EMBOLISM OF THE RIGHT BRACHIAL ARTERY AS A COMPLICATION OF LOBAR PNEUMONIA.

BY

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LANELLY.

THE rarity of embolism of a main arterial trunk as a complication of lobar pneumonia, and the unusual bacteriological findings on examination of the blood, prompt me to make a record of this case.

W. J., aged 39, an insured person who worked as a labourer in a tinplate works, on October 16th, 1922, felt chilly and had a rigor after having been exposed to cold and getting wet through during a drinking bout. On October 17th I found him complaining of pain in the right side of the chest. The temperature was 104°, pulse 110, and the sputum was rusty. The right upper lobe was found to be the site of lobar pneumonia. All over the rest of the chest were signs of chronic bronchitis. Crisis occurred on the ninth day; at no time was there any real cause for anxiety, and apart from a soft systolic bruit at the apex there were no physical signs or symptoms of any cardiac affection. After the crisis convalescence was uneventful until the fourth day, when he began coughing badly, expectoration was profuse, the temperature was elevated, and sweating was marked. There were signs of bronchitis over both lungs; over the pneumonic area in the right lung the breath sounds were cavernous, and there were abundant crepitations. Night sweating was profuse and occasionally delirium was marked. Tubercle bacilli were not found in the sputum, but the clinical picture and the facts which came to light in the family and personal history strongly supported the view that the patient was now suffering from an acute tuberculous infection of the right lung. I was informed that the patient had, after his discharge from the army, been under treatment at a military hospital for tuberculous disease of the spine. His only brother had died recently of tuberculous infection of the lungs. Examination of the heart during this period showed no abnormality save for the soft systolic bruit at the apex previously noticed.

This was the clinical picture up to November 9th. On November 10th, during my visit in the morning, the patient told me that during the night he had been seized with very acute pain in the right arm; examination of that arm, conducted with difficulty owing to the great pain caused by the slightest movement, showed no apparent difference from the other; the heat in both was equal; there was no swelling in the right arm and no difference in colour between it and the sound arm. There was, however, exquisite pain in the affected arm and paresthesia of the muscles. The hyperaesthesia was confined to the area from the wrist to the shoulder. There was almost complete anaesthesia in the tips of the thumb and fingers. The right radial pulse was absent both to the finger and to the stethoscope. Following the course of the radial upward no pulsation could be felt in the ante-cubital fossa. At the level of the posterior wall of the axilla, in the line of the vessel, a very tender lump about the size of a small hazel nut could be felt. The surrounding structures appeared normal. Above this level the vessel could be felt pulsating strongly. The radial pulse in the left side was present and bounding in type. An examination of the heart revealed nothing further than the soft systolic bruit already noted. I felt I was dealing with an embolism of the right brachial artery, and thought that the probable source was an infective endocarditis. The cardiac physical signs at this stage were, however, not in any way pronounced.

The right arm was wrapped in cotton-wool and immobilized. For the next three days treatment was expectant, and a careful examination of the arm and heart was made daily. The appearance of the arm never changed. Compared with the sound arm there was no difference in size, heat, or colour. The hyperaesthesia, however, remained marked, as did the anaesthesia of the tips of the thumb and fingers. The patient was profoundly intoxicated, delirium became marked, and night sweats became very heavy. The cough was less, but the physical signs in the lung were still those of acute phthisis. On November 11th and 12th there was no alteration in the physical signs in the heart. On the latter day he was seen with me by Dr. Samuel Williams, who agreed that the diagnosis was probably septic endocarditis following lobar pneumonia, and that the embolus was a result of the endocarditis. On that day definite signs of endocarditis were present in the shape of marked systolic and presystolic bruits at the apex. The murmurs were harsh and ran into each other. At the base of the heart a soft high-pitched systolic bruit could be heard distinct in pitch from the murmur present at the apex.

