A CLINICAL LECTURE

ON
LEAD-POISONING.

By THOMAS OLIVER, M.D., M.R.C.P.Lond.,

Physician to the Infirmary, Newcastle-upon-Tyne, and Lecturer on Practical Physiology, University of Durham College of Medicine.

It is seldom that a physician to a general hospital has so many as five cases of lead-poisoning under his care at one and the same time. Most of you have seen these cases with me in the wards up-stairs, and have heard my remarks upon them at the bedside; but, as one of the cases has terminated fatally, I wish to-day to draw the attention of this class to the general question of lead-poisoning.

In and around Newcastle are several lead-factories, from which we draw most of our cases. It is amongst people who are engaged in its manufacture, or amongst potters, who use it for glazing purposes, that the symptoms of lead-poisoning are chiefly met with, and not, as a rule, amongst those who handle the metallic ore—the lead-miners. In fact, so little is lead-poisoning known amongst lead-miners, that Mr. Montgomery, of Blanchland, who has practised for forty years amongst them, has never met with one case. It is different with the smelters, or those who reduce the ore to pig-lead. From time to time; of course, symptoms of accidental lead-poisoning arise, owing to the employment of contaminated articles of food, and the use of drinking-water (soft in character) which has lain in leaden cisterns or pipes. You have learnt elsewhere how the presence of certain salts—nitrates, nitrites, and chlorides—increases the solvent action of water upon lead; and, on the other hand, how others—chiefly salts of lime, phosphates, carbonates, and sulphates—by becoming encrusted upon the interior of these vessels, prevent the water becoming impregnated with lead. Great reason, therefore, for us exercising care that the drinking-water which reaches the interior of our houses should come direct from the main, and never be stored; and that pipes of galvanised iron should take the place of lead. I know of one gentleman (a clergyman) who suffered from lead-poisoning, as the result of his drinking water daily from the tap attached to a leaden pipe in his garden, where he was wont to say he always found the water coolest and most refreshing. Here be it remarked, the evil effects of lead are not due to the swallowing of large doses, repeated for a short time, but are due rather to the entrance into the system of lead in infinitesimal quantities, regularly and for an extended period.

The subject of my remarks, G. S., aged 33, a lead-refiner from Wear-dale, was admitted under my care on December 9th, 1884, complaining of headache, fits, loss of vision, and epistaxis. In the early part of 1883, the patient had been in the infirmary, under my care, for loss of power in both arms, particularly the wrists. At that time, there was a history of his having had fits ten weeks previously, and, during his residence up-stairs, he had a very severe hæmoptysis, which was never repeated. A loud, rough, systolic *bruit* was detected over the aortic area, followed by an accentuated second sound. The urine had a specific gravity of 1010, and contained a small quantity of albumen. Under the use of iodide of potassium, and the application of electricity, the patient recovered to a very great extent the use of the extensors of each forearm. Returning to Stanhope, he went back to the lead-factory, where he was employed in smelting the ore, and latterly in working a crane. This he continued at until last October. He had been suffering from frontal headache, accompanied at times by vomiting and a dimness of vision, but he still followed his employment. The serious state of his health only dawned upon him when one day, at a pigeon-shooting match, he found that all at once he could not see. He had not had drink, nor could it be said of him that he had ever been intemperate. The loss of sight was only temporary; for he tells us that he followed his employment for a few days after this. His power of seeing gradually decreasing, he relinquished work—a step hastened by attacks of epistaxis, which frequently recurred. Beyond the nature of his employment, there is nothing personal that calls for mention. He never suffered from syphilis; his father died of apoplexy at the age of 56; and he lost one brother nine months ago from lead-poisoning, in whom the symptoms of lead-poisoning were at first "dropped wrist," and, latterly, symptoms of a cerebral type. At the present time, the patient has five brothers working in the lead-factory. All of them are well. With the exception of the illness which brought

[1294]

him to me two years ago, the patient had always enjoyed the best of health.

Towards the end of November, and without any warning, the patient had a fit, epileptiform in character, which rendered him unconscious for some little time; and, on the night before his admission, he had a severe bleeding from the nose, which lasted from 9.30 p.m. until 2.30 a.m. of the following day.

On admission, I found him sallow and cachectic, his cheeks somewhat pendulous, fuller than usual, and expressionless. There was slight paresis of the left cheek; he could depress both angles of the mouth, but had great difficulty in elevating them. When the tongue, which was moist and clean, was protruded, it was noticed that the left angle of the mouth overlapped it more than the right. His teeth in front were fairly good, but were remarkably long, owing to a retracted condition of the gums, which exhibited a blue line. He did not complain of any bad taste in his mouth, but his breath was very fetid. There was no anaesthesia of the skin of the face. The arms were not so plump as they should be for a man of his age. As for his right arm, its muscles were attenuated. He could supinate his hand well enough; but, when it was lying in a prone condition, he could not extend the wrist nor the fingers; paralysis of the extensors was most marked in those of the index and little finger. When asked to extend his fingers, it was noticed that the whole of them were thrown into a state of disordered movement, during which the movements of flexion were seen to overpower his abortive attempts at extension. There was slight anaesthesia and analgesia over the back of the wrist. On the front of the arm, the veins were seen to be unduly distended, especially about the bend of the elbow. Sensation was normal over the arm and upper part of forearm and ball of thumb; while over the front of wrist, palm of hand, and finger-tips, there was diminished sensation to touch and pain. The interosseous spaces on the back of hand were hollowed out, and the phalangeo-metacarpal joints were more prominent than usual; but, neither grating was felt nor pain experienced in them. In the left arm, the appearances were the same as in the right; there was a similar distribution of the loss of muscular power, and of the loss of extension, only not quite so well marked as in the right. His legs were fairly developed. The knee-jerk was normal. The plantar reflex was slightly exaggerated on the right side—but, with that exception, his sense of touch and pain was normal.

