Patients who have a mechanical obstruction nearing completion suffer from attacks of spasmodic colicky pain with short intervals of relief. If this fits in with the time factor the presence of obstruction should be suspected. Visible coils of gut are almost diagnostic of obstruction, and if peristalsis is either visible or audible the diagnosis is no longer in doubt. Vomiting is a late sign of obstruction. It means that the distension has reached the upper coils of gut. It is matter for congratulation if the diagnosis is made and the second operation performed before the patient has vomited.

Lastly, though the results of an enema and the passage of flatus are strong negative evidence that obstruction is not complete, they are not absolutely so. The worst post-appendix obstruction I ever operated on had had a "good result" that morning. In this case the small gut was so distended that it blew out and burst like a bicycle tyre when the abdomen was opened.

Post-operative Peritoneal Infection.

Repeated dark vomits, distension, hiccough, and marked toxæmia, are evidence that the patient, as a whole, and his peritoneum in particular, are failing to cope with the virulence or numbers of the organism. Further operation holds out little prospect of doing any good. There is no one collection of pus that can be effectively drained. The gut is friable and will not bear the gentlest handling, and stitches will not hold if it is torn. The only chance for such patients, and it is a slender one, lies in absolute rest, morphine, and rectal or subcutaneous salines with glucose.

Remittent pyrexia should lead to examination of the abdomen, digital examination of the rectum, and examination of the bases of the lungs. If a residual abscess is diagnosed it is well to wait a little and watch progress unless the patient is going downhill. The majority get smaller and finally resolve. If the abscess gets bigger or the patient deteriorates it must be drained in the appropriate manner. It is best not to await rupture into the rectum or vagina. Such cases do better if deliberately and adequately drained.

A CASE OF PRIMARY CARCINOMA OF THE LUNG.

BY

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THIS case was under continuous observation by one of us in the wards of Addenbrooke's Hospital for four months before death, and in the earlier stages simulated tuberculosis with somewhat interesting and rapid alterations in the physical signs. The correct diagnosis was only reached when definite enlargement of glands above the left clavicle was found to have taken place. Owing to the length of time the case was under observation it seemed to us to be worth putting on record.

Primary carcinoma of the lung is an uncommon disease. Hamman¹ states that 105 cases occurred in 46,169 autopsies, a percentage of 0.2; figures given by Ewing² show that 1.0 per cent. of primary cancers occur in the lung.

Primary growths are classified in three groups according to their origin: (1) From the bronchial epithelium; (2) from the bronchial mucous glands; (3) from the lung substance. The type of tumour found in group (1) seems to be localized to the larger bronchi and not to spread diffusely, to break down easily and form bronchiectatic cavities, being histologically essentially columnar-celled. Tumours of type (2) are characterized by abundant mucous secretion derived from the special type of cell of which the growth is composed. They also tend to be localized round the larger bronchi. The third group comprises the diffuse cancers with widespread destruction of lung tissue by rapidly spreading infiltration. The type of cell found in these varies, even in different parts of the same tumour, from columnar to flattened epithelium-like cells. The greater mass of cells are usually cubical in shape. The point of origin of tumours in the third group is difficult to determine, as their growth is rapid and diffuse. The cubical type of cell points to an origin either from the bronchioles or from the lining cells of the alveoli, whose shape often becomes cubical in collapsed lung. The matter is, however, of academic importance. Our case falls within the third group, the tumour being diffuse and the preponderating cells being cubical in type.

Clinically, carcinoma of the lung presents itself as a chronic pulmonary inflammatory disease, as a chronic pleurisy with effusion, or as a local patch of permanent consolidation; the usual diagnosis is pulmonary tuberculosis, fibroid phthisis, or unresolved pneumonia. The symptoms which suggest growth are rapid loss of weight and strength, pain, dyspnoea, and peculiarities in the physical signs, which are frequently unilateral. Pain is by no means always present, but severe continuous pain in the absence of pleurisy, or a constant boring pain with indefinite or unusual physical signs, should arouse suspicion. Fever, though always present in some degree, is usually much less than is found in tuberculosis rapidly advancing; the temperature may not rise above normal for days or even weeks. Sputum may be absent for some time, but in the later stages is frequently abundant; it is usually mixed with blood, and has been likened to currant jelly. Persistence of bloody sputum for weeks and months is very suggestive. The pleura is so commonly involved that pleural effusion is one of the chief signs of the condition; it is often smaller in amount than the physical signs would lead one to anticipate, and it is frequently tinged with blood; but the commonest cause of blood-stained pleural effusion is not malignant disease, it is tuberculosis (Horder³). Pneumothorax is a not uncommon occurrence in the course of the disease. Most of these points are illustrated in our case.