On November 13th a blood culture was made and sent by hand to Dr. Sladden, pathologist, Beck Laboratory, Swansea General Hospital. At this time the physical signs were as on the previous day. The general condition of the patient was very poor indeed, and he might be described as being in a typhoid state with the low type of delirium. I was called hurriedly to see him about 10.30 that night. On my arrival a few minutes later I found he was dead. A few minutes prior to my arrival he had been talking quite sensibly to his friends. They said he seemed to fall off to sleep and then they found he was dead. I was unfortunately unable to get permission to perform a post-mortem examination.

I have recorded the case because embolism of a main arterial trunk as a post-pneumonic complication is unusual. Osler¹ records a case of embolism of the femoral artery as a

pneumonic complication and states that it is very rare. In his case the complication occurred during the height of the fever, and the limb had to be amputated. The patient recovered.

In the case here recorded the obstruction was evidently complete and sudden. At no time was there any sign of interference with the circulation such as might necessitate surgical interference. Apart from the paresis and the nerve symptoms the limb was apparently normal. The obstruction must have been below the circumflex artery, and the condition of the arm again demonstrates the wonderful collateral circulation which exists in the upper limb. Complete obstruction of a main trunk with so little interference must, I think, be a very rare condition. Other points of interest in the case are:

1. The absence of definite signs of endocarditis in the early stage of the embolus.

2. The difficulties of diagnosis owing to the clinical picture in the lungs, so suggestive of acute phthisis.

3. The result of the bacteriological findings of the blood. Dr. Sladden's report was as follows:

"A plentiful growth of streptococcal chains occurred. Fuller tests showed this to be a non-haemolytic strain with the sugar reactions of *Streptococcus salivarius*. It was not bile-soluble, and grew readily on ordinary broth medium. It was therefore not in any way related to pneumococcus as usually identified."

I had expected the blood culture to reveal the presence of pneumococcus. Were the endocarditis and embolus the result of pneumonia or were they independent of the primary condition? Unfortunately the sputum was not examined for the streptococcus which was found in the blood.

REFERENCE.

¹ Osler: *Principles and Practice of Medicine*, p. 99.

THE BLOOD AS GUIDE TO EARLY DIAGNOSIS IN LEAD POISONING.

BY

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EALING.

LEAD poisoning is commoner than is generally supposed, and it is useful for practical purposes to recognize four classes of cases. The industrial includes workers engaged in smelting or making white lead, etc. This class is invariably detected by experienced medical men at the works. In general practice cases are mainly occupational—painter, plumber, etc. Recently there have been more cases than usual in painters. The practice of moistening the sandpaper with oil is practically unknown in London, and it is fine dust arising from dry sandpapering indoors which does the damage. I have known a healthy man to contract acute plumbism in this way in three weeks. Painters often drink much beer, and in such cases constipation is not to be expected with the colic.

Criminal cases used to be frequent in the Midlands, but I have seen only one in London. Diachylon is taken to produce abortion early in pregnancy by women—generally married women. Symptoms are often vague or misleading: constipation, vomiting, even albuminuria and headache, may be attributed to the condition, and abdominal pain that would otherwise secure attention is minimized by the woman as she hopes it is the prelude to miscarriage. The gums may give no help as the patient may have artificial teeth. Further, slight pyrexia is not unusual and agonizing colic may be attended with urgent vomiting—not unlike that due to an acute abdominal lesion. Only early diagnosis can save these women from serious illness, and in no class is blood examination so helpful.

All other cases may be lumped together as domestic and arising from food, drink, etc. Recently there has been an outbreak in this district due to accidental contamination of beer. But of 9 cases seen since midsummer only 3 were due to beer alone, though in 3 others it was probably an important factor. In the remaining 3 beer could be excluded—in one the source remains unknown, but in two painters it was occupational. These two were severe and conspicuous examples of acute lead colic.

