the body, and secured it temporarily with a wire splint; having sewn up the incision—leaving one drainage tube in it, as there had been quite extensive laceration of the soft parts in exposing the joint—I then turned her on the other side and did a subcutaneous osteotomy, also below the smaller trochanter, without attempting to form a false joint, as I had proposed to do on the other side. This wound was closed and sealed with iodoform collodion. The patient was then encased in plaster-of-paris bandages from the armpits to the toes of both feet, the plaster being reinforced by iron wire splints laid on the front of each thigh and running up over the thorax, and secured by additional layers of plaster bandages.

I cannot too highly recommend this way of putting up osteotomies of the femur and also fractures of the femur high up, the ease with which patients can be shifted from one position to another and their necessary wants attended to being altogether greater than by means of any other dressings which I have tried. When the patient is an adult, weighing perhaps 180 pounds, it is very difficult for him to be moved except by means of a block and tackle attached to a stout beam over the bed, and unless a very rigid splint is applied the body is almost certain to bend at the hips when the patient's weight hangs suspended by means of straps passing under the shoulders and lower extremities. I therefore reinforce the plaster with iron strips at the hips. It is of the utmost importance in these cases that the dressing should be sufficiently secure not to require readjustment until the fractured bone shall have had a chance to become consolidated.

The drainage tube was removed at the end of two days, the outer layer of gauze only being removed, as the end of the tube had purposely been left projecting through the inner layer of gauze. A fenestra was cut in the plaster to facilitate removal of the tube.

At the end of two weeks I cut the dressing in two on the right femur at the point where the hip and body join, and adjusted the position of the limb slightly, and rejoined the portions of this splint by new bandages and wire. The patient made an uneventful recovery, and her position before and after the operation is shown in the accompanying photographs.

From my experience in various cases of ankylosis about the hip, I have found that many of the distortions following hip disease, which are supposed to be due to contraction of the tendons around the joint, are in reality retained in place to a greater extent by the adhesions about the capsule and between the bones, and that in many cases the cutting of the tendons alone will be insufficient to allow reduction of the deformity. I believe it is a simpler and safer operation in many cases to do a subcutaneous osteotomy below the trochanter than to endeavour to cut, either subcutaneously or through an open incision, the adhesions which bind the joint in its distorted position, and I have learned that it is very easy to be mistaken in regard to the presence of movement in a joint, and we very frequently imagine that we feel a slight amount of motion in ankylosed hips, whereas the motion is really between the flesh and the bone or in the sacro-iliac synchondrosis, and even in joints which are more easily accessible (as the knee and elbow) I have seen men of large experience deceived in supposing that movement was present, when subsequent operation showed firm bony ankylosis.

In regard to the choice between osteotomy or *brisement forcé*, a good deal must be left to the judgment of the operator and the conditions of each individual case, and there are certain cases where I believe the dangers of re-exciting inflammation in the joint by *brisement forcé* are so great that I think it is safer to do an osteotomy, and if the joint is to be broken up, I believe in making section of all constricting bands which can be safely cut, breaking up the adhesions thoroughly under an anæsthetic, placing the limb at once in the required position, and then keeping the part absolutely at rest until all pain, swelling, and tenderness have subsided, and I find benefit in many instances from diminishing the blood supply to the joint by means of moderate compression over the artery supplying it with blood. After the joint is completely free from pain slight motions should be given every day to restore to function, but never carried to

the point of producing pain or tenderness which will last as long as twenty-four hours. If your efforts to restore functions are followed by increased heat or tenderness, cease your manipulations and be content with the improved position and a stiff joint. If, however, as in Case 1, there is slight motion in the joint and the employment of gradual manipulation and massage is not followed by inflammatory reaction, continue the procedures daily, increasing in force and frequency as experience warrants, and in many instances you will be able to cause absorption of inflammatory deposits, break down old adhesions, and restore excellent motion to joints which have been considered useless for months and sometimes for years.

REMARKS MADE AT THE BEDSIDE OF A PATIENT SUFFERING FROM LEAD ENCEPHALOPATHY.