Chest.—The apex of the heart was felt beating in a line with the left nipple, and about 2½ inches below it. Over this area was heard a well marked reduplicate first sound, followed by accentuated second sound. On tracing this reduplicate first sound upwards, in an oblique direction to the right, it became a loud rough systolic murmur, heard most particularly over the aortic area, where, too, the second sound was deeply booming in character. In the vessels of the neck the systolic *bruit* was only faintly heard; the second sound was very distinct. Over the apex of the left lung, the inspiratory murmur was interrupted; the expiratory was prolonged. The pulse was 104. The vessels were hard and incompressible.

Eyes.—The border of the right disc was extremely ill-defined; it could not be separated from the adjacent retina. The disc exhibited a peculiar marbled or mottled appearance; vessels could not be traced in their entirety for any distance. Surrounding the vessels could be seen here and there numerous white patches, most marked on the retina. The retina was generally pale, and studded with numerous patches of a glistening white chalky colour; a few of the retinal vessels had ruptured. The border of the left disc could not be defined except inferiorly and internally. Here the disc was atrophied, and extremely pale. Just below the central point of the disc, a few hæmorrhages could be seen. In the retina was a large white field, on which small greyish-white points could be seen. Surrounding the vessels were masses of white exudation. The disc itself was mottled in appearance, and showed traces of hæmorrhage.

On microscopic examination of the blood, the white corpuscles were found to be slightly increased, as many as from eight to ten being seen in the field; they were, if anything, larger than normal, and extremely granular; the red seemed healthy, and formed rouleaux. The number of red corpuscles was diminished; of three enumerations with Gowers' hæmacytometer, there were only found, on an average, 2,636,666 in a cubic millimetre, a little more than half the usual number met with in health.

Urine was passed in small quantity latterly, but on admission as much as three pints daily was passed; it had a specific gravity of 1010; contained about one-eighth of albumen, which gradually increased to about one-third.

As time wore on, a large bulla appeared on the under surface of the tip of the right middle finger; it gradually filled with pus, and was

successfully treated by incision and poulticing. A few days after this, the patient began to complain of great pain in the right foot. This was soon followed by the appearance of a large bulla, about the size of a shilling, on the under surface of the big toe. Its contents at first were serous, afterwards purulent.

From day to day, the patient varied very little; his course was downward from the day of his admission. Sometimes, at our visit, we found him completely blind; at other times, he could not only see us, but could count fingers held up before him. Occasionally taking the place of this blindness, which was temporary, there was deafness. Frequently, he was delirious, almost maniacal. He would at times keep shouting for people whom he named; would get out of bed, seemingly unconscious of what he was doing, but he was never severely convulsed. His pulse kept high, always 104 to 112. Epistaxis recurred pretty frequently, leaving him weaker and weaker. The fits, though frequent, were minor in character. During his sleep, which was heavy and at times comatose, his breathing was stertorous. Debility gradually increased; dulness on percussion was detected over the base of each lung, and here moist crepitation could be heard. On the evening of January 19th, 1885, I found him sitting by the fire, breathing very heavily and deeply asleep. His pupils were dilated just beyond the normal, and did not react to light. He was unconscious. In a few hours, he regained consciousness, but died during the night, comatose.

A *post mortem* examination was made on the next day, of which the following is an abstract. The lower extremities were cedematous. Both pleural cavities contained serum. The right lung was adherent at the apex; on section, it was almost entirely airless; the upper lobe was cedematous; the left lung was in a similar condition. The heart was very much enlarged; the left ventricle was hypertrophied, its walls being at least three-fourths of an inch in thickness; the right ventricle was normal, both in regard to cavity and to thickness of its walls. The tricuspid orifice admitted four fingers readily, and the mitral orifice three. The aortic valves were very slightly incompetent. The anterior right cusp, on its ventricular aspect, presented just beneath the corpus Arantii a firm elevated body, about the size of half a small orange-pip. The valves were otherwise healthy. There was a slight atheromatous deposit in the wall of the aorta above the valves. The mitral curtains were slightly thickened. Weight of heart, 1 lb. 6½ oz. The liver presented nothing remarkable. The spleen weighed 10½ oz. The capsule of the right kidney was adherent; on removing it, a portion of the cortex was torn away with it; the surface was granular. On section, the cortex was seen to be much diminished, in some places being only a line or two in thickness. The structure was generally firm and fibrous. The organ weighed 5 oz. The left kidney presented similar appearances; it weighed 4½ oz. In both kidneys small cysts were found filled with clear fluid. The vessels at the base of the brain were normal. The substance of the brain on section was seen to be pale, sodden, and cedematous. Beyond this, nothing abnormal was detected.

