ON
THE PATHOLOGICAL TRACES
OF
PULMONARY HÆMORRHAGE.

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THE relation of pulmonary hæmorrhage to pulmonary phthisis is a subject which has always, even from the time of Hippocrates, attracted the attention of physicians, and the possibility of a phthisis *ab hæmoptoe*, firmly held in old times, concisely and clearly urged by Morton and Hoffman, denied by Laennec, has again in modern days received fresh confirmation in the well-known opinions put forward by Niemeyer in his writings on pulmonary phthisis.

On searching the evidence brought forward to sustain or controvert this theory, it is a little surprising to find a remarkable paucity of pathological proof of the effects of hæmoptysis, notwithstanding the great frequency of such an occurrence in cases of phthisis; much reliance appears rather to have been put on the clinical histories and symptoms of cases.

Valuable, however, as such a method of investigating the

question may be taken to be, I am inclined to believe, after considerable experience in the clinical aspect of the matter, that it is from the pathological point of view that the solution of a most difficult and intricate problem must be hoped for; and it is with the object of supplementing the pathological evidence which bears upon the subject of pulmonary hæmorrhage, that the following results, obtained from the examination of many cases in which hæmoptysis was known to have occurred, and in some of which the hæmorrhage was the immediate cause of death, are put forward as some contributions to the elucidation of a question which is as interesting as it is complex.

The opportunities afforded in the pathological department of the Hospital for Consumption, at Brompton, are considerable; and I must here express my thanks to my colleagues, Dr. Pollock, and Dr. Douglas Powell, who have with most courteous readiness placed many cases under their care at my disposal.

The details of a few of the cases which have furnished material for the conclusions which are here given are supplied in the form of an appendix, as it seemed advisable not to obstruct the general results obtained from their consideration.

The first question that requires an answer is this:—What traces are most frequently found in the lungs after death which indicate a previous hæmorrhage? There are certain peculiar pathological conditions which are often to be found in cases in which there has been a history of hæmoptysis, and they occur in no other instances. These consist of well-defined circumscribed nodules of an oval or round form, in colour varying, according to their age, from blood-red to yellowish-red and white, in size varying from $\frac{1}{8}$ of an inch to an inch, but generally assuming the size of a threepenny piece, say half an inch in diameter, marked in their centre with the openings of two or more bronchioles, which are usually surrounded or spotted with pigment; they have a slightly granular surface when red, but when white are firm, tough, unyielding, and hard (Plate XV, fig. 2 and 3). These have

usually been held to be simply nodules of caseous pneumonia, and are recognised as such in the cases recorded by Niemeyer, Bäumlér, Weber, and Powell. If I do not misunderstand the opinions of these observers, they consider these nodules to be the caseous results of a pneumonia, due to the irritation of blood remaining behind in the alveoli acting as a foreign body.

The opinion of Hoffman (1740), quoted by Niemeyer in his lectures on phthisis (pp. 30), that blood is extravasated into the alveoli and *stasi concepta putrescit*, is approved and appropriated by Niemeyer in the following words: "The blood which remained behind in the bronchi and alveoli has led to a pneumonia undergoing cheesy transformation, the retained blood and the products of inflammation afterwards breaking down, as is plainly described in the quotation from Hoffmann" (pp. 32). This appears also to be the opinion of Dr. Bäumlér, from his paper in the 'Clinical Transactions,' in which he says (vol. ii, pp. 83), quoting Niemeyer's views which he supports, "Part of the blood which is not expectorated, and which has coagulated in the smaller bronchi and air vesicles, acting by its decomposition as an irritant, and causing inflammation of the lung-tissue. This opinion appears also to be shared by Dr. Weber, although without complete acceptance of all the opinions of Niemeyer.

Although these opinions are in some measure correct, yet they do not represent the exact state of the case, these nodules not being the caseous products of a pneumonia. They differ in the following points from a pneumonic product: they are round, well-defined, and circumscribed, whereas pneumonic products are always of irregular shape, in patches, and generally shading off into the tissue near; they are tough and indurated, and show a marked tendency to separate around their circumference from the lung-tissue which surrounds them, a property which distinguishes them conclusively from pneumonic products. They are not due to the results of a hæmorrhage *in situ*, for they may be found in the lung opposite to that in which the wounded vessel or aneurism has been found.

Nor are they due to the simple gravitation of blood dribbling downwards, for they are found in the apex of the lung as well as the base. To what then do they owe their presence? The answer to this question is obtained from a consideration of the preferential regions which these nodules inhabit.

