left iliac region, where there was some increased resistance over the line of the colon. There had been constipation for two or three days, so the bowels were relieved by an enema, a well-formed motion being followed by an acute diarrhoea with plenty of mucus and bright blood. The above symptoms were accompanied by vomiting which occurred several times. The free hæmorrhage from the bowel continuing through the day, the condition of the patient became such that it was imperative to do something to stop it. As stated before there were some fulness and increased resistance in the left iliac fossa with tenderness; no actual tumour was to be made out here or elsewhere in the abdomen. These signs with the bright blood pointed to the part of the bowel involved—namely, the sigmoid flexure, and it was evident that some large vessel was opened. The diagnosis rested between malignant disease and ulceration of the colon and the signs in general suggested that it was the latter condition. I decided to open the abdomen in the left inguinal region so as to explore the sigmoid flexure and to control the bleeding spot.

An incision three inches long was made, the muscle was divided, and the parietal peritoneum was fixed to the skin margin. The sigmoid loop was drawn out and the cause of the hæmorrhage was fortunately discovered. There was a large ulcer about two inches by one inch in the long axis of the bowel and along its free edge; the base of the ulcer appeared to be practically only peritoneum. As it seemed certain that this ulcer would perforate this segment of the gut was brought up out of the wound and fixed there by passing a glass rod through the mesosigmoid and packing it round with gauze. The bowel was not opened. There was no further pain or hæmorrhage and the patient took his fluid nourishment well. On May 7th in the early morning the bowel opened spontaneously at the site of the ulcer; during the day he was not so well, getting gradually more feeble, and he succumbed during the night.

At the post-mortem examination there was found well-marked ulcerative colitis beginning in the cæcum and extending to the lower end of the sigmoid flexure where the changes were most marked. Here the bowel was thickened and contracted, showing many small circular sharply cut ulcers as well as the one large ulcer before mentioned; many of these were evidently of long duration. The rectum was practically healthy. Under the microscope the bowel gave no evidence of malignant disease. Very marked granular contracted kidneys were present.

Upper Wimpole-street, W.

A CASE OF ANEURYSM OF THE AORTA RUPTURING INTO THE PULMONARY ARTERY ASSOCIATED WITH FIBROSIS OF ONE LUNG.

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(With a Note on the Lung by Dr. R. N. SALAMAN, Acting
Director of the Pathological Institute, London Hospital.)

THE following case seems sufficiently rare to merit publication. The patient was a soldier, aged 31 years, sent to me by Dr. Alexander of Bromley on Jan. 26th, 1904, on account of shortness of breath and some swelling of the right side of the neck and face; he also complained of a cough and pain in the back. For the following notes I am indebted to my house physician, Dr. H. E. Ridewood. Of the patient's family history nothing need be said. His personal history was that he had been a soldier for many years in India and had had there gonorrhoea with bubo and gleet but had no recollection of syphilis, though it was obviously possible if not probable. He had malaria in 1895 and recently—up to three or four months previously—had been working as a labourer. He drank moderately. The history of his present illness was that he was well enough up to some three or four months ago, when he began to complain of a cough and of shortness of breath. Both these symptoms had gradually but steadily and progressively got worse; he also complained that they were worse in the morning after lying down and

occasionally had caused a distinct feeling as though he would choke. On one or two occasions lately they had actually caused a fainting attack. In addition to these symptoms he had (1) a dull aching pain starting from the right side of the root of the neck radiating upwards into the head, worse at night and on coughing; (2) swelling of the right side of the neck, face, and shoulder, which was worse on lying down; and (3) difficulty in swallowing, all three of which had, like the other symptoms, gradually increased in the last three months.

On admission the patient was a thick-set muscular man of medium height, very obviously suffering from genuine difficulty of breathing associated with a swollen condition of the upper part of the right chest and neck. He was not at all wasted nor cachectic nor, indeed, feeling very ill in himself and had walked up to the hospital and to the ward unaided. His temperature was normal. Examination of his chest gave the following physical signs. Over the swollen area the veins were more obvious to the eye than on the corresponding parts of the left side but they did not pulsate and would not fill from the cardiac side. The swelling itself was doughy but without any pitting on pressure anywhere. He had perceived it himself because he could no longer wear his usual 16½ inch collars. His breathing, which he himself described as shortness of breath, was very typically—so much so that I used him for clinical demonstration of the point—that due to some form of laryngeal obstruction with slight stridor on inspiration, more marked on slight exertion or on lying flat on his back in bed. The cough was dry and brassy in character and the respirations were 40 per minute.