In 30 cases of all kinds I have seen wrist-drop only once, and the patient (potman) developed encephalopathy about a year later and died. Another case (criminal) developed the encephalopathic type, but recovered after a long illness. In 28 there was practically no obvious symptom after a

fortnight's treatment. The longer diagnosis is deferred the greater are the variety and the duration of the symptoms. To hesitate in the absence of wrist-drop is like waiting for locomotor ataxia before diagnosing syphilis. In 19 out of 20 cases the patient first seeks advice for abdominal pain, which may be severe colic or only vague discomfort, and it is only in occupational cases that lead is likely to be suspected. In the latter a blue line is seldom absent, but this is not conclusive evidence.

1. *Blue Line*.—In industrial cases this is practically always present, but it may also be found in healthy workers. It means deposit of lead on the gums and is acquired from dust. In occupational cases the line is of more importance and means faulty habits, which are sure to end in plumbism if they have not already done so. In domestic cases where lead has been ingested in minute quantities in beverages, even when symptoms of poisoning are present, this sign may be absent. It is generally rather faint, and I have known such a case still undetected a fortnight after admission as alcoholic neuritis to the ward of a teaching hospital. Blue line demands careful examination of the patient or worker, but diagnosis must always be independent of blue line, which remains only valuable presumptive evidence. All are agreed as to the importance of early diagnosis in syphilis, and this is not less essential in plumbism. In order to effect this, diagnosis must depend mainly on a clear conception of the importance of two factors—lead in the urine and punctate basophilia in the blood film.

2. *Lead in the Urine*.—We are assured that lead may be found in the urine of healthy industrials. In them minute quantities are always present in the system, but satisfactory elimination by kidneys and bowel is said to prevent accumulation and poisoning. This can hardly be regarded as satisfactory, and in my opinion such cases require further investigation. In lead poisoning the metal is always present in the urine and remains for weeks or months after treatment is undertaken. In the case of suspicious symptoms examination of the urine affords valuable diagnostic evidence, or, alternatively, altogether excludes lead. It should be oftener employed.

3. *Punctate Basophilia*.—This sign is neglected because its significance is misunderstood. It is necessary to recall a few facts of haematology. Polychromasia is a sign of unusually active erythrocytogenesis, and is conspicuous in the blood of the newborn babe, where it is associated with nucleated reds. In adults polychrome erythrocytes are still readily attracted into the blood stream, and appear in minor anaemias of convalescents and even after moderate haemorrhage, whereas erythroblasts only appear in more serious anaemias or after severe haemorrhage. In the so-called pernicious anaemias a toxin is also exciting the marrow to pathological regeneration. The first indication of this is the appearance of punctated erythrocytes, which later on are associated with megaloblasts, giving a blood picture of anisocytosis, polychromasia, and normoblasts (normal regeneration), combined with poikilocytosis, punctate basophilia, Cabot rings, and megaloblasts (pathological regeneration). All these may appear fairly early in mild lead intoxication, but for some weeks there is usually only anisocytosis, polychromasia, and punctate basophilia. Thus lead acts directly on the marrow in a similar way to the toxin in a pernicious anaemia. In malaria at every paroxysm the marrow is excited in the same way, but the reaction is transient. With these exceptions, pathological regeneration is seldom seen, and it is safe to say that, in the absence of serious anaemia, punctate basophilia is more likely to be caused by lead than by anything else. Now, anaemia is never serious in the early weeks of lead poisoning. Basophilia is pathological, and demands investigation just as much as albuminuria or glycosuria. So long as it is present something is acting injuriously on the marrow, and if this is lead the urine will contain a trace of the metal even in the absence of corroborative symptoms.