The upper lobe is sometimes a seat of these nodules, which when situated here run into each other and occasionally form rather large masses. In considering the special reasons in each case which influence the respective position of the nodules, it is necessary to consider also the condition of the lungs, inasmuch as the upper lobe is so generally affected with previous disease, either as excavated or adherent from old pleurisy. The base is another favourite locality, for they are often found close to the periphery of the diaphragmatic surface and far from being found in the most dependent part, the posterior border, they may be found limited to a region of narrow width at a place corresponding to the arched part of the diaphragm. There is also another region in which they may be found, and that is in the anterior axillary border close to the periphery, in the region of the nipple, or between the third and fifth ribs. These three regions are notably those parts of the lung in which inspiration produces the greatest expansion of the lungs, and which are most called upon in any great inspiratory effort. These nodules are sometimes found curiously isolated in the regions just mentioned, and it seems, therefore, that they owe their presence there to the inspiratory force expanding the lung in these parts, and to the force of the atmosphere driving home some substance which it meets in the bronchial tubes.

It is quite evident from the microscopical examination of sections and from the microscopical appearances that it is blood which is forced into the alveoli, and from the frequency with which both lungs (in cases of fatal hæmoptysis) show the presence of clotted blood in the tubes, the only conclusion that appears a solution of the problem is this, that in cases of profuse hæmoptysis, the welling up of the blood causes a dyspnœa, which results in forced inspira-

tory efforts; the blood which clots in the tubes is driven backwards and forced into alveoli in the apex, the base, and the axillary region; the lobules are distended with the clot, which finally loses colour and becomes hard, white, and tough, closely resembling the hard white infarcts which are found so often in the spleen, and like them may be found unaltered and inert some time after their first formation.

The microscopical examination of these nodules is a little difficult, owing to the fact that when by reason of their age they have assumed the white and tough condition, they lose a great deal of their early appearance, and the different elements become confused. But even in this stage the markings of the alveolar walls may be made out, and also the

FIG. 1.

presence within them of an opaque, homogeneous, fibrinous material.

The tissue in the surrounding region (see fig. 1) shows marked evidence of irritation, the result being similar to that which obtains in the irritation of pulmonary catarrh, with general thickening and increase of the connective tissue

elements. An examination of the neighbourhood of these nodules shows more decidedly and conclusively what has taken place. On examining the section with a lens small elevated nodules may be found, which on further investigation prove to be solitary alveoli or bronchioles plugged with blood globules firmly packed into the lumina, and in some cases distending them. The irritation which their presence excites is very marked, and occasionally leads to considerable thickening of the pleura if the nodule be situated close under the pleura, and always of the interlobular tissue which surrounds the nodules.

From these observations it appears that these nodules are due to the forcible impaction of clotted blood driven from a distance into the bronchi and alveoli, and that a certain amount of irritation is produced by the blood; but it must be remembered that an irritation of this kind need only result in a fibrosis of the lung, and the fibrinous mass may, and does remain often, quiescent and inert. This, however, is not always the case, and the next question is, what alterations these nodules undergo?

Allusion has already been made to the fact that they show a strong tendency to drag away from the surrounding tissue, and this especially happens when there is any strain on the respiratory tract from previous disease, or a tendency to congestive softening of the pulmonary tissue.

In two cases a remarkable condition of things resulted from this tendency (Cases 2, 3). Twice I have found the thickening envelope of the nodules lying loose in a cavity, resembling the sac of a pulmonary aneurism; in one case this free capsule was found in the fourth intercostal space and in the other quite at the base of the lung (see fig. 2). The contents of the capsule had softened at the same time and been ejected. The process of necrosis which these nodules undergo may take two forms. They may undergo a gradual process of erosion, that is, the nodule may be eaten away bit by bit, nibbled as it were by a special corrosive poison, or they may undergo a general liquefaction which involves the whole mass equally.

In the first case, that of erosion, the nodules are found

FIG. 2.

remaining *in situ*, but with bits eaten out of them corresponding to an amount of staining derived from the presence of a sanious fluid arising from sloughing of the lung tissue around them or introduced from a distant cavity (Cases 2, 3).

It is very striking to see this staining of the white nodule with the claret-coloured sanious fluid, and to note the corresponding erosion which takes place, a fact bearing strongly upon the question of the auto-infection of the lung, especially in cases of destructive sloughing of the lung tissue in which this sanious fluid is secreted often in large quantities, and of most destructive virulence.