Palpation enabled us to fix the heart's apex beat just above the sixth left rib five inches from the mid-sternal line. The general cardiac impulse was slightly heaving as though the organ were somewhat hypertrophied. On percussion the cardiac dulness was practically normal, extending from the apex in a slanting direction inward to the junction of the third rib with the sternum and bounded by the left border of the sternum. There was an area dull to percussion over the first and second right ribs and the intercostal space between them and extending over the upper part of the sternum even to the first intercostal space on the left side. Auscultation of the heart revealed the following condition. At the apex the sounds were clear and somewhat exaggerated, at the base the second aortic was slightly accentuated, over the dull area the cardiac sounds were very clearly heard, but nowhere was there anything like a bruit nor thrill nor undue pulsation. On auscultation of the lungs the inspiratory breath sounds were exaggerated apparently, but this was, I think, merely a conduction of the tracheal sound; otherwise there was nothing unusual except an occasional rhonchus and a few crepitations at either apex. In view of the post-mortem changes it must be distinctly noted that I could not remember any noticeable difference in the breath sounds on the two sides except the ordinary normal louder sounds on the right.

Features external to the chest—Tracheal tugging was looked for and my house physician thought he could appreciate it but I was unable to do so; he also states that it became more marked in the course of time. The pulse was 84 per minute, of medium tension; the vessel wall was palpable on rolling it under the finger but could not be styled very degenerate. There was no appreciable difference in time or volume between the two radial pulses. The carotids could not be compared owing to the swelling of the neck. The pupils were equal and active for accommodation and to light. There was no paralysis of either vocal cord. To account for the pain in the back nothing could be seen, felt, or heard; an occasional pain down nearly to the wrist on the right arm seemed to own a causation in common with the swelling. The blood, urine, and abdominal organs were all carefully examined but showed nothing pathological. There was some dysphagia which had recently become more marked. The only other point that seems to call for notice was one which Dr. Alexander communicated to me but which I did not notice in hospital, that the left side of the face would swell if he lay on his left side.

The patient was treated with morphine, iodide of potassium, &c., but the treatment presents no features of interest except that he may be said to have improved somewhat. On Feb. 3rd, 1904, he seemed as well as usual. On the 4th he asked for the nightstool but did not use it, for he was at this moment seized with an attack of coughing and then suddenly complained of an acute pain in the cardiac region, his face and lips became blanched, the

pulse at the wrist disappeared, his respirations became rapid, from 60 to 70 per minute, and the stridor mentioned earlier disappeared. Dr. Ridewood was opportunely in the ward and saw him at once; he noticed the above points and naturally thought the aneurysm had ruptured into some natural sac; he examined him as carefully as the condition would allow and noted that there was no sign of fluid in either pleura nor was there any alteration in the pulmonary physical signs. There was no increase in the area of cardiac dullness, the heart was acting regularly and quietly, about 85 per minute, and the sounds were quite clear and well heard, contrasting, as he remarks, strongly with the absence of radial pulse. No bruit was heard, the patient did not vomit, there was no hæmoptysis, in fact, the above points—pallor, rapid breathing, and absent pulse—were the only indications of some accident. He remained for some time without change and then the respirations became of a sighing character and he died as nearly as possible two hours after the pain suddenly seized him.

Necropsy.—Post-mortem examination revealed an aneurysm of the ascending and transverse arch of the aorta of about the size of a small orange which had ruptured into the pulmonary artery; the ruptured aperture was large enough to admit the tip of the little finger and was situated about an inch or rather more from the pulmonary valve well on the heart side of the bifurcation of the pulmonary artery; the edges of the rupture were evidently fresh. This aneurysm showed no laminated clot; it did not involve the aortic valves; these were quite sound as far as ordinary appearance would show; it reached backwards so as to press upon the bifurcation of the trachea, thus explaining the dyspnoea and dysphagia. On the posterior surface of the abdominal aorta was a second aneurysm just opposite the origin of the celiac axis; this was of about the size of a hen's egg, typically sacculated, and filled with laminated clot; it had eroded the vertebral column, making for itself a nest as it were; this cleared up satisfactorily the pain in the back, though it had given no other indication of its presence. The heart was slightly larger than usual but showed no other pathological condition to the naked eye; the valves were healthy. The spleen weighed 17 ounces and was very adherent to the diaphragm. The liver weighed 74 ounces and was adherent to the diaphragm. The kidneys were practically healthy. Of the lungs, the right was slightly cedematous and showed a small patch of tiny subpleural ecchymoses; these without close scrutiny might have escaped observation and except for them there was not the slightest trace of any unusual ante-mortem changes in the circulation through either lung. This was particularly noticed owing to the nature of the accident that had caused death. The left lung was in a peculiar condition of fibrosis (there was no trace of this in the right one). I am indebted to Dr. Salaman, acting director of the pathological institute of the London Hospital, for the following careful description of it which adds greatly to the interest of the case.

