip in lead worker. Female.

FIG. IV.—Blue line in gums in lead worker. Rapid development of exposure to lead dust.

each instance being very small? The myopathic theory, however, need no longer detain us; for Heubel has also demonstrated that with equal weight 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 been partly in favour of the neuropathic theory, which assumes that in lead paralysis the intramuscular 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. Why, then, should the 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 intramuscular 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 these 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.

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, suddenly lose their sight and become convulsed. Sight may be regained, or it may be permanently destroyed. The coloured lithograph (Fig. III) represents 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 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 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 these 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 centred around it. Some observers, notably Jeaffreson,⁵ 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; or complete recovery or to optic atrophy, followed by permanent blindness—without the least trace of albumen having appeared in the urine.

Acute Lead Poisoning: Blindness: Recovery: Fresh Exposure to Lead: further Loss of Eyesight for Twenty-One Weeks: Recovery of Sight: Atrophy of Discs.—C. R., aged 23, single, a white lead worker, was seen by me along with Dr. Bowlan at the Newcastle Workhouse in July of last year, 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, 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 wrist, 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. Mr. Dodd and Dr. Bowlan found the urine to be quite free from albumen on the date of her admission—May 14th, 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 (coloured lithograph, Fig. 1), 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 July 30th I brought her into the Newcastle Infirmary. This was eight weeks after the onset of her illness, and she was still totally blind, otherwise she was 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 October 10th, that is, exactly twenty-one weeks to the day on which she became

⁵ Clinical Lecture, Optic Neuritis, *Lancet* May 3rd, 1890.

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 coloured lithograph (Fig. 11) are atrophied. In one of Mr. Baumgartner's cases sight was restored after a period of five weeks' blindness.

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. Leadsalts, or the consequences of 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, that therein may lie an explanation of the causes of optic neuritis. As bearing upon this, the observation of Schreiber¹⁰ 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. Mr. Baumgartner, who has medical charge of lead factories in Newcastle, tells me that he has seen this on a few occasions. It was Jaccoud who introduced the comprehensive term of cerebro-spinal saturninism, so as to include the great variety of nervous symptoms met with in lead poisoning. The presence of anaesthesia has rendered the diagnosis of lead poisoning difficult. George 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. 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 anaesthesias, even when they occur in the male. It was Debove and Achard, however, who first employed the term toxic hysteria to indicate cases in which the neurosis was developed under the influence of intoxications. 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.

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,¹¹ 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 subarachnoid 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 for 1871, Dr. Maccabe¹² 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 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 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 a well-marked line on his gums, but there was neither paralysis, anaesthesia, nor hyperaesthesia; 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 questions before replying to them. He regained his memory.

EXPERIMENTS WITH TUBERCULIN ON CATTLE.¹

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A FEW weeks since I had sent to me for examination a tumour excised by Mr. Douglas, veterinary surgeon, Kilmarnock, from the cervical region of a cow. On section this tumour presented the usual macroscopic appearances of a lymphatic gland in an advanced stage of tuberculosis, and a cover-glass preparation from it showed a large number of tubercle bacilli. Mr. Douglas suggested that the cow would form a suitable subject upon which to test the effect of Koch's fluid, and the animal was accordingly sent by rail to the Veterinary College, where she arrived on February 19th.

The cow was of the Ayrshire breed, rather under-sized, and in poor condition. Pulse (56) and temperature (fluctuating between 100° and 102° F.) were almost normal. The appetite was good. The animal coughed occasionally, but the respiratory movements were not otherwise notably abnormal. In front of the right shoulder there was a large suppurating wound marking the site of the tumour above referred to; at the corresponding point on the left side there was a firm tumour as large as the two fists, and a similar but somewhat smaller enlargement was present in the parotid region on the left side. These tumours had the usual characters of hyperplastic tuberculous lymphatic glands.

First Experiment.—On February 21st the cow received 20 milligrammes of Koch's fluid.² The injection was made on the chest wall immediately behind the left elbow, the hair

¹¹ *Journal of Mental Science*, July, 1886.

¹² *Ibid.*, 1872-73, p. 233.

¹ A more detailed report will appear in the *Journal of Comparative Pathology*.

² For the lymph used in these experiments I am indebted to Professor Greenfield and Dr. Felkin.

¹⁰ *Deutsches Archiv für klin. Med.*, 1878, p. 99.