By STEPHEN MACKENZIE, M.D., F.R.C.P.,

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Physician to the Royal London Ophthalmic Hospital.

E. C., aged 26, was admitted into the London Hospital under my care on October 27th, 1893, and the following history and notice of the case were taken by Messrs. Palmer and A. L. Smith.

The family history has no bearing on the case. The patient was born at Poplar, and lived there until 14 years of age, when she went into service as a helper at a coffee shop. She married at 17, and has had seven children and several miscarriages. Only one of the children is living now, aged 9 years; the remainder only lived a few days. She has been of temperate habits. She has lived badly, having meat only once a week as a rule. She has lately been employed in lead works, but has only followed this avocation for three weeks. She was engaged in the manufacture of white lead. Two months before her illness she had an attack of pain in the chest, and vomited about a tablespoonful of thick dark blood. This happened only once, and she has never been laid up, except with her confinements and miscarriages.

She states that she was quite well on Sunday, October 16th. The next day she went to work, but, to use her own words, "did not feel very grand." On the Tuesday and Wednesday she had the same feeling of weakness and pains in her limbs; she felt it was too much trouble even to comb her hair. On the Thursday, October 20th, more urgent symptoms arose. At 8 in the morning some breakfast was got ready for her, but she felt so sick she was quite unable to eat it. What she vomited was white froth—no blood. She took no solid food, and any liquid she took was immediately vomited. On the Friday she brought up some blood, about a tablespoonful; it was pale and tasted sweet. This was the only occasion on which she vomited blood, but the vomit consisted of froth, and was incessant, at least fifty times a day. The day before admission it became greenish.

She lies in bed on one side, markedly anæmic but not cachectic. She complains of constant and severe headache, which she localises chiefly in the occipital region. It is evidently, from her appearance of suffering, very severe. She vomits every few minutes, the vomited matter consisting chiefly of yellowish-green froth and fluid. The bowels are confined, the tongue is furred, and along the edge of the gums of some of the teeth only there is a faint but distinct blue-black line. The abdomen is not distended but is slightly tender, as is the liver, which is of normal size. The heart's position and dulness are normal; the first sound at the apex is murmurish; the precordial region is tender, the pulse is 75, the tension rather below the normal. She is intelligent but very much oppressed with the headache, which has never left her since it commenced. It is chiefly occipital, but extends from thence over the vertex to the frontal region. She no sooner takes anything into the stomach than it returns, and is frequently sick without taking anything. The vomit amounts to 3 or 4 pints per diem; it is acid, bile-stained, and frothy. The pupils are large, and react to light and accommodation. There is internal strabismus of the right eye, the result of an accident when 15 years old. Ophthalmoscopic examination showed swelling of each optic papilla, indis-

distinctness of its margins, distension and tortuosity of the vessels, which in places are partly concealed by the swelling. The special senses are not affected; there is no paralysis of motion or sensation in the limbs; the knee-jerks are slightly exaggerated, and the plantar reflexes readily elicited. She is too ill to test her powers of walking, but she states she noticed her gait had been staggering, and she felt as if she was losing her head, and had no strength in her legs for three weeks. The urine is of pale amber colour, acid in reaction, specific gravity 1020, and is free from sugar and albumen. The temperature is normal.

These are the facts of the case, and the first thing we have to do is to endeavour to arrive at the diagnosis.

The patient was admitted for ulcer of the stomach. The vomiting and a history of bringing up blood probably suggested this, but the more detailed history of the case Mr. Palmer has elicited for us, and the condition of the patient since she has been in the hospital negative this diagnosis. Moreover, the patient has double optic neuritis. Now this is not accounted for by ulcer of the stomach, even if she has this, and there must be something else to have given rise to it. It will be convenient to make this symptom—double optic neuritis or papillitis—the starting point in our diagnosis.

What may this double optic neuritis depend on? In the first place, it may be due to cerebral tumour or some other coarse lesion of the brain. The intense cephalalgia and the frequent and purposeless vomiting would be quite in accordance with this view. It is true there is no evidence of local disease of the brain, such as paralysis or convulsion, but it not infrequently happens that we do diagnose, and that correctly, tumour of the brain from this symptom, optic neuritis, especially when accompanied by pain in the head and vomiting, even when there is not a single localising symptom.