The second process of necrosis is that of general liquefaction. When this takes place the nodule softens in the centre and gradually assumes a straw-coloured, glairy fluidity, which might be (and often is) mistaken for a purulent liquid, but the colour is too yellow and pus globules are absent (Cases

3, 5). This liquefaction is often local; for instance, it occurs at the apex (upper lobe), while the nodules in the lower lobe remain hard.

From the facts here recorded relating to the alteration of these nodules it must be admitted that hæmorrhage may result in excavation of the lung, or, in other words, they establish the possibility of a phthisis ab hæmoptoe.

2. There is another pathological condition which I understand as a condition arising from hæmorrhage *in situ*, not as a result of blood transferred from a distant locality. This is a condition which occurs less frequently than that just described (Pl. XVI); it consists of irregular blackened patches, sometimes of considerable size, as much as two inches and a half across, which may be found in the upper part of the lower lobes, or irregularly placed in the upper lobe. There appears to be no special locality for them (Cases 4, 5). They are formed of calcareous matter, loosely coherent, mingled with the black pigment of old blood, and surrounded with a defined but irregular envelope of some thickness, which is deeply pigmented with the same black granules. Occasionally the yellow colouring matter, which is often found in old blood clots, is found scattered about, indicating the original source of formation.

These patches are apparently due to hæmorrhage of some violence, producing laceration and contusion of the lung-substance, and thus leading to a secondary condition of disintegration of blood and lung-tissue, and the ultimate formation of these mortar-like patches.

In some cases, when they are found near the periphery of the lung, they assume a circular form, and ultimately, by the traction of the lung, they make their way into a bronchus and clear out from their nidus, leaving a remarkably clean and well-defined cavity. An example of this is given in Case 4, in which such a cavity had been formed, the proof of its previous condition being the pigmented state of the peripheral portion (Case 4).

With regard to the possibility of an infection of secondary tubercle following upon this hæmorrhagic condition, that

depends upon the point of view from which the subject is considered. If simply the presence of secondary tubercle is looked for, it might be admitted that it may occur as an ultimate result of such disease, but if the question be asked in this form, namely, whether the secondary tubercle arises simply from the blood products, then the answer must be that up to this time no case has presented itself in which the blood products have not undergone a secondary process, and that it requires more evidence to determine this point in a satisfactory manner.¹

The conclusions which may be deduced from a consideration of the cases which have come under my notice are these:

That in cases of severe hæmoptysis portions of the blood are driven into the alveoli, which they occupy finally in the form of fibrinous nodules, setting up some irritation in their vicinity.

That in cases of capillary hæmorrhage, with laceration of the pulmonary tissue, the resultant effect produces a calcareous mass, sometimes of considerable size.

That under special circumstances cavities may be formed by the liquefaction of the hæmorrhagic nodules in the first instance, or by the removal of the calcareous masses in the second.

That whether secondary tubercle can result from inhaled blood without the intervention of secondary processes introducing a new septic condition is a point that requires further evidence before it can be accepted.

¹ Since the above was written a fatal case of hæmoptysis in a woman has come under notice. The lung showed the presence of hæmorrhagic nodules of large size in the region of the 4th rib, at the first axillary border, and also in the anterior inferior margin, where they were found as large as peas. None were found where they are so frequently found in man, in the middle of the lowest surface of the lung.

APPENDIX OF CASES.

CASE 1.—James S—, æt. 28, seaman, admitted October 16th, 1877, under the care of Dr. Powell.

Family predisposition.—Father died of phthisis at forty years of age. Three brothers of the father died of phthisis.

Personal history.—Erysipelas of leg following an injury in 1869. Suppurating bubo in 1872.

Present illness.—Caught cold in September, 1876, which was followed by cough. Expectoration slight. In January, 1877, had an attack of hæmoptysis. Very copious for three days, chiefly in large clots. Has been unable to work for thirteen months. Much emaciation. Has lost two stone. Pain occasionally severe, with cough.

Symptoms on admission.—Cough severe at night. Expectoration stained with blood. Dyspnœa on exertion. Height 5 ft. 4½ in., weight 7 st. 12 lbs.

During November the expectoration was blood-tinged.

On the 14th November he was seized with two or three epileptic convulsions, which ended fatally on the 15th.

Synopsis.—Hereditary phthisis. Hæmoptysis, preceded by cough, in January. Death ten months afterwards.

Post-mortem appearances.—Both lungs were much congested and blood stained. The bronchial tubes were also congested and granular looking, the vessels being well marked and in a condition of hyperæmia.