Sections of the kidney, on microscopic examination, showed a marked increase of stroma, the corpuscles of which had undergone great proliferation. The tubules were here and there almost obliterated; many of them were distorted; some were lined with a layer of small round cells, while others were completely empty or contained heaps of broken down cells. The stroma was generally increased, but in some places the hyperplasia was more marked than at others. Many of the glomeruli appeared healthy, while others had their capsule thickened; others, again, were shrunken and appeared more like small irregularly shaped masses of fibrous tissue than anything else. The walls of the arteries were much thickened; so much was this the case with the smaller vessels, that they looked as if they were occluded. The capsule of the kidney was thickened. Sections of the medulla oblongata, examined microscopically, showed nothing abnormal beyond marked thickening of the small vessels.

REMARKS.—Amongst the earliest symptoms of plumbism which attract our attention, I should feel inclined to place cachexia. Previously to this, there has been experienced by the patient a general feeling of ill-health, and nutrition has not been maintained. An anæmic condition is soon developed, in which the skin acquires a dull earthy hue. To the dull, anæmic, and somewhat listless look, there is frequently added a peculiar fulness of the cheeks, that we are often able, especially in the case of women, to diagnose lead-poisoning when we see the patients in the streets. Many of them are young women. They have the anæmic look of patients who have lost largely of blood, without presenting anything like an uterine facies. On the other hand, the appearance is different from the greenish pale hue of chlorosis. Associated with, or closely consequent upon, this saturnine cachexia, there is generally digestive derangement; constipation, with

colic; the breath is fœtid; the tongue foul; the teeth discoloured; the gums retracted, showing an unusual length of teeth, and exhibiting a blue line at the junction of teeth and gum. I have not noticed vomiting as a frequent symptom in lead-poisoning, unless the kidneys have been affected or head-symptoms predominant; nor can I say that I have found the pulse slower than in health, as some assert. It is rather the reverse of this; for, in most of my cases, the pulse has been higher than normal, even when the kidneys have been affected, strange to say. Sooner or later, there is developed paresis of the extensors of the forearm, which gradually deepens into a true paralysis. Many of these muscles undergo atrophy. This condition is due partly to disuse; but, as the atrophy extends to other muscles which are not paralysed, and is of itself at times a symptom of lead-poisoning—its seat being frequently the muscles between the thumb and forefinger—the cause of the atrophy must be something other than want of use of muscle. The extensors of the forearm are the muscles generally and most deeply paralysed, but the loss of power is by no means confined to them. Frequently they are spared, and the muscles of the leg affected, as in one case I have seen, where the peronei are involved. In those cases where “dropped wrist” occurs, the symptoms of lead-poisoning have been as a rule slowly developed. You have this taking place without any marked disturbance of the central nervous system, and with or without the presence of albumen in the urine. Many of these patients have been employed in the lead-factory for years, and only by degrees have they felt their health give way. It is otherwise, however, with some; in not a few—and I refer particularly to young females—symptoms of lead-poisoning are often suddenly developed, a few months of work in a factory being sufficient to call forth the most alarming symptoms—extreme headache, dimness of vision, and fits of an epileptiform character. In these, the urine may or may not be albuminous. Frequently, the optic disc is seen to be the seat of very marked changes. What is of interest in these cases—the most dangerous because they are the most acute—is that the symptoms often come on suddenly, are frequently the first warning that the patient has of being poisoned. Once having suffered in this manner, the patient is thereafter completely disqualified for undertaking further duties in the factory. No medical inspector would allow, if he knew, such a woman to begin work again amongst lead; experience having shown that a renewed brief exposure to the poison is sufficient to bring back the symptoms with tenfold vigour.

Of the eighteen cases of lead-poisoning which have been under my care during the last six years, eight exhibited “dropped wrist”; one suffered from right hemiplegia, with aphasia; one from a peculiar tremor of the arm and leg. Dropped wrist was double in seven of the eight cases, and one had ataxia of the arms. Of the remainder, five exhibited cerebral symptoms; one had marked general muscular atrophy; one is suffering from cirrhosis of the liver. In most of the cases, we find that the patient, after having suffered from colic, becomes aware of a gradual loss of power in the muscles of the forearm. This paresis passes into a true paralysis, which shows itself, generally, at first in the extensor communis digitorum, and extends to the other muscles supplied by the musculo-spiral nerve, with the exception of the supinator longus. With this loss of voluntary control, the muscles also lose their faradic contractility; and, according to Duchenne, “the extensor communis digitorum, extensor indicis, extensor minimi digiti, and the extensor secundi internodii, are the first to suffer in their electro-contractility.” It is only, however, when the extensores carpi radiales longiores et breviores, and the extensores carpi ulnares are paralysed together, that the wrist drops, and the patient is unable to extend them. Pronation and supination are not, as a rule, interfered with. The loss of voluntary power and electro-contractility is frequently followed by atrophy; and here I would again remind you that these changes may proceed to muscles beyond the forearm. In one of my cases, there was pretty general muscular atrophy. At times, the muscles of the ball of the thumb become affected, and when this is the case, the appearance is not unlike that seen in progressive muscular atrophy. The slow development of progressive muscular atrophy, and the fact that the wasting precedes the loss of the electrical contractility, would help you to distinguish between the two diseases, bearing in mind, however, that a progressive muscular atrophy may be the result of lead-poisoning. As a rule, paralysis from lead-poisoning is double and symmetrical. In only one of my cases was it confined to the muscles of one forearm. Loss of sensation is less commonly met with. In G. S.’s case, there was marked anæsthesia and analgesia over the back and front of the wrist, the palm of the hand, and the finger-tips. I have not seen any mention of tremor as a symptom of lead-poisoning. In one of my cases—a male, kindly sent to me by Dr. Beatley—there is a peculiar shaking

