here and there show attempts at a palisade-like arrangement; they contain very little protoplasm. The original structure of the lung is only indicated by still surviving bronchial cartilage belonging to the larger bronchi, and an occasional small group of glands belonging to the bronchial wall. Fairly numerous blood vessels with well-formed walls are present which may be still surviving vessels of the lung. In many regions marked degeneration is taking place with disintegration of nuclei and general loss of staining power. In these areas accumulations of fat are present. Fat is also present in considerable quantities in tissue which appears to be fibrous in nature, rapidly degenerating and undergoing invasion by columns of cells. The nature of the tumour is carcinoma of the cubical-cell type which has probably grown from the terminations of the bronchioles. The secondary nodules in the liver are very similar except that much more fibrous tissue stroma is present, the cells tend to more regular palisade arrangement, and are in places more of a columnar type. The tumour is not sharply marked off from the liver, which is being rapidly invaded at the growing edge. Isolated batches of liver cells can still be identified here in the fibrous stroma; further in the nodule they have become reduced to little masses of bile only.

The chief points of interest in this case are: the age of the patient, 27 years; the duration of symptoms, five months; the similarity to tuberculosis of the lungs—for example, wasting, fever, cough, haemoptysis, pleural effusion, and pneumothorax; the temporary improvement under open-air treatment, due to the elimination of secondary infection; the total absence of pain; the involvement of one lung only, and, finally, the rapid enlargement of the mediastinal and cervical glands. The right lung was extensively involved while the left lung was unaffected, but yet the left cervical glands were considerably more affected than the right, the path of spread being probably along the course of the thoracic duct.

REFERENCES.

¹ *Oxford Medicine*, vol. ii, p. 82, 1921. ² *Neoplastic Diseases*, Philadelphia, 1920. ³ *Medical Notes*, p. 55, Oxford Medical Publications, 1921.

BLINDNESS AS AN IMMEDIATE SEQUELA OF INFLUENZA: RECOVERY.

BY

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THE following case well exemplifies the conditions described by Jelliffe and White as quoted below. The patient, a young woman aged 17, was, during convalescence from influenza, attacked with papillitis, which was accompanied by blindness of rapid onset and frontal and temporal pain. The sight was gradually recovered in the course of three or four weeks. There was no evidence of cerebral tumour or abscess, or of syphilis, and the Wassermann reaction of the cerebro-spinal fluid was negative.

In view of the large number of cases of influenza at present occurring, the case may be of interest, and useful in that it shows that the prognosis of the blindness is good.

A girl, aged 17, was admitted into the Hull Royal Infirmary on January 4th, 1922, under my care, from the ophthalmic surgeon's out-patient department, by Dr. Bainbridge, who sent the patient in for medical treatment with a diagnosis of "papillitis." He reported the eye condition as follows: Pupils widely dilated, but active; no pain on moving eyes by palpation; consensual reaction present; periphery of fundus not much affected; discs swollen; edge blurred, veins dilated.

The history was that three weeks before admission she was taken ill with influenza, and was in bed for one week. There was no cough. A week later her eyes and eyelids hurt her, and "a dark mist came over her eyes." She could not see to read in bad light or good. In the ward we found that she was blind in the right eye, with no perception of light. With the left eye she could see fingers at one foot. On ophthalmoscopic examination there was marked optic neuritis (papillitis) of both eyes. She had a headache (frontal and left temporal region) which had lasted fourteen days. There was no discharge from the ears, and the right and left drums were normal; in the right ear a whisper was heard at seven feet, with the left conversational voice at five feet only. Bone conduction was slightly diminished in the left ear. There was no tenderness over either mastoid, and no discharge from the nose.

Dr. Ritchie Rodger reported that he could not find anything to suggest ear, nose, or throat origin. The heart was not enlarged; the first and second sounds were loud, and there was no bruit. Lungs and abdomen showed nothing abnormal. The urine, specific gravity 1020, was acid, and contained a trace of albumin; the daily amount was thirty-six ounces; urea, eight grains per ounce. Blood pressure, 95 mm. Hg.