Left lung.—The upper lobe was riddled with small excavations, generally of the size of a filbert, which were lined with yellow membrane and secreted yellow puriform matter. The rest of the lung was much congested. In the axillary anterior border of the lung, on a level with the root of the lung, were found one or two round curdy masses, situated close to the periphery, softening in the centre and slightly blood stained—that is, there was a slight pink stain in the centre where liquefaction was going on, and which appeared to be due to extraneous sanious matter imported into these nodules through the bronchioles. Near the root were one or two

groups of racemose tubercles. Through the rest of the lower part of the lung were disseminated some separate and irregularly formed small masses of fibrin imbedded in alveoli.

Right lung.—There was a ragged cavity of considerable size in the axillary region of the upper lobe. The rest of the lung was much congested and blood-stained. A considerable portion, viz. a band of two inches in width, of the peripheral portion of the lower lobe was studded with circular, curdy, fibrinous nodules, of a white colour, hard and unyielding, with openings of one or more bronchioles containing black pigment around these tubes. Some of them were hard and unaltered, but many were tinged with a pink sanious fluid, possibly derived from the ragged cavity above-mentioned, and gradually softening from its influence. Some of these nodules showed a distinct tendency to separate, there being an interval or chink between the circumference of the nodule and the lung tissue. There were also recent patches of inhaled blood in the same part of the lung which had not lost its red colour, and some in the middle part of the upper lobe.

In this case no aneurysm and no wounded vessel could be found, but from the general congestion and blood-staining of the pulmonary tissue and the bronchial tissue it was concluded that the hæmorrhage had probably resulted from capillary rupture.

CASE 2.—John M—, æt. 34; police constable at the House of Commons.

Family history.—Two brothers died of phthisis.

Personal history.—Had suffered from pleurisy in the left side when he was twenty years of age. In 1871 he began to suffer from winter cough. In November, 1873, he first suffered from slight hæmoptysis.

He first came under my notice as an out-patient in October, 1874, and I then found the signs of cavity situated between the third and fifth ribs, on the left side, in the axillary region. In December he had another attack of hæmor-

rhage. In July, 1875, he had another attack of hæmorrhage, which was very severe. He lost more than a pint of blood. There was another return of hæmorrhage, to the amount again of a pint, in August, and he continued under my treatment until the end of April, 1876.

He was admitted into the Hospital, under Dr. Powell, in November, 1876. He gained weight, had no recurrence of hæmorrhage, and was discharged improved on the 26th February, 1877. He continued to improve up to the 28th of August, when he had a sudden copious hæmoptysis without exertion or violent cough. This recurred again the following evening (after walking up stairs) to the amount of two pints.

He was readmitted into the hospital November 6th, 1877, and on the 13th was out walking in the grounds, when he was suddenly seized with a profuse hæmorrhage, which proved fatal.

Synopsis.—Hereditary phthisis. Old cavity of four years standing. Frequently recurring attacks of profuse hæmorrhage for two years.

Section cadaveris.—Body blanched and thin. Left lung very much retracted, universally and firmly adherent, so that it was removed with much difficulty. It was very much dwindled. There was a cavity at the apex, about one inch in diameter, and two and a half inches long. There was a larger cavity (2×2) situated in the axillary region between the third and fifth ribs. The rest of the lung was hard and compressed. The bronchial tubes were large and dilated; they contained some recent clots of blood.

About the centre of the pericardial surface of the lung was a cavity the size of a filbert, which was filled with coagulum of blood, and in this was a ragged aneurysmal sac which had burst into the cavity. Above and behind this were found two or three curdy oval masses three fourths of an inch in diameter, white, but pigmented with black in the centre, which were softened and eroded at the upper portions, and below were undercut, so as to separate from the surrounding pulmonary tissue.

Right lung.—Very adherent at the apex behind. It was generally much distended and expanded. No active tubercular matter was found, but some old puckered tubercle at the apex. Under the fourth intercostal space in the anterior axillary line a cavity was found; on cutting into which there appeared a round yellow bag or capsule lying loose and empty in the cavity. There was no blood nor purulent matter in the cavity. The sac would have fitted the tip of the little finger, was rough and ragged inside and out; it was evidently the remains of a curdy mass, similar to those in the other lung, which had softened, emptied itself, and separated from the surrounding lung.