of the left arm and leg. The man has worked for twenty-eight years in a pottery, his duty being to dip the jars into a solution of lead, and place them in the oven. He admits having drunk beer pretty freely, but states that he always enjoyed good health until two years ago. The tremor is very noticeable; beginning in the left thumb, the shaking extends up the arm, throwing the limb into a state of violent tremor, over which he has no control. It is not a painful shaking, nor does it altogether prevent him following his occupation, though he says it is very detrimental to him. It is only within the last six months that the shaking has extended to the left leg, but here it is not so bad as in the arm. Strange, too, the shaking never begins in the two limbs together at the same time, and never in the leg first. It is always conveyed from the arm to the leg; the leg may be in a state of complete rest, while the arm is violently agitated. In his case, there is no marked paralysis; the grasp of the left hand is feebler than that of the right. The shaking of the arm can be stopped by forcibly extending the thumb. In the left leg, though there is neither knee-jerk nor ankle-clonus, foot-trepidation can be easily induced by causing the patient to bear his weight upon the toes. His gums show a well marked blue line; his urine is not albuminous.

The presence of a blue line on the gums I regard as a very important sign of lead-poisoning, but only when accompanied by other symptoms. In thirteen of the eighteen cases, it was present; of the remainder, some had had a blue line a few years ago, and had lost it. I make it a rule to examine the gums in cases of suspected lead-poisoning; and, in its absence, would attach very great importance to the character of a paralysis, or muscular atrophy if present, and to the facial appearance—pale and rounded. Buzzard (*Diseases of the Nervous System*) has, in the absence of blue line, detected lead-poisoning by the seat of the paralysis, and the diminution of the excitability of muscles to faradism. Knowing that the presence of a blue line on the gums is quite consistent with good health—that it may disappear, and return after the use of potassium iodide—and that it is seldom, if ever, met with in people who scrupulously use the tooth-brush, you see why we attach little importance to this sign alone. If present, however, it is a valuable aid to a diagnosis. It is due to the formation of sulphide of lead—caused, as Mr. Tones has shown, by the decomposition of food left about the margins of the teeth and their interstices; the sulphuretted hydrogen thus liberated acts upon the lead which is in the tissues, and causes its deposition. I regard this as the origin of the sulphur rather than the sulphocyanic acid of the saliva. Hilton Fagge showed that the discoloration of the gums is not uniform, but is distributed in loops corresponding to the vascular papillæ of the mucous membrane, that the pigment consists of granules, some of which are deposited inside, others outside, the small blood-vessels. The lead, to be acted upon by the sulphuretted hydrogen, is circulating either within the blood-vessels, or is in the nutritive fluid which bathes the tissues. Precipitated from the blood in the manner described, it would explain the atrophy which the gums undergo at their junction with the teeth; the sulphide of lead, by obstructing the circulation through the capillaries, would interfere to a greater or less extent with their nutrition.

In 11 of my 18 cases, colic was present; that is a smaller average than most have noticed. Tanquerel found colic present in 12 out of 14 cases, and Dr. Porter, of Sheffield, in his very interesting thesis on lead-poisoning, found it present in 27 out of 30 cases. Combined with colic, there is generally a history of constipation, occasionally of nausea and vomiting. As a rule, the attacks do not last long; rest from work and the use of saline purgatives, sulphate of magnesia and iodide of potassium, and the use of baths, being sufficient in most to bring about a cure in a few days.

Of the tendinous swellings, mentioned by Ferrier as forming on the tendons at the back of the wrist, and of neuralgic pains in the muscles and joints, I have not seen anything, nor do we in Newcastle see anything of gout and its relation to lead-poisoning—a strange fact, knowing the frequency with which it is noticed by London physicians. We get the kidney-affection, as I shall speak of later on. I have met with rheumatism, but in Newcastle I have never yet seen gout caused by lead-poisoning. In this, I believe I am giving expression not only of my own experience, but of that of my colleagues. I find menstruation profuse and frequent, a very common symptom amongst all classes of women, and the same remark applies to abortion. How far this menorrhagia has to do with causing the peculiar anæmia or cachexia, it is impossible to say. The fact that saturnine cachexia is met with in males as well as females, shows that it is not all due to loss of blood; that some specific effect, in other words, is produced by lead upon the blood. The blood examined by Gowers' hæmacytometer

showed in every case a marked diminution of the coloured corpuscles, from three to four millions in each cubic millimetre.