Central Nervous System.—No Kernig's sign, but slight tension on hamstrings of both legs; knee-jerks normal; Babinski's sign not

present; no ankle clonus; no inco-ordination. Mentally decidedly dull, answers questions slowly.

A swab taken from the nasopharynx, from which there was a slight discharge, on culture showed pneumococci, staphylococci, and *Micrococcus catarrhalis*.

Treatment.

The patient was given a farinaceous diet, and milk and fish. The following mixture was prescribed: Potassium citrate gr. 5, liq. ammon. acet. in 15, hexamine gr. 5, aq. ad 1 oz.; to be taken four times a day and followed by barley water 2 oz. One hot pack to be given.

On January 7th lumbar puncture yielded 15 c.cm. clear cerebro-spinal fluid; it contained a few lymphocytes but no organisms on culture.

January 9th. No headache; can see hand with right eye.

January 10th. Sees fingers with right eye at 2 feet; with left eye at 7 feet. No headache; is taking food well. There was now no tension on hamstrings; no ankle clonus; no anaesthesia; no inco-ordination; knee-jerks normal; optic neuritis was still present (oedema of discs).

January 11th. No headache; vision the same; 10 c.cm. clear cerebro-spinal fluid withdrawn by lumbar puncture was negative to Wassermann test.

January 13th. Blood pressure 95 mm. Hg; optic discs slightly improved. Pneumococcal vaccine 1 million given.

January 20th. Ophthalmic surgeon's report: She can now read ordinary print; optic discs nearly normal. Right fundus, veins dilated, otherwise nearly normal. Left fundus, disc normal. There was no albumin in the urine. Blood pressure, 102 mm. Hg.

January 23rd. Can read without pain or headache; she feels quite well; oedema of legs and albumin have disappeared.

January 27th. She feels quite well, and is getting up. The ophthalmic surgeon reported: Slight swelling of right optic disc and veins moderately dilated. R.V. 6/24; with -1 cyl. axis 90 vision equals 6/18. Left eye: Fundus normal, vision 6/12; emmetropic. In the ward she was found to be able to read ordinary small print quite well, in not too good a light. She stated that she felt nothing wrong with her head.

De Schweinitz, in his *Diseases of the Eye* (ninth edition), says: "In addition to the intracranial causes of papillitis this phenomenon may arise from a general infection." To this form Uthoff gives the name "infectious optic neuritis." According to this observer it should be differentiated from those cases which are caused by orbital, intra-ocular, or intracranial lesions, and may be caused by any of the following diseases, placed in order of their frequency: Influenza, syphilis, rheumatism, malaria, typhus fever, measles, whooping-cough, diphtheria, polyneuritis, small-pox, beri-beri, erysipelas, scarlet fever, tuberculosis, typhoid fever, gonorrhoea, relapsing fever.

The neuritis may manifest itself as a papillitis or as a retrobulbar neuritis, and Uthoff thinks that the optic nerve conditions are most apt to arise during the stage of convalescence and are probably due to the action of toxins and not directly due to micro-organisms.

Again, Jelliffe and White, in *Diseases of the Nervous System* (third edition), say: "Acute axial neuritis occurs usually in young adults, 12 to 24 years of age, more particularly women; frontal or temporal headache, or deep pain in orbit, made worse by pressure or by movements of the eyeballs; loss of sight is rapid, reaching a maximum in five days, and often is so severe that the patient can just count fingers at fifteen feet or is blind."

After the acute stage is over there is gradual recovery of sight. For a more detailed account see article by Jelliffe, "Nervous and mental involvements of influenza," *New York Medical Journal*, November, 1918.

