or personal sources about eosinophile ascites, but a few words may be added about eosinophile pleural and cerebro-spinal effusions.

Eosinophile pleural effusions have been reported by Vidal and Revaut⁹ (4 cases), Barjon and Cade¹ (5 cases), Perkins and Dudgeon.⁷ According to Emerson,³ there is a slight eosinophilia (2 to 5 per cent.) in tuberculous pleurisy, and Mosny, Dumont, and Saint-Girons⁴ state that eosinophile pleural effusions are common in Paris. In a traumatic haemothorax the latter observers found 92 per cent. eosinophiles, while the haemic eosinophile count was 4 per cent.; and in 2 cases of pleural effusion due to bullet wounds H. S. Carter² reported eosinophilia of 70.2 and 87.8 per cent. respectively. Eosinophile pleural effusions have been regarded as usually transient, benign, and as indicating a process on the way to cure (Vidal and Revaut). Perkins and Dudgeon's case, however, proved fatal. Eosinophilia may occur in pleural effusions due to such widely different causes (post-pneumonic, post-typhoid, haemorrhage, granular kidney, rheumatism, tuberculosis) that its presence cannot be correlated with any special etiological factor. In many of the cases the effusions have been sterile on culture. With eosinophile pleural effusions there is usually, though not invariably, haemic eosinophilia, which, however, never approaches the count seen in the serous effusions. Mosny, Dumont, and Saint-Girons contend that in these cases the eosinophilia is a local process, and that under some unknown influence the granulations of the polymorphonuclear neutrophile leucocytes become eosinophile, the curves of the number of neutrophile and eosinophile leucocytes in these effusions varying inversely. This hypothesis as to the origin of eosinophile cells is opposed to the usually accepted view, originally put forward by Ehrlich and subsequently supported by Opie,⁶ that the eosinophile cells are always derived from the bone marrow.

Mosny and Harvier⁵ report a case of chronic syphilitic meningitis in which four lumbar punctures were performed. In the first puncture fluid eosinophiles were absent, in the second they formed 0.3 of the cells present, in the third 5 per cent., and in the fourth 9 per cent. All transitions from lymphocytes to eosinophile cells were seen. The eosinophile cells in the blood were never more than 1 per cent. This was regarded as an example of local eosinophilia due to the transformation of lymphocytes into eosinophile cells, as stated by Dominici.

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SYPHILITIC PARALYSIS OF THE OESOPHAGUS.

BY

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I AM induced to put on record the following case, as, so far as I have been able to look up the literature of the subject, it is an example of a very rare condition.

A gentleman, aged 35, consulted me on March 30th, 1895, complaining of his sight going dark at times, with giddiness, but no pain; he was occasionally faint; he had been ill for a month, and was gradually getting worse; he did not think he ever lost consciousness, and he had certainly never fallen down. His previous health had been good, and he had never had gout or rheumatism; twelve years before he had had a primary syphilitic sore. His father was alive and well; his mother was alive but insane. Two brothers were alive, one well; the other suffered from chronic heart disease, and a sister, aged 42, also suffered from chronic heart disease and kidney mischief. There was no other case of insanity in the family known to him, and no case of fits. During the attacks, which began in the fingers of the right hand and ran up to the shoulder, the arm seemed powerless; his head was drawn to the right, and his speech became thick "like a man who is

drunk"; he saw double. Ophthalmoscopic examination showed some haziness of the margin of both discs, especially the left. Physical signs were normal; the urine was thick with urates, but free from albumin and sugar.

He was given a mixture containing iodide and bromide of potassium, and on May 8th he reported that there had been no return of his symptoms since the previous visit, and on December 10th that there had been only two slight attacks of giddiness.

In January, 1898, he saw me again, complaining of giddiness and difficulty of articulation lasting a few minutes. The urine was still normal and the ophthalmoscopic examination still showed some haziness at the margin of the discs. The knee-jerks were present. His pupils reacted well to light, and Romberg's sign was absent. He was advised to resume the iodide and bromide of potassium twice a day. In June he reported that he had been quite well except for occasional giddiness with no loss of consciousness; memory and attention were good. In August he was still keeping to the medicine and was still occasionally giddy, but so slightly that people who were with him did not notice it.

He was not seen again until November, 1902, when I met his family doctor in consultation. He had had difficulty in swallowing, but there was no obstruction to the passage of a sound into the stomach; fluids regurgitated when he attempted to swallow them, and for eight days it was necessary to feed him with a tube. Under antisyphilitic treatment he made a rapid recovery and swallowed naturally, although he could not drink a draught of liquid. There was no giddiness, but he had a sensation of warmth down the left side and the left hand was unable to distinguish between hot and cold. There was no analgesia or anaesthesia. The knee-jerks were present; the pupils reacted to light, and there was no Romberg's sign.