As I have indicated, basophilia is only one in a series of biological reactions inevitably excited by small doses of lead. When diagnosis is early—whatever the treatment—the reaction usually disappears within three months, and it is interesting to watch the subsidence of the reaction in the blood film. Not much difference is seen for a month, but soon afterwards blasts disappear in mild cases and search for basophils becomes necessary, though these can usually be found for two months. With the help of the Clinical Research Association I have compared this with disappearance of lead from the urine. In a mild case due to beer basophilia was still seen fifty-two days after ingestion ceased, and half a pint

of morning urine gave a trace of lead. In a more severe case of painters' colic erythroblasts were found thirty-nine days after leaving work, but a chance sample of eight ounces of urine gave no result. On the fiftieth day there were easily found anisocytosis, polychromasia, and punctate basophilia, and, after a search, Cabot rings and one punctated erythroblast. The whole twenty-four hours' urine was collected for the same day and gave a trace of lead. Apparently these two signs disappear about the same time.

I have already pointed out how basophilia may be used to exclude lead poisoning (BRITISH MEDICAL JOURNAL, 1917, ii, p. 650). It serves another purpose in enabling one to follow the progress of a case. On finding brisk pathological regeneration in a potman some months after leaving work with colic and limp hands, I convicted him of continued indulgence. He disappeared for some months, to return with double wrist-drop, albuminuria, and brisk pathological regeneration. His protestations suggested to me that possibly a second unknown source of poisoning existed, and I advised him to leave the district. This he hesitated to do, and a few weeks after was taken, comatose and convulsed, to Isleworth, where he soon died. In conclusion, I suggest that diagnosis should depend, not on gross signs, but on appreciation of suspicious symptoms and investigation of blood and urine. Whereas examination of the urine is a tedious process, the blood film is quick, easy, and almost as reliable. I can testify that early diagnosis will avert gross symptoms, so that in a month Government referees will reject cases still showing blue line on the gums, punctated erythroblasts in the blood film, and lead in the urine.

Memoranda : MEDICAL, SURGICAL, OBSTETRICAL.

ULCERATIVE STOMATITIS IN CHILDREN, AND ITS TREATMENT.

DURING the war a form of stomatitis was described the causal organisms of which were the Vincent spirochaete and the fusiform bacillus; to this the name of "trench mouth" was given. This form of stomatitis has long been described in books on children's diseases under the term "ulcerative stomatitis." The treatment, however, which was extremely successful for "trench mouth" has not been applied, so far as I am aware, to children up to the present. This disease, which usually occurs after the primary dentition is well advanced, is common among the hospital class of patient, and is characterized by swelling, redness, oedema, and extreme tenderness of the gums round about the teeth. The gums may show actual ulceration. Those parts of the cheek which come in contact with the ulcerated areas may themselves show infection. The teeth are frequently carious; pressure on the gums may or may not cause pus to appear at their margins. The tongue is coated and the breath fetid; the temperature is generally raised to 101°; the child refuses food, and there may be swelling and tenderness of the lymphatic glands beneath the jaw at its angle.

Smears of the pus will usually show the fusiform bacillus and the Vincent spirochaete. The clinical picture is thus very like that of the so-called "trench mouth." The treatment which I have carried out in a large number of out-patients is as follows: The following mixture was applied to the mucous membrane of the gums, cheeks, and tongue at six-hourly intervals: liquor arsenicalis 2 drachms, vin. ipecac. 2 drachms, glycerin 2 drachms, aq. menth. pip. to 1 oz. As this mixture is poisonous, and could not be used as a mouth wash in small children, I ordered it in the following fashion: 15 drops diluted in 2 drachms of water were to be applied by means of a soft piece of muslin on the end of the finger or a camel's-hair brush. In children old enough to use this mixture as a mouth wash—that is, old enough to spit out the mixture after its application—a soft boiled toothbrush was used, a few drops of the mixture undiluted being applied to the gums by the toothbrush. The arsenic seems to be specific for the spirochaete and the vin. ipecac. for the fusiform bacillus.

In conjunction with this treatment, which was that recommended during the war for "trench mouth," potassium chlorate was administered, 12 grains in the twenty-four hours being given to children under 1 year, and 18 grains in the day to children under 2 years. In the administration of the potassium chlorate the child's meals were carefully