I should like to draw attention to the condition of "slight tension on the hamstrings of both legs." I have noticed it several times in cases where I was of the opinion that an infection had become generalized. It is not Kernig's sign, but approaching it. It is probably due to an excess of toxin in the blood; possibly it may be due to organisms in the blood, but this is, I think, unlikely, as cases in which I have noticed it have in some instances cleared up too rapidly. There is no head retraction or even stiffness or pain on twisting the head or neck in any direction. Also if in a case showing it the test be made daily it will be found gradually to get less in cases that are improving.

A patient showing this sign I was asked to see some months ago was a child, aged 6 years, with acute abdominal pain and vomiting, accompanied by diarrhoea and fever. I decided against any operative interference, chiefly on this sign. The abdomen was tender all over and slightly rigid. The fact that there was slight tension on the hamstrings pointed, in my opinion, to a generalized infection. In appendicitis the same condition is sometimes seen, but only in one leg, or it is much more marked in one leg—namely, the right. In the case of this child I thought that there was a slight early

peritonitis, or a condition which is a prelude to that, possibly pneumococcal. The condition cleared up in twenty-four hours on a powder of calomel gr. 1/8, sodium salicylate gr. 2, bismuth carbonate gr. 3. The implication of the nervous system is probably more intense in such infections.

The Lettsomian Lectures

ON

AMOEBIIC LIVER ABSCESS.

DELIVERED BEFORE THE MEDICAL SOCIETY OF LONDON

BY

SIR LEONARD ROGERS, C.I.E., M.D., F.R.S.

[Abstract.]

LECTURE I.—ETIOLOGY AND PATHOLOGY.

SIR LEONARD ROGERS began by saying that when he received the invitation to deliver the Lettsomian Lectures he cast about in his mind for a subject from among the many diseases which he had been investigating during his twenty-seven years of research in India. In view of the fact that he had been awarded the Fothergillian medal of the society for his work on amoebic diseases, he came to the conclusion that he might suitably deal with amoebic hepatitis, more especially as the late Dr. Sandwith had ably dealt with dysentery in the Lettsomian Lectures of 1914. The present course, therefore, would be complementary to that given by Dr. Sandwith, and would deal with that phase of amoebic disease investigated at Calcutta during a period when the knowledge of hepatitis generally had made great advances. The work on the liver had been one of the most significant episodes in recent tropical medicine, although perhaps less dramatic than some discoveries, because more gradual.

A Review of Progress.

At the beginning of the present century there was a good deal of confusion with regard to the relationship between dysentery and large single tropical liver abscess. The confusion was due to ignorance of the fact that there were two great classes of dysentery which, until recently, had been regarded as one definite disease. Those whose experience was in countries where the bacillary form prevailed denied its relation to liver abscess; while in Egypt and India, where amoebic dysentery was known to be common, observers had noted a relation. It was now understood that the prevailing type of dysentery in temperate climates was bacillary. In John Hunter's account of dysentery in the army in Jamaica in 1788 he did not mention liver abscess, and considered the dysentery to be the same as that described by Sydenham as so prevalent in London in 1779-80. Recent investigation showed that to this day the bacillary type prevailed in Indian gaols, where the amoebic form was comparatively rare. In the *Researches into the Causes, Nature, and Treatment of the More Prevalent Diseases of India and of Warm Climates Generally*, by James Annesley, of the Madras Medical Establishment, published in two volumes by the India Office in 1828, it was clearly stated that liver abscess was secondary to dysentery. It was quite certain from these finely produced and illustrated volumes that amoebic dysentery was common in Madras early in the nineteenth century.

In the first three editions of his classical work on tropical diseases Manson described dysentery as a single entity; he pointed out that the term probably included a group of diseases, but that up to then the claims for different causative agents, whether bacterial or protozoan, were far from having been established. The confusion at the beginning of the present century was well illustrated in the contradictory nature of articles on the subject in the first edition of Allbutt's *System of Medicine*. Altogether, it was not surprising that the amoeba was not accepted as the cause of the disease. Manson, in his early edition, stated that amoebae were present in more than half his cases, but this did not convince him of their causative powers. At the annual meeting of the British Medical Association in 1902 Andrew Duncan, who had then recently retired from the Indian Medical Service, opened a discussion on dysentery, and advanced the view that the amoeba had nothing to do with dysentery in any country as a causative agent.¹ In Duncan's view, large tropical liver

abscess did not bear any specific relation to antecedent dysenteries, although small multiple abscesses did. Very similar views were also expressed at this time in a short editorial note in the *Indian Medical Gazette*. In spite of the valuable work of several observers, especially Councilman and Kartulis, medical opinion at this time was against the theory of amoebic causation.