I have noticed, too, slight increase in the number of white cells. There was, I may remind you, frequent and profuse epistaxis in Sanderson's case. As for the large bullæ, which suddenly, and with pain, appeared on the hand and foot, and which soon became purulent, I offer no explanation.

Of the cerebral phenomena met with in lead-poisoning, and to which the term *saturnine encephalopathies* has been applied, epileptiform convulsions are the most common. By many writers they are regarded as occurring late in the disease. I cannot say this is my experience. Preceded, as these fits generally are, by intense headache, vertigo, and dimness of vision, we meet with them, and that without prodromata, in young women who have worked only a few months amongst white lead. Too frequently, indeed, they are ushered in suddenly, and are the first indications of lead-poisoning. I am not referring to the headache, vertigo, and epileptiform convulsions due to uræmic poisoning, but to those which occur early in the course of the disease, and when the urine is free of albumen. Such symptoms we can only regard as specific, and due to a special action of lead upon the brain. The convulsions which come on late in the course of the disease, when the urine contains albumen, are in all probability the result of uræmia. In the brains of those who have died from lead-poisoning under my care, who, during life, suffered from convulsions, nothing abnormal was found after death, more than the hydræmia (pallor and oedema) seen in Bright's disease. No lead was found in a portion of Sanderson's brain, submitted to chemical analysis by Professor Bedson—a fact which forces us to acknowledge in his case a causal relationship between the kidneys and the fits. It is more difficult to account for the noisy, and, at times, maniacal, delirium which was present. At times loud, boisterous, and unconscious, Sanderson could occasionally be suddenly brought to understand where he was, and to answer rationally, only to wander off again, however. The subject of transient deafness, he was also the victim of temporary amblyopia. Completely blind when we approached his bed, he frequently became able to see us, and even to count objects held before him ere we left. When conscious, he was always in a state of dread, and his one complaint was intense weakness.

Though a late symptom, in the case here recorded, obscurity of vision, temporary or permanent, and even complete loss of vision, are frequently met with early in the illness. In the transient form of amblyopia, which comes on suddenly, and disappears just as suddenly, no changes may be observed in the disc or retina on ophthalmoscopic examination. An anæsthetic condition of the retina probably explains these cases. That lead acts directly upon disc and retina, there is little doubt. There is a neuro-retinitis due to lead-poisoning. I do not believe that the intraocular changes in plumbism are always secondary to disease of the kidney. In many of my cases, marked neuro-retinitis existed long before albumen appeared in the urine; nay, more than that, I am inclined to think, from Sanderson's case, that a plumbic and albuminuric neuro-retinitis may run side by side. A girl, who is at present upstairs, has most marked optic neuritis, associated with intense headache, but the urine is entirely free from albumen. She had only worked a few months in the lead-factory, when cerebral symptoms developed, and we find her to-day with her optic discs swollen, their edges obscured, the arteries small, and veins swollen, no hæmorrhages. Sanderson's case was one of longer standing, and in him the discs were mottled, their outline irregular, and borders ill-defined, while here and there large patches of hæmorrhage were noticeable, and the vessels were obscured in their course by a dense whitish exudation. It is difficult to say how much of this was due to the disease of the kidneys. A pale zone surrounding the retinal arteries, I have occasionally noticed, in those who are suffering from lead-poisoning, and in whom there is no albuminuria. In others, the disc is the seat of a primary atrophy. Here the sight is naturally much affected, and the blindness is progressive; conditions explained by the extreme change which the nerve-expansion has undergone. For an atrophied disc there is little hope; for an oedematous one, always the chance that the fluid may be absorbed, and the nerve-terminations be little the worse for their temporary compression.

Garrod (I quote from Warburton Begbie's papers, Sydenham Society's publication) was the first to draw attention to the relationship between lead-impregnation and gout. Although we see many cases of lead-poisoning in Newcastle, we seldom meet with gout as one of its manifestations. I have seen one or two cases in which there has been co-existent rheumatic arthritis, of the wrists particularly, and three or four in which the heart and pericardium have been affected; but I do not remember seeing one case in which gout has been associated with lead-poisoning, although after death the kidney

has been found contracted and puckered, but not red. It is possible that the explanation lies in the different habits of the working-classes in London and the north. In both of the cases recorded by Dr. Begbie, there was a history of the intemperate use of beer and porter, drinks which have always been regarded as having an influence in the development of gout.

