observation has been made in all experiments in which injection has been followed by death.

The alternative hypothesis is that the symptoms are toxic in character, due either to substances dissolved out of the stromata before injection or dissolving in part, at any rate, in the blood plasma of the animal after injection. This hypothesis does not appear to us to account so satisfactorily for the variability observed in the time of onset and severity of the symptoms following injection of stromata holding liquids, as does the mechanical explanation above suggested.

Neither of the two hypotheses appears to furnish a satisfactory explanation of the occasional appearance of acute symptoms following upon the injection of solution of haemoglobin free from stromata.

Before concluding this article reference may be made to a series of seven experiments, in which from 7 c.cm. to 2.5 c.cm. per kilogram of body weight of laked rabbit's red cells were injected intravenously into rabbits in which both vagi had been divided before injection. The object of the latter procedure was to avoid the production of reflex arrest of respiration should the stromata injected be capable of exerting any marked local action upon the pulmonary tissue. These experiments, however, afforded no evidence of such action; at the time of death the heart was observed to continue beating after the cessation of respiration, exactly as occurred in those experiments in which division of the vagi had not been practised.

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A CASE OF EOSINOPHILE ASCITES: WITH REMARKS.

By H. D. ROLLESTON, M.D., F.R.C.P.,

SENIOR PHYSICIAN, ST. GEORGE'S HOSPITAL; PHYSICIAN, VICTORIA
HOSPITAL FOR CHILDREN, CHELSEA.

EOSINOPHILE ascites has, so far as I can find out, hardly ever been recorded. This rather contrasts with the attention which has been devoted to the occurrence of eosinophile cells in the exudate of experimental acute peritonitis in its earliest stages, especially by Durham, Opie, Dudgeon and Ross. But the exudate of acute peritonitis in human beings rarely contains as much as 1 per cent. of eosinophile cells. In this case of eosinophile ascites acute peritonitis brought the eosinophile count down from 22 to 0.5 per cent. The explanation of a hæmic eosinophilia of 12 per cent. is not obvious in the absence of animal parasites. Possibly it may be connected with the presence of carcinoma, for Weinberg and U. Mello¹ found 3.33 per cent. of eosinophiles in carcinoma of the colon, and 6.6 in carcinoma of the liver.

History and Course.

A male, aged 52, at the end of September, 1913, had colic and vomiting two days after eating some fish, followed gradually by jaundice and putty-like stools, and about the middle of October by transient pruritus. He was admitted to St. George's Hospital on November 4th with considerable jaundice, some rather vague resistance in the right loin, an enlarged and tender liver, and tenderness near the umbilicus. The gall bladder and the spleen could not be felt. For a time there was some apparent improvement, but on November 21st ascites was obvious, and on December 3rd 7 pints of fluid were removed; Dr. E. L. Hunt found that this contained red blood corpuscles, lymphocytes 46 per cent., endothelial cells 29, eosinophiles 22, polymorphonuclears 2, mast cells 1. The Wassermann reaction in the ascitic fluid and in the blood was negative (Mr. H. W. Bayly). A differential count of the hæmic leucocytes (total count 17,040) showed: Polymorphonuclears, 71 per cent.; lymphocytes, 16.5; mononuclears, 0.5; eosinophiles, 12; mast cells, 0. Examination of the stools did not reveal the presence of any ova of worms. The urine constantly contained bile pigment, but not bile salts, and there was no sugar; in the last week of life there was a trace of albumin on two occasions. Early in December the base of the right lung was found to be dull on percussion, breath sounds to be absent, and skiagraphy showed opacity. On December 12th the patient had severe abdominal pain, and the pulse rose to over 100. The pain gradually increased and necessitated the use of morphine. On December 14th the patient became comatose and died on December 15th at 2 a.m. His temperature, which had always been about normal, rose to 99.6° F. on December 14th.

Necropsy.