Note by Dr. SALAMAN—The left lung was firmly adherent to the chest wall; the pleura was thickened, especially along a line corresponding to the necks of the ribs behind. The fibrous tissue of the thickened pleura did not extend into the lung tissue. The bronchial glands were not enlarged and except for some anthracosis were normal. On section the whole lung tissue, although aerated throughout, was found to be uniformly dense and fibrotic; the fibrous tissue was to the naked eye more obvious at the root of the lung and along the track of the bronchi. There was no caseation nor puckered scar anywhere.

Microscopical examination of the tissues at the root as well as of those along the free margin of the lung showed a very definite fibrosis of the alveolar tissue brought about by granulation tissue in the septa and alveolar walls which had spread in from the large collection of older granulation of fibrous tissue accumulated around the bronchi and vessels. In many places, both in sections of the lung at the root and at the free margin, groups of condensed granulation tissue were met in which were numerous large giant cells sometimes surrounded by definite epithelioid cells and sometimes by more dense fibrous tissue. The giant cells were mostly very large and contained some 20 or more peripherally arranged nuclei. Some giant cells were smaller but they also contained a large number of nuclei. 13 preparations were most carefully searched for tubercle bacilli without success. In the younger granulation tissue at the periphery of the older nodules there were very many alveoli inclosed which, although possessing no lumina,

still were lined by well-preserved cells whilst occasionally the appearances strongly suggested a new formation of epithelial tubular structures—such an apparent or real formation I have noticed in an undoubted specimen of syphilitic lung disease. In no section was there any sign of caseous degeneration. The blood-vessels, except for the marked periarteritis, were normal.

The result of the macroscopic and microscopic investigation tends to the view that the cirrhosis of the lung is syphilitic in origin; such a change in the lung in acquired syphilis is of the greatest rarity, and it is interesting to note that the condition was present in one lung only. The morbid change is very different from the one observed in congenital syphilis where both lungs are uniformly involved and the cirrhosis is not essentially bronchial or vascular in its distribution.

Note by Dr. SMITH.—Aneurysms of the aorta bursting into the pulmonary artery seem to be very rare. Ziegler mentions the incident as occasionally occurring but I could nowhere find a detailed account of the symptoms arising from it, which must be my excuse for a somewhat prolix account of the present case. Dr. G. Newton Pitt informs me that he has met with a case which lived for three weeks after communication had been established and in which a secondary aneurysm arose in the pulmonary artery. It is to be hoped that he will place the case on record. In my case, judging by the absence of pulse at the wrist, aortic blood pressure must have very rapidly sunk to the level of that in the pulmonary artery and death must have ensued from imperfect distribution of blood to the brain which was rapidly starved. Speculation on this point is more of physiological than clinical interest.

Harley-street, W.

THE TREATMENT OF HYPERTROPHIC PHARYNGITIS AND LARYNGITIS BY THE EMANATIONS FROM THORIUM NITRATE.

By J. GORDON SHARP, M.D. EDIN.

IN October, 1902, I attended a man, aged 36 years, in an attack of left-sided pleurisy with a small amount of effusion which cleared up in six weeks' time without tapping and caused no subsequent trouble. During this period he had persistent cough and brought up small quantities of white frothy mucus. This was examined by a competent authority on one occasion but no tubercle bacillus could be found. The patient had only lost some four or five pounds in weight. Despite the negative bacteriological result the case was suggestive of tubercle. He spoke in a husky tone and complained of "weakness in his voice" when he talked much. This huskiness had existed for ten years, he said, and at the time of its onset he sought advice for the trouble at Guy's Hospital and stated that he was there treated with cod-liver oil emulsion but could give no more definite information on the subject.

As soon as the man was able to come to my house I examined the nose, pharynx, larynx, and neighbouring parts. There was nothing of note wrong in the nose but the soft palate and pillars were studded with yellowish tubercles in size rather less than the dimensions of a pin's head and set in a red mucous membrane. These tubercles were not enlarged lymph follicles but might have passed for the bodies seen in miliary tuberculosis of the pharynx. When the tongue was pulled forward the lingual tonsil was seen to be much hypertrophied. The back wall of the pharynx presented the usual appearances of a chronic inflammation and the epiglottis was red and enlarged. The interarytenoid region was infiltrated with dark red tissue and here also could be seen the same yellow bodies found on the soft palate. No ulcer was detected. The ventricular bands were swollen, extending so far towards the middle line as to prevent the vocal cords being seen. Here also were to be seen one or two yellow tubercles. As may be expected there was a chink between the true cords on account of the interarytenoid swelling. All the parts, including the interior of the larynx, were covered by a whitish, sticky secretion. To come to an exact diagnosis of the pathological condition was a matter of great difficulty.