Dr. Garrod found, in his cases, that the blood was rich in uric acid, while the urine contained a deficiency of it, and in Dr. Begbie's two cases the amount of uric acid eliminated from the system was diminished. In the early stage of all my cases, the daily amount of urine was much in excess of the normal. It was of low specific gravity, and always contained a marked deficiency of urea. This has gone on for long, without albuminuria appearing, and what are we to regard as the explanation? When albumen has been present in the urine, the deficiency in the elimination of urea has not been interfered with. I think pathologists have laid too much stress upon interstitial nephritis as being the constant and typical anatomical change met with in the kidney in lead-impregnation. The kidneys I have seen have been certainly very much smaller than they ought to have been. Their cortex has been adherent, their stroma has been increased, but there has been a degree of tubular, as well as intertubular, nephritis. Many of the renal cells have been seen to be voluminous, their contents undergoing fatty granular degeneration, while others have completely broken down, and filled the tubules with their debris.

I am inclined to believe that lead acts as a special poison to the renal cells—paralyses their function, and hence, as the result of this low form of irritation, of which the kidney is the seat, more blood naturally passes through it in a given time, and the balance of the removal of waste is sought to be attained in the larger quantity of urine which is eliminated. This in no way specially raises the question of whether urea exists preformed in the blood of the renal artery, the renal cells simply removing it, or whether the urea is formed in reality in the renal cells themselves. If these cells have their function paralysed, then, according to the one theory, the urea which is already formed will go on increasing in the blood, producing its own special effects; whereas, by the other theory, only its elements would accumulate.

Physiology teaches us that urea exists in the blood of the renal artery, and that the liver is the organ which contributes most largely to its formation, the primary source being the nitrogen of the food, and yet I cannot say that my clinical experience is at one with this teaching. An appeal is made to pathology to support the view that the liver is the source of urea. In acute atrophy of the liver, we are told (Foster's *Physiology*, p. 439) that urea disappears entirely from the urine, and its place is taken up by leucin and tyrosin. These, the products of the pancreatic digestion of nitrogenised food, are taken up by the portal vein, carried to the liver, where they are unacted upon by the diseased hepatic cells (which, had they been healthy, would have, in all probability, formed urea), and afterwards, entering the general circulation, pass out of the system through the urine in the same form as they entered the blood. Without denying the presence of leucin and tyrosin in the urine of patients suffering from acute atrophy of the liver, I maintain that too much has been made out for the liver in its relationship to the formation of urea. In Sutugin's case of acute atrophy of the liver (*London Medical Record*, April, 1885), the urine is stated to have contained bile, albumen, leucin, and tyrosin, also a normal amount of urea.

An isolated case goes for little; but what is our experience in other forms of liver-disease? I find that, while in Bright's disease the urine has contained a percentage of urea varying from .87 to 1.5, the urine of patients suffering from cirrhosis of the liver has shown a percentage of 3.5, and that, too, in a case which terminated fatally, and where, after death, a very large portion of true hepatic tissue had been replaced by the dense white fibrous bands frequently met with in that disease. Here, then, in a case—not one only—where the supposed urea-forming organ is diseased, more urea is passed off by the urine than in cases of Bright's disease, where the liver has not been much, if at all, at fault, structurally at least. While working at this subject, I have been interested in the facts revealed by a chemical analysis of effusions—pleural and abdominal—in various diseases. I have found urea present in the effusion of pleurisy, in the ascitic fluid of cirrhosis of the liver, and in abdominal effusions in Bright's disease. Add to these the urea-eliminating function of the skin, and we at once see that it requires no highly specialised cell to remove urea from the blood; that, while the cells of the convoluted tubules of the kidney undoubtedly excrete urea, the same substance, owing to its extreme solubility, must pass with the urine through the glomeruli. We would naturally expect, if the kidney is simply an excretor, and

the liver the chief organ forming urea, that, in cases of Bright's disease, where the kidneys are at fault, and the liver fairly healthy, and especially, too, as the urine in these cases generally shows a deficiency of urea, that, owing to its retention in the blood and system generally, any serous effusion met with in this disease would exhibit a high percentage of urea. Now, my experience is just the opposite of this. The serous effusion of pleurisy in otherwise a very healthy man contained .236 per cent. of urea; the ascitic fluid of advanced cirrhosis of the liver, .168 per cent.; the same percentage, too, was met with in another case of hepatic cirrhosis; while the abdominal fluid of a case of large white kidney contained only .101 per cent. of urea. Making every allowance for the difference in the amount of the various effusions, and for the difference in the diet of the patients, I fail to see, on the theory of the liver-origin of urea, why, in those cases where the liver is diseased, not only does the urine show a higher percentage of urea, but abdominal effusions in that and other diseases show a higher percentage of urea than the same in Bright's disease, wherein we are told urea is retained in the blood. These results have made me doubt the high urea-forming power ascribed to the liver, and have somewhat shaken my belief in the relationship of the highly organised cells in the kidney to the urea which is said to pre-exist in the blood.