The necropsy was performed by Dr. R. S. Trevor. The skin was universally jaundiced. There was acute peritonitis with a copious effusion which, Dr. E. L. Hunt reported, contained polymorphonuclear cells 79 per cent., lymphocytes and endothelial cells 21 per cent.; a count of 400 white cells showing two eosinophiles. There were numerous minute secondary growths on the peritoneum, especially on the diaphragm, which was much thickened. There was a primary "colloid" carcinoma in the ascending colon just above the ileo-caecal valve, which was not narrowed. The caecum was high up in the abdomen on the right side. The liver, 5½ lb., contained numerous secondary growths, some of which were "colloid." There was much matting in the portal fissure, but the portal vein was free from thrombosis. The walls of the gall bladder were white, thickened, and microscopically infiltrated with carcinoma. There were no biliary calculi. The kidneys, 8 oz. each, were bile-stained, but otherwise normal. The spleen, 8 oz., was soft. There was a bilateral bile-stained pleural effusion; there was recent pleurisy on the right side; the fluid was not examined. There was extensive collapse of the lower lobes of both lungs. The apex of the right lung was free from tuberculosis, but near the apex of the lower lobe there was a small calcareous mass. There was no evidence of tuberculosis in the left lung, or of metastases in the lungs or pleurae. The heart, 11 oz., was soft, and the endocardium bile-stained. On the anterior cusp of the mitral valve there was a small serous cyst, the size of a sixpence; as this burst on examining the heart its contents could not be investigated.

Microscopically the cyst was shown to be due to oedematous separation between the superficial and the deep layers of the valve segment, and not to present any evidence of a hydatid cyst. There was no trace of hydatid disease elsewhere in the body. Microscopically the growth in the colon was a columnar celled carcinoma showing extensive myxomatous degeneration. Sections of the primary growth and of the peritoneal metastases did not show eosinophilia.

In this case the discovery of ascitic eosinophilia suggested hydatid infection of the peritoneum, on the ground that hæmic eosinophilia may accompany hydatid disease. But I was somewhat surprised at the apparent absence of any published evidence that rupture of a hydatid cyst into the peritoneum caused a local eosinophilia in that serous sac. I therefore wrote for information to Professor F. Dévé, of Rouen, the authority on hydatid disease, who very kindly replied:

Peritoneal eosinophilia does, indeed, sometimes occur as the result of intraperitoneal rupture of a hydatid cyst—with or without choleperitoneum—but, so far as I know, one case only has been recorded—namely, in Brouqueyre's thesis, "De l'éosinophilie générale et locale dans les kystes hydatiques" (*Thèse de Bordeaux*, 1905, p. 70). This observation was also given *in extenso* (without any fresh cytological details) in Tremblin's thesis, "De l'ouverture spontanée des kystes hydatiques du foie dans le péritoine" (*Thèse de Paris*, 1906, No. 283, p. 45, Obs. I). This is the extract from Brouqueyre's thesis: "M. Leveboullet has told me of an extremely interesting case which he watched when under the care of Professor Gilbert. A hydatid cyst of the liver ruptured, but none of its contents passed into the peritoneum. The usual phenomena due to a secondary ascites of no great size led to paracentesis. The fluid withdrawn was deeply bile-stained, and, on centrifuging, gave a deposit containing a considerable quantity of eosinophile cells, at least 50 per cent."

I have seen a case of the same kind, which, though I intend to publish it eventually, you are at liberty to quote. In February, 1913, I diagnosed a hydatid of the liver in a man of 23 years of age, and advised operation without delay. Four days later it ruptured spontaneously when he was sitting quietly at his desk doing accounts. Laparotomy twenty-seven hours after the rupture revealed rupture of a hydatid cyst on the anterior surface of the left lobe of the liver and 3 or 4 litres of turbid fibrinous fluid the colour of orangeade and containing scolices. Bacteriological cultures proved that the fluid was sterile, and chemical analysis that it contained urea (0.45 per cent.), fibrin, cholesterin, bile pigments, and traces of glucose, but no bile salts. Cytologically I found polymorphonuclear neutrophiles 85 per cent., eosinophiles 8 per cent., small and medium-sized mononuclears 0.5, large mononuclears (macrophages) 0.5, and red blood cells 5.3 per cent. At the time of the operation the blood contained 2.7 per cent. of eosinophiles (before the rupture it was only 0.9 per cent.).

Dr. d'Este Emery has told me that on one occasion he found 10 per cent. of eosinophiles in the ascitic fluid of a man who afterwards died of abdominal malignant disease. I have not obtained any other information from published

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

ROBERT SAUNDBY, M.D. EDIN., F.R.C.P. LOND. AND L.,
LL.D., M.Sc.,

PROFESSOR OF MEDICINE, UNIVERSITY OF BIRMINGHAM; CONSULTING
PHYSICIAN TO THE GENERAL HOSPITAL, BIRMINGHAM.

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