But—and this is an exceedingly important qualification—before we are justified in concluding that optic neuritis, with or without headache and vomiting, unaccompanied by evidence of local brain, is due to a cerebral tumour or some gross encephalic lesion, we must exclude the other causes of optic neuritis—at least the fairly common ones. What are these?—

1. *Anæmia*.—Anæmia not infrequently in my experience gives rise to double papillitis and sometimes to papillo-retinitis. This is most common in chlorosis, but may occur in pernicious anæmia. I have published in the *BRITISH MEDICAL JOURNAL*, vol. i, 1885, p. 328, a very pronounced case of double papillo-retinitis from chlorosis, with remarks on the subject. Now when I examined this patient I pointed out the anæmia. I held up the hand and looked at the nail bed of the fingers, and from the absence of all pink colour ventured to predict that she had less than 50 per cent. of red corpuscles (subsequent examination showed 38 per cent. and great diminution of hæmoglobin). So far, then, the optic neuritis might be due to the anæmia which is present. There is, however, another symptom, to be presently adverted to, which in my mind renders it improbable that anæmia is the cause of the neuritis. Other blood conditions, such as leucocythæmia, may give rise to neuritis, but in leucocythæmia there is more usually a great distension of the retinal veins and hæmorrhages, without marked swelling of the optic nerve.

2. *Bright's Disease*.—You are probably familiar with the ocular changes in this disease. In the great majority of cases it takes the form of a retinitis or papillo-retinitis, numerous hæmorrhages and white spots being seen in the retina, especially in the region of the macula, with or without some swelling of the disc. But in rare cases, of which I have seen a few, optic neuritis, indistinguishable from that met with in connection with cerebral tumour, is encountered. In the present case Bright's disease is excluded by the absence of albuminuria or casts, absence of œdema, and by the absence of increased arterial tension and cardiac hypertrophy. We may therefore dismiss it.

3. *Diabetes*.—I have never met with simple papillitis in connection with diabetes, but have seen several cases of diabetic retinitis closely resembling the retinitis of Bright's disease. In this case diabetes is excluded by the absence of glycosuria and polyuria.

4. *Lead Toxæmia*.—In the conditions in which lead gains admission to the system and is deposited in the tissues a great variety of symptoms may be caused. A case of lead poisoning may present itself as colic, or drop-wrist, or with acute and severe cerebral symptoms. In these conditions double optic neuritis or papillo-retinitis may be caused. I have seen a fair number of cases and published several. Now, in our patient we have the statement that she has recently been employed at lead works; further, she has a "lead line." It is true this is a very slight one, but to one experienced in such cases the testimony is unequivocal. It is a thin blue-black line just at the junction of the gums and teeth. Where a tooth is absent no such line occurs, and in this case it is not present at the edge of the gums over all the teeth. It has to be distinguished from a reddish-blue line near the edge of the gums, which is often present and misleads the inexperienced. Its sharp, well-defined character, blue-black colour, and position at the free extremity of the gums is pathognomonic. It is, therefore, a very tell-tale symptom.

In the presence, then, of this symptom, and the exclusion of positive or probable evidence of cerebral tumour, Bright's disease, and diabetes, we are reduced to two causes of the optic neuritis—anæmia and lead poisoning. As we have an undoubted history of our patient working in lead, and the pathognomonic sign of the lead line on the gums, the presumption is greatly in favour of lead poisoning causing the neuritis. Lead poisoning will account for the anæmia; the anæmia could not cause the blue line on the gums. I therefore arrive at the conclusion that the double optic neuritis is due to lead poisoning.

Having excluded other possible causes of the optic neuritis, we have, to make our diagnosis complete, to ask ourselves the question, Will this diagnosis explain all the phenomena of the case?