All this is by the way, so far as lead-poisoning is immediately concerned. What I have found in plumbism is that, as a rule—and this applies particularly to women—whether the urine is albuminous or not, it is passed for a long time in large quantity, is of lower specific gravity than in health, and contains a deficiency of urea; that, sooner or later, the urine becomes albuminous, and then cerebral symptoms, which already may have been present, become increased in severity; that the kidneys in young people, particularly women, become frequently early affected in the disease, and often lead quickly to a fatal termination, while motor trouble may be absent from first to last; that acute cerebral symptoms, such as cephalalgia, temporary amblyopia, and optic neuritis, are frequently the first symptoms of the disease; that in these cases the urine is generally found of low specific gravity, is passed in larger amount than in health, shows a deficiency of urea; and, as the urine in such does not always contain albumen, the cerebral symptoms must be regarded either as the toxic result of lead acting directly upon nerve-tissue, or indirectly, in which it is aided by the inadequate manner in which the kidneys are removing waste material from the body. By these means, and not upon albuminuria, as some maintain, do I seek to explain the occurrence of cerebral symptoms early in the course of the disease. The effect is analogous to that produced by many poisons. The action of lead upon the system may be local, as in cases of "dropped wrist" pure and simple, where the lesion is either at first a myositis, followed by an ascending neuritis, or is primarily a peripheral neuritis; or, more central still, is in the anterior horns of grey matter of the spinal cord, causing anterior poliomyelitis. Lead acts more injuriously upon nerve-tissue than upon any other, disturbing in the most serious manner its function without producing any visible change in its structure. *Post mortem*, though death has apparently been caused by the condition of the brain, nothing may be found in that organ to explain it.

In the matter of treatment, means of prevention must occupy the first place, and of these, cleanliness is the most important. Baths; frequent ablution; avoiding eating with unwashed hands, in the factory particularly; wearing of respirators in the dusty atmosphere of the workshop; the habitual use of lemonade, made with sulphuric acid; the occasional use of sulphate of magnesia, acidulated with sulphuric acid, have been recommended, and are very beneficial. The difficulty, however, is to get the workpeople, unless compelled, to make use of them. In some of the large lead-factories on Tyneside, baths and all other sanitary requisites have been provided for the workers; but only I am told for the employers to find that their workpeople will scarcely, if at all, make use of them. When lead-colic has been established, and is severe, the employment of opium is called for, and the use of fomentations.¹ Should the bowels not be relieved in a day or two,

¹ Within the last few months, lead-manufacturers in this district have insisted upon their special sanitary regulations being complied with—and that not without cause. I have before me a copy of the special rules and regulations bearing upon this point in use at the Tyne Lead Works, Hebburn, a careful following out of which will reduce, I am sure, to a minimum the risk of poisoning by lead. The regulations of this Company formed the basis of the present Act of Parliament in reference to this subject, and are complete in almost every detail. Messrs. Foster, Blackett, and Wilson tell me that "all the men and women employed in our white lead department are carefully inspected every week by our medical officer, and any he may find of weakly constitution, or suffering in any way from contact with lead, are suspended from working for three months, when they are allowed to resume work, and are again inspected by our doctor. Notice of this fact is at once sent to the other lead-manufacturers in the district. We find this system

sulphate of magnesia becomes necessary. When there is paralysis or headache, iodide of potassium, with magnesia-sulphate, is of all others the remedy. The slowly-interrupted current has, in my hands, combined with iodide of potassium, proved very efficacious in the treatment of lead-paralysis. Nearly all have recovered, some entirely, others slowly and somewhat imperfectly; of these latter, most have had albuminuria.

In the words of the BRITISH MEDICAL JOURNAL (March 7th, 1885), "it is a disgrace to our civilisation that any industry should be attended by such terrible sacrifice of health and life." If experience should prove that the method of making white lead by Professor Gardner's patent is attended with less risk to health—electrolysis here taking the place of slower chemical action—the process conducted in a closed chamber, and the lead never coming into contact with the hand, then it is the duty of Parliament to interfere and prevent by law what we cannot but regard as an industry whose baneful effects are far reaching, and employment in which, particularly in the case of women, who seem to be very susceptible to its pernicious influence, tends only to pauperise those who are trying to gain an independent livelihood.

BRITISH MEDICAL ASSOCIATION.

FIFTY-THIRD ANNUAL MEETING.

PROCEEDINGS OF SECTIONS.

A SERIES OF CASES ILLUSTRATING THE USE OF THE EUCALYPTUS-AIR AND DRY DRESSINGS.

Read in the Section of Surgery at the Annual Meeting of the British Medical Association in Cardiff.

By A. W. MAYO ROBSON, F.R.C.S.,

Honorary Surgeon to the Leeds General Infirmary; and Lecturer on Operative Surgery at the Yorkshire College.

ALTHOUGH it is becoming the fashion, in certain quarters, to considerably modify the strict rules of antiseptic surgery advocated by Professor Lister, I cannot help thinking that the use of some atmospheric purifier gives us greater certainty of success, especially in hospital practice. Believing this, I still use either carbolic spray or eucalyptus-air in operating.

The carbolic spray often chills the patient, and wets and obscures the vision of the operator, so that if one can find a substitute free from these defects, it must be advantageous to employ it.