Constant Presence of Amoebae.

Sir Leonard Rogers went on to say that he had published his first paper on dysentery in 1902. It was based on two years' research he had conducted in Calcutta between April, 1900, and the end of 1902, making use of all the clinical and *post-mortem* material he could obtain. The first point he had set out to determine was the frequency with which living amoebae might be found in large tropical liver abscess. It was easy to detect the amoebae *post-mortem* in the walls by examining scrapings under the microscope, but much more difficult to detect them in the thick pus of such abscesses. In order to get a larger range of cases than was afforded by *post-mortem* examinations he arranged with his surgical colleagues, when they opened a liver abscess, to place some of the thick pus in one sterile test tube, and in another the light scrapings from the wall of the cavity. Out of some 18 cases examined in this way, he found the amoeba in the pus in four, but in the scrapings of the wall he found it in every case except one. In 20 cases examined *post-mortem* he again found the amoeba in the wall in every case except one, the exception being an instance in which the abscess had been opened and drained for fourteen days before the patient died. From this he concluded that amoebae were practically invariably present, a fact which he had verified in scores of later cases, the only exceptions being small and encysted abscesses in which the amoebae seemed to have died out.

Usual Sterility as regards Bacteria.

He turned next to the question of the frequency of pus-forming bacteria in large tropical abscess. Out of 24 cases in which cultures were made from the pus taken in sterile test tubes at the time of operation, 15, or 62 per cent., were sterile. The *post-mortem* examinations told the same story. In three out of five unopened abscesses found *post-mortem* there were no bacteria. In a later series of observations, out of a large number of consecutive cases he found 86 per cent. sterile when first opened. It was clear that the great majority of such abscesses were free from bacteria that could be cultivated, although few such abscesses remained sterile in a tropical climate after opening and drainage. His conclusion was that not only were amoebae constantly present, but other causal bacterial organisms were constantly absent. This indicated that *Entamoeba histolytica* was the causative organism in these cases of liver abscess.

The Antecedent Dysentery.

The striking fact had not been explained that the proportion of cases of liver abscess to cases of dysentery in the British Army in India was 1 to 5 or 1 to 7, while in Indian gaols it was one to several hundred. But if the prevailing type of dysentery was amoebic in the army, and bacillary in the gaols, the problem was solved; this was now known to be the case.

It had next to be determined what was the frequency of association between any form of dysentery and liver abscess. Among the cases of liver abscess which he had tabulated for the purpose of the lecture, in four there was no history of dysentery, but even in these, dysenteric ulcers were found *post-mortem*, proving that the absence of clinical signs of previous dysentery did not exclude the presence of amoebic ulcers in the large bowel. In three other cases there had been a history of dysentery and no ulcers or scars were found *post-mortem*, but only slight congestion; thus it was possible for antecedent dysentery to clear up without necessarily involving any ulceration before the patient died of liver abscess. In order to get accurate data it was essential to obtain both clinical and *post-mortem* records, and even then it was possible that a mild attack of bowel trouble might be overlooked clinically and leave no *post-mortem* evidences. The cases shown in his tables in which the clinical and *post-mortem* records were complete numbered 24, and in 23 (or over 95 per cent.) there were either clinical or *post-mortem* evidences of dysentery, while in 14 (or 58 per cent.) there were both clinical and *post-mortem* evidences. On adding certain older records which clinically were less complete, only

¹ BRITISH MEDICAL JOURNAL, 1902, vol. ii.