We see a good deal of lead poisoning in all its forms at this hospital, owing to there being some large manufactories in the East End of London. These cases assume various types, but I must confine my remarks to cases of acute lead poisoning with cerebral symptoms. This class of case was well described by Tanquerel des Planches in a treatise on Lead Diseases, published in 1838, and translated by Dr. Samuel L. Dana, of Lowell, U.S.A., in 1848. Tanquerel described three varieties of this form of acute cerebral disease from lead, which he calls lead encephalopathy, the delirious, the comatose, and the convulsive forms, which may exist separately or combined. I have seen examples of each, and my experience confirms that of Tanquerel, that the combined form is the most common. Sometimes the disease manifests itself by convulsions, which pass into coma, in which condition the patient dies; or it may begin with delirium accompanied by convulsions. A good case of this kind, with optic neuritis and hæmorrhage, was under my care in this hospital in 1878, and is published in Gowers's *Medical Ophthalmoscopy*, with an illustration. The patient recovered completely. I had a case in George Ward about two years ago. I found the patient, a middle-aged man, comatose, and he had been convulsed. His illness had been a short one, and investigation showed a lead line on his gums. It was elicited that he had been working in lead. He died, and no naked-eye disease was found in the brain or kidneys. I have known insanity to be caused by acute lead poisoning. In the majority of cases it will be found the patient has been losing colour for some time. Even in acute cases, but not in all, there is usually anæmia. Anæmia is usually the first symptom of lead poisoning, then follows vomiting and often headache, sometimes colic.

The diagnosis in lead toxæmia is often much assisted by the use of the ophthalmoscope. It will be observed that there is no mention of defect of sight in our patient; and this illustrates the importance of the routine examination of the eyes, as is done in all cases under my care. The occurrence of changes in the fundus often puts us on the right track, but the most conclusive evidence is furnished by the condition of the gums.

In what circumstances does lead encephalopathy occur? In my own experience I have seen it in those who have worked in lead for years, and in those who have only quite recently been so employed. In our patient, the time she has

been working in lead is unusually short, only three weeks. Women, as shown by Dr. Thomas Oliver,¹ are more susceptible to the influence of lead. Mr. F. M. Corner, in a paper read before the Hunterian Society in 1887,² reported five fatal cases of acute lead encephalopathy; all were women. In my own practice, however, in this hospital I have seen as many men as women. The condition of those who work in lead is a factor in the causation. It is especially in those who have been living badly, like our patient, that the severe symptoms are most apt to arise. Many, from the well-known effects of this employment, will not work at it until they are driven to it by destitution. The pay is good, and employment easily obtained. I have several times urged patients who have suffered from various forms of lead poisoning to shun the lead works as they would the plague. But the hard pressure of want has driven them back to the works, to suffer again and again.

A patient who has once suffered from lead poisoning is very likely to be again affected. It is possible this is due to the influence that the lead has already exerted on the tissues and organs, but probably it is due in a greater measure to individual susceptibility, as many work for years without being affected. There are certain processes in the manufacture of lead which are peculiarly liable to produce lead toxæmia. It is in the "unstacking the white lead" that the greatest danger arises, as this is always attended with the giving off of much dust, consisting of carbonate of lead. At the lead factories, as a rule, every precaution is taken to minimise the risk, and the factories are periodically inspected, but in spite of these precautions many cases occur, often from the carelessness and neglect of the workers. Alcoholic habits—and from various causes these are common in lead workers—predispose to its occurrence, and intensify its evils.

What is found in such cases after death? To the naked eye nothing in the majority of cases. The brain, as a rule, shows no disease; the lead line is found on the gums, and often the colon is pigmented from the same cause as the gums, namely, the production of sulphide of lead. There is no reason for supposing that lead is present in these parts more than in others; it is simply due to the fact that in these parts the lead is brought into contact with sulphuretted hydrogen. I often say that potentially a patient has a lead line all over the body. The late Dr. Meymott Tidy examined some brains chemically for me, and found appreciable quantities of lead. In a case reported by Dr. A. Atkinson,³ Dr. Tidy calculated that the whole brain contained as much as five grains. Dr. Oliver and some other workers have shown the proportion in which lead is found in the various tissues. The intestines and liver contain the most, and this is evidence of the entrance of the poison by the digestive tract. The brain and other nervous structures always show a high percentage, and this accounts for the frequency of symptoms due to affections of the nervous system. From the changes seen in the optic papillæ and retina I have been led to the conclusion that probably the lead induces a condition of cerebritis, but I have made no microscopical examination of the brain. Dr. Charlewood Turner, however, who has examined two cases of fatal plumbic intoxication with cerebral symptoms, found "exudation in perivascular spaces round some of the vessels, which led him to regard the symptoms as due to an inflammatory œdema of the convolutions,"⁴ and Dr. Drummond has found similar changes.