The next note is on March 11th, 1904, when he was again seen in consultation; he thought that his swallowing was not quite what it had been, but there was no apparent difficulty either with solids or liquids. There was still imperfect sensation of heat in the left hand; his eyesight was good; he weighed 13 st. 12 lb.

On June 15th in the same year he came to see me, saying that he had kept well until recently, when he had had a good deal of neuralgia, and his eyesight had gone dim for about twenty minutes. He did not feel giddy, but he was writing at the time and only saw half the page distinctly. The haziness of the optic discs was still present. He was recommended to take his old medicine for a fortnight, and he then reported that he had had one similar attack.

I did not see this patient again until November 19th, 1913, when he consulted me complaining of sleeplessness, which had troubled him for the previous eighteen months. He said he had had no return of his old troubles, but he had had gout several times in the ankle, although not for three or four years. His pulse was 72, regular, and soft; blood pressure 130. The right hand shook, especially when he was writing; it had, he said, done so for many years, and he thought ever since the time when he had difficulty in speaking (March 30th, 1895). He was a stoutly built, healthy looking man, now aged 53; he weighed 13 st. 7 lb. He used to smoke an ounce of tobacco in a day, but had lately reduced this to two ounces a week, and had always taken little alcohol—for years none at all—but lately a glass of whisky the last thing at night; no coffee, and only one cup of tea about 5 o'clock in the afternoon. The knee-jerks were lively; the pupils reacted well to light; there was no Romberg's sign, and on ophthalmoscopic examination the discs seemed quite clear. I ordered him a draught containing 7 grains of veronal sodium and 10 grains of potassium bromide, to be taken at bedtime; on this he has slept quite well, and when last seen had apparently recovered his normal state of health.

Paralysis of the oesophagus is generally dealt with in a summary fashion by systematic writers. The causes given are diphtheria, haemorrhage, softening, tumour or sclerosis in the brain, and pressure from enlarged lymphatic glands on the vagus nerve or disease of the vertebrae, chronic alcoholism, or lead poisoning setting up neuritis. Nowhere have I been able to find any reference to syphilitic paralysis of the oesophagus, but Dr. F. W. Mott¹ has recorded the following case:

A commercial traveller, aged 38, who six months after infection, while seated at the piano singing a comic song to his own accompaniment, suddenly became faint and speechless. His

friends were at first amused, and did not realize that it was not part of the entertainment. They then came to his help and gave him a glass of water, which he was unable to swallow, the fluid regurgitating through the nose. This was followed the next day by paralysis of the left leg, left arm, and left side of the face, including the upper part, and for a month he had to be fed with a tube. This happened in Japan; the doctor treated him by inunction, and after a month he was able to swallow and speak. He was sent to me on his return from Japan suffering with the remains of a left-sided paralysis, including the upper facial muscles, and there was also paresis of the left vocal cord and soft palate. He attended my practice for some years without further symptoms, and gradually improved.

I do not believe there is any reasonable ground for questioning the syphilitic origin of my case, as it occurred as one of a series of syphilitic phenomena which yielded rapidly to specific treatment, and the patient's condition at the present time is not in favour of its being a manifestation of that curiously irregular disease—disseminated sclerosis.

REFERENCE.

¹ *A System of Syphilis*, Power and Murphy, vol. iv. Syphilis of the Nervous System, p. 92.

THE IMPORTANCE OF A LEUCOCYTE COUNT IN DOUBTFUL CASES OF APPENDICITIS.

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THE cases here recorded illustrate in a marked degree the importance of taking a leucocyte count in doubtful cases of appendicitis. This weapon of diagnosis is without doubt daily used, and is, in my opinion, of paramount importance in elucidating vague symptoms referable to the appendix region; it tells whether we may temporize, or whether, on the other hand, we have no time to spare. It is on these cases with a high leucocytosis, and with the constitutional symptoms masked, often both pulse and temperature below 100, that I wish particularly to lay stress; it is these cases which, if watched, so often end in the tragedy of acute suppurative peritonitis; it is, moreover, more common in a first attack, where adhesions do not exist to shut off an appendix distended with infected contents, with usually a stercolith, gradually burrowing its way through the muco-muscular wall of some portion of the appendicular canal; if, in these cases a count shows a leucocytosis of anything between 15,000 and 30,000, the appendix should be at once explored, and the picture usually presented is a semi-gangrenous appendix, the serous coat of which is intact, the lumen distended with foul pus, with one or more stercoliths, but no adhesions, which renders the operation of removal easy and safe.

CASE I.