A. T. R., aged 27, a french polisher, was admitted to Addenbrooke's Hospital, Cambridge, on December 11th, 1920. He complained that for the past three weeks he had been troubled by shortness of breath, cough, and blood-spitting. He had previously had good health; he had, however, been discharged from the army in March, 1918, for deafness following shell concussion; for this disability he was pensioned. At the time of his discharge he had a cough, which disappeared after treatment for a few weeks in hospital.

He was a poorly nourished man, weight 7 st. 9 lb., a little dyspnoeic, not cyanosed. The physical signs were those of pleurisy, with a little effusion and patchy consolidation of the lower lobe of the right lung; the condition was thought to be tuberculous. During the first fortnight in hospital there was some loss in weight, a daily evening rise of temperature to just under 100°, and an increasing pulse rate. He had a little cough, with the daily expectoration of thin blood-stained sputum in which tubercle bacilli could not be found. Towards the end of December the patient had lost 6 lb. in weight, the evening temperature was rising to just over 100°, the pulse rate still increasing and the physical signs extending.

He was now placed on a balcony under open-air conditions, and at the end of three weeks had improved; he had regained 6 lb. in weight, there was little cough or sputum, though this was still blood-tinged; the evening temperature was not over 99° F. for fourteen days. The physical signs were much the same in the right lung; there were only occasional rhonchi in the left.

On January 25th, 1921, well marked signs of pneumothorax were observed; there was no pain or distress and the onset had evidently been insidious. A skiagram showed a right hydro-pneumothorax, and that the heart and aorta were displaced towards the right. A few days later blood-stained fluid to the amount of 4 c.cm. was withdrawn from the right thorax; the fluid was sterile and contained polymorphonuclear and mononuclear cells in equal quantity. In the middle of February the patient, who had become much emaciated, complained of constant gnawing pain in the right hip and thigh; in spite of treatment this continued almost without remission to the end.

The pneumothorax slowly disappeared, leaving the whole of the right side of the chest flat and immobile, dull to percussion, with very feeble breath sounds; the heart was displaced towards the right. Early in March enlargement of the left cervical glands was noted; they were soft, painless, and not tender; they rapidly increased in size, and those on the right side became palpable and visible. Towards the end of the month hoarseness, stridor and oedema of the face commenced and the superficial veins of the upper thorax became engorged. These signs of obstruction increased, and the patient died on April 15th, 1921. During the last eight weeks the temperature was normal or subnormal.

At the necropsy the whole of the right lung was found to be tightly adherent to the thoracic wall; the lobes were indistinguishable, almost the whole lung being involved in a hard white fibrous growth, from the cut surface of which thin white material could be easily scraped. The growth was most dense at the hilum and was found to involve the wall of the bronchus almost up to the bifurcation. The only portion of the lung comparatively normal was the extreme lateral part, which was collapsed. The left lung was entirely free from growth, and was emphysematous and congested. The left pleural cavity contained about a pint of milky fluid, chylous in nature and probably due to involvement of the thoracic duct; the pleural walls were in a condition of gelatinous oedema. Bronchial and tracheal glands were involved on both sides, forming a continuous mass with the greatly enlarged glands in the neck. The lumen of the superior vena cava was much narrowed by constriction. On the upper surface of the liver were four small nodules of growth; two of these were half an inch across and two less. The posterior abdominal glands were enlarged, but none were more than half an inch in diameter; on section they showed a similar appearance to that of the main growth.

Microscopically the lung substance is replaced by a mass of cubical cells which, though in most places irregularly arranged,

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.

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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