It is quite clear, then, that the diagnosis of acute lead poisoning is supported by the symptoms present in this case, and that the diagnosis is adequate to explain them all.

Such a condition is exceedingly dangerous, the majority of cases of acute lead encephalopathy proving fatal. Our patient is evidently very seriously ill. What are we to do to avert her danger? As lead is the cause of all her symptoms—the cerebral condition, the optic neuritis—its elimination must be our principal aim in treatment. Now we have a remedy which exercises a powerful influence in discharging lead from the system by the kidneys, the bowels, and the skin. This remedy is iodide of potassium. I shall order the following

prescription: Potassii iodidi gr. x; magnesis sulphatii 3ij; aquam chloroformi ad 3j; to be given every four hours. Chlorides, as well as iodides, are believed by the French to eliminate lead. I always encourage patients suffering from lead poisoning to take freely of table salt.

(POSTSCRIPT, November 24th.—It is satisfactory to be able to report that the patient is making a good recovery. The headache became less frequent and severe, and has now disappeared. The vomiting, such an urgent and distressing symptom, yielded more slowly, but has now entirely ceased. Morphine was given twice subcutaneously. The bowels were very obstinate, and required repeated enemata in addition to the sulphate of magnesium, but are now acting freely each day. The patient is now convalescent, but still anæmic, and the optic neuritis is still present, but subsiding.)

A CASE OF SPINDLE-CELL SARCOMA OF THE IRIS.

Read in the Section of Ophthalmology at the Annual Meeting of the British Medical Association held in Newcastle-on-Tyne, August, 1893.

By G. E. WILLIAMSON, M.A., F.R.C.S.,

President of the Section; Surgeon to the Royal Infirmary, Newcastle-upon-Tyne.

A HEALTHY-LOOKING girl, 14 years of age, was admitted under my care, into the Royal Infirmary, Newcastle-upon-Tyne, with a condition of her left eye that will be best appreciated from the accompanying drawing (No. 1). The growth was first noticed by the patient about six months before admission, as a small yellow speck. It began apparently at the peripheral part of the iris, and as it grew, it encroached on

Fig. 1.—Sarcoma of iris.

the pupillary margin. There had been no inflammation about the eye, and vision was unaffected.

The growth was of a bright yellow colour, and stood out strikingly against the background of the iris. Little vascular tufts could be seen on the surface by using a strong light and a high magnifying power. No sign of the growth could be detected behind the lens on examining obliquely by the direct method.

From the history and appearance, I made out that I was dealing with a sarcoma of the iris, and as nothing could be seen of it in the deeper parts of the eye, I tried to remove it through an incision in the cornea. It was, however, so firm that I could not move it on account of its deep connections. Four days later I excised the eye. The patient did well, and there has been no recurrence after the lapse of two years.

After excision I cut the eye into anterior and posterior halves, and put them for a quarter of an hour into a half-saturated solution of perchloride of mercury in methylated spirit. They were stained in bulk in borax carmine, and ultimately embedded in paraffin by the interstitial method. I cut the sections with a Thomas's sledge microtome.

Drawing No. 1 shows the eye as it appeared before the operation. No. 2 represents a section through the tumour and the surrounding parts. In front are the cornea and sclerotic, behind are the club-shaped growth, with its narrow neck, and the ciliary body below it.

No. 3 is from a small portion of the growth seen under a

¹ *Lead Poisoning in its Acute and Chronic Forms*, 1891, p. 22.

² *Hunterian Society Report*, 1886-87, p. 58.

³ *Lancet*, 1878, vol. i, p. 784.

⁴ *Hunterian Society Report*, 1886-87, p. 61.