The eucalyptus-air apparatus, which I had the honour of showing at the meeting of the British Medical Association in Worcester, seems to me to supply the place of the carbolic spray, without its disadvantages; and, as shown by the brief reports of the following cases, from among many others in which it has been employed, the wounds, as a rule, run a perfectly aseptic course. The dressings employed are a single layer of gauze, wet with a solution of carbolic acid, 1 in 40, or of perchloride of mercury 1 in 2,000, applied next the wound, and over this a thick cover of salicylic silk or wool, the whole being retained in position by means of a gauze bandage. If no drainage-tubes have been employed, there is a first and final dressing; but if tubes have been inserted, they are removed on the third day, and another dressing is applied. This system of dry dressing is employed by my colleagues at the Infirmary in Leeds, and we have every reason to be satisfied with it, as being cheap, convenient, and efficient.

1. Double Macewen's operation for genu valgum, in a boy, aged 15. Operation, February 5th, 1885; temperature never above normal. Dressed twice; plaster-of-Paris applied on the tenth day, when he was made an out-patient.

2. Double Macewen's operation for genu valgum, March 12th, 1885, in a girl, aged 8, only once dressed. Temperature never above 99° F.

3. Numerous osteotomies, running a similar course.

4. Amputation of breast and axillary glands for scirrhus, in a patient aged 58. Dressed on the third and seventh days. Union by first intention: Made an out-patient on the ninth day. Temperature never above 99.2° F.

works to the advantage of both the workpeople and ourselves, and we rarely have any case of serious illness. In fact, if the rules now in force are carefully attended to, there is no more danger to health than in any other employment. We have no difficulty in getting our workpeople to comply with the rules; if they in any way objected, we would not employ them."

5. Excision of cystic tumour of breast, in lady, aged 45. Union by first intention. Only once dressed, to remove drainage-tubes. Temperature never above normal.

6. Numerous amputations of mammary gland, in hospital and private practice, with very similar courses.

7. Excision of knee, in girl aged 12; dressed at the end of the week, to remove drainage-tubes, and only thrice dressed afterwards; union by first intention. Temperature normal throughout.

8. Excision of sac of spina bifida, with aseptic course, and cure. Two cases.

9. Amputation for gangrene of leg. Temperature never above 99° F. Dressed on the fourth, seventh, and eleventh days, and made an out-patient on the seventeenth day.

10. Case of excision of varicocele. Temperature never above normal. Dressed on the sixth and tenth days. Several cases similar to the above.

11. Abdominal sections for pyo-salpinx; aseptic course and rapid recovery.

12. Numerous abscesses in various parts, incised and drained. Aseptic throughout.

13. Incision of large scrotal hæmatocele. Temperature never above 99° F. Five dressings; aseptic course.

14. Empyema. Several cases, with satisfactory course.

15. Operation for strangulated femoral hernia, with radical cure. Temperature never above 99.6° F.

16. Ophthalmic cases, as reported by my friend and colleague, Mr. Hewetson, who informs me that since he has used the eucalyptus-air in ophthalmic operations, he has never lost a single case of cataract.

17. Excision of several bones of the tarsus, in a girl aged 13; dressed weekly for four months; perfectly sweet throughout, and temperature never above normal.

The above brief record will, I trust, serve to show that the eucalyptus-air machine is capable of enveloping the region of a wound in a pure atmosphere, free from organic particles which may set up putrefaction, and that the dry dressings are thoroughly efficient in keeping the wound aseptic.

It is important to bear in mind that the chamber containing the cotton requires recharging occasionally, and that a little fresh eucalyptol should be added to both tubes, after the apparatus has been employed a few times.

I need scarcely add, that every antiseptic precaution is adopted in the way of attention to hands, sponges, instruments, etc.

THE IMMEDIATE IMPROVEMENT OF HEARING FOLLOWING DIVISION OF CICATRICES IN THE MEMBRANA TYMPANI.

Read in the Section of Ophthalmology and Otology at the Annual Meeting of the British Medical Association in Cardiff.

By H. BENDELACK HEWETSON, M.R.C.S.,

Ophthalmic and Aural Surgeon to the General Infirmary, Leeds.

It must have been observed very frequently by those who are constantly treating perforations of the membrana tympani, associated with chronic otorrhœa, that there is a great tendency to a diminution in the acuteness of hearing in cases in which the membrana tympani has become healed, and the perforation closed.

When attending the meeting of the International Aural Congress at Bâle last autumn, I was much struck by some observations which were made on the subject by Professor Politzer. It occurred to him that the cicatrix, or semilunar cicatricial band, which frequently exists in these cases, extending from the middle of the membrana tympani, skirting the edge of a healed perforation, so dragged on the malleus as to limit its power of recording sonorous vibrations. Professor Politzer himself quoted several cases in which the hearing-power was actually doubled immediately this cicatricial band was divided. I accordingly took an early opportunity of testing the operation.

The first case in which I performed the operation was in a hospital-patient of mine, attending my aural clinic at the Leeds General Infirmary. He came under treatment in March, 1884, and was then suffering from chronic otorrhœa following suppurative otitis, with a perforation in the left membrane, about the size of a No. 4 shot, straight below the handle of the malleus. So soon as the discharge was healed (under treatment by injection of a one in sixty solution of carbolic acid, after which iodoform was blown on to the ear, which took place in two months and a half), it was evident that the boy did not hear so well. Originally, the discharging ear was the better of the