R. C., aged 14 years, had had no serious illness, but had suffered occasionally from attacks of indigestion.

He was quite well at school on November 13th. He was seen by Dr. Leslie Watt at 9 a.m. on November 14th, when he complained of pain in the right iliac fossa, increased on pressure; he had vomited several times during the night. The pulse was 84, the temperature 98.4° F.; there was no rigidity of the right lower rectus abdominis. He was seen again on the same day at 8 p.m., when he looked flushed, the pulse was 104, and the temperature 100.8° F. There had been no vomiting since the early morning. Hot belladonna fomentations were applied. He was seen again at 8.30 a.m. next day, November 15th. He had had a comfortable night, and looked much better; the pulse was 84, the temperature normal, and pain slight. I was asked to see him at noon; his general appearance was good, and did not in any way suggest the necessity for an operation; pulse and temperature were normal. I took a specimen of blood, and Dr. Pethybridge reported a leucocytosis of 25,000. Immediate operation was urged.

The appendix was found lying free, without adhesions; it was of large size, 1 in. in diameter at about the centre; towards the base it appeared normal, and this was clamped, the stump buried in the caecal wall, the meso-appendix tied off, and the parietes closed in layers without drainage. On opening the appendix it was found tightly distended with foul pus, and there was a large stercolith; the anterior two-thirds of the mucous and submucous coats were gangrenous. Recovery was uneventful.

I have not the slightest doubt that, but for the blood

count, this case would in twenty-four hours have become an example of a fulminating appendicitis.

CASE II.

T. L., aged 46, seen in consultation with Dr. George Hillyar. On November 19th he played golf; at night he had pain in the right iliac fossa. I was asked to see him next day (November 20th). There was some tenderness in the right iliac fossa, but no rigidity of the rectus; the patient did not appear very ill. I was doubtful whether the appendix was at fault, as he had previously suffered from attacks of pain referred to the region of the gall bladder; a specimen of blood was taken: Dr. Pethybridge reported a leucocytosis of 14,800.

He was at once removed to a nursing home, and, assisted by Dr. Hillyar, I made an incision over the right rectus; the appendix was found much swollen, coiling round, and plastered to the fundus of the caecum; the parietes were sutured without drainage. The appendix contained a stercolith and 2 drachms of foul pus. This body had a marked resemblance to a gall stone, but it proved to be a stercolith. The patient made a good recovery and left the nursing home in seventeen days.

CASE III.

D. T., aged 8, seen in consultation with Dr. W. L. Hodge. On August 30th he was watching a cricket match; when he returned home at 7 p.m. he complained of abdominal pain; he vomited during the night and had several loose motions; the pain, which was paroxysmal and extended over the abdomen, subsided. At 8 a.m. next day (August 31st) the temperature was 100.6°, the pulse 120; acute abdominal pain returned at noon and continued till 4 p.m. and was more localized in the right iliac fossa; his condition then improved. The temperature was 100° and the pulse 98; the tongue was moist. A leucocyte count was made by Dr. Pethybridge, who reported 22,000.

Immediate operation was performed and I found the appendix of large size and gangrenous, and the gangrene had invaded the serous coat, although rupture had not taken place; infection of the fossa was evident, necessitating drainage. The base of the appendix was divided and buried in the caecal wall. The patient left the nursing home in four weeks and rapidly regained health.

In this case the symptoms were apparently improving and the patient's father, a medical man, was in favour of delay before the blood count was made. I am of opinion that with delay of twenty-four hours the result would probably have been different.

OBSERVATIONS ON THE FUNCTIONS OF THE COLON, WITH SPECIAL REFERENCE TO THE MOVEMENTS OF ENEMATA.

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A STATEMENT by Professor Rutherford Morison to the effect that enemata will travel along the whole of the large intestine and that succussion of the fluid injected can be obtained in the caecum, directed my attention specially to the subject and led to the following observations. He made this remark before x-ray apparatus was in use, and therefore before the method of examination by bismuth enemata was carried out as an aid to diagnosis of disease, and at the time, I believe, its truth was generally doubted.

It has now been definitely proved that in animals as well as human beings an enema will travel through the whole course of the large bowel, and the object of my paper is to illustrate this and to record some interesting observations, the result of personal experience with the fluorescent screen, in investigating a large number of cases when bismuth and barium enemata were employed for diagnostic purposes.

Cannon¹ has shown experimentally that in cats enemata will not only find their way into the caecum, but when injected in large quantities will travel through the ileo-caecal sphincter into the small intestine; and on one occasion he observed that the enema injected entered the stomach, for the animal actually vomited a portion of it. He found, however, that the ileo-caecal sphincter remained competent when small enemata were employed, and that to pass the caecum an enema had to be injected under considerable pressure.

Gruttyner's² observations, published in 1894, seemed to prove that a salt solution containing powdered charcoal when injected into the rectum was carried up the whole