At the lower peripheral margin of the lower lobe were a number of small, round, white curdy nodules, half an inch in diameter, with openings of bronchioles in their centre. Some of these were slightly stained with pink and were beginning to soften. The rest of the lower lobe of the lung was in a condition of intense congestion.

CASE 3.—H. G. C—, æt. 22, single, billiard marker, admitted first into the hospital May 8th, 1877, under the care of Dr. Powell.

Family predisposition.—Nil.

Personal history.—Diseases of childhood. In 1874 sudden hæmoptysis of bright-red blood on stooping sharply. Had noticed for some months previously a slight dry cough. Lost 2 st. last year. Slight streaky hæmoptysis, once or twice amounting to 1 oz. Weight in May, 8 st.; Nov., 6 st. 12 lbs.

May 16th.—Hæmoptysis (1 oz.) at night. Temp. 99° F.

June 14th.—Copious hæmoptysis.

28th.—Hæmoptysis, which continued off and on during his stay in the hospital.

Sept. 18th.—Hæmoptysis 5 to 6 oz.

Nov. 20th.—Hæmoptysis sudden, 13 oz.

Dec. 1st.—Hæmoptysis about 2 oz. From this time he gradually lost strength, the pulse being rapid and very small.

He died on the 27th Dec., having been delirious a few hours before.

Synopsis.—No history of inherited disease. Hæmoptysis three years before death. Attacks of hæmorrhage frequent, and profuse in amount occasionally.

Sectio cadaveris (Dec. 28th, 1877).—Body emaciated; blanched.

Right lung.—Adherent behind and in front at the apex and base; adhesions firm. A large cavity full of sanious fluid occupied the whole upper lobe. The floor of the cavity was soft and flaccid, and two or three small rounded chambers were found communicating with it.

The ends of two small vessels which traversed the floor of the cavity were found corked at their mouths by a clot of blood, and which had probably been lately pervious, but were found closed a little below the ends. The lining membrane of this cavity was of a dark purple colour with a lurid congestion. Below this cavity were a number of small, well-defined, circumscribed, oval cavities, generally about the size of a small nut, that is, half an inch in diameter, closely packed together, surrounded with flaccid tissue, and floored with reddened tissue secreting sanious fluid. These cavities contained the remains of curdy nodules, which had been eroded in places by the sanious matter, and the remains of these masses remained *in situ*; pigmented bronchioles were found in the centres of these nodules. Towards the base these cavities were in some instances found to contain a glairy, yellow, puriform fluid. The lower border of this lung was firmly adherent to the diaphragm, and the pleura thickened.

Near the periphery was found a cavity as big as a walnut, which contained, lying free within it, a capsule an inch long filled with recently clotted blood, which could be shelled out of it. On making sections of this capsule it was found to be formed by interlobular tissue, which was much thickened, and inside the sac were found remains of lung tissue marked with blood and filled in the alveoli with fibrinous matter. Further section of the lung in the immediate neighbourhood of this capsule showed many little rounded masses varying

from $\frac{1}{8}$ to $\frac{3}{8}$ of an inch, with the openings of bronchioles in their centre, which were pigmented.

The glands throughout were pigmented. Some of them were washed free from blood stain, and pounded in a mortar and digested with strong nitric acid, and a distinct blue tint was obtained on the addition of ferrocyanide of potassium. At the upper part of the middle lobe below the main bronchus was found an aneurism of the pulmonary artery, as large as a pea, hanging loose by an attenuated bit of artery into the small cavity, which was of the size of a nut. On careful examination the pedicle of the aneurism was found closed by a small adherent clot of fibrine, while the aneurismal sac was full of hardening, but coloured clot.

Left lung was in a much less advanced stage of disease. It was enlarged and distended. The upper lobe was scooped out into small cavities about the size of a small filbert, which showed yellow matter close to the circumference. These cavities were formed in the same manner as those of the middle lobe of the right lung from the evacuation of the fibrinous material. The lower lobe was much congested, and of a lurid red colour. Well-defined miliary tubercle was found chiefly in the axillary region. In the middle of this axillary region were found numerous round, hard nodules, with central pigment, the size of a pea, scattered about close to the periphery. Also quite at the base, and close to the diaphragmatic surface, were found a number of hard, fibrinous nodules of a similar character.

CASE 4.—John W—, æt. 35, hospital porter, admitted Oct. 9th, under Dr. Pollock.

Hereditary predisposition.—Nil.

Personal history.—Rheumatic fever in 1870. For four months before admission had suffered from cough with hæmoptysis. Hæmorrhage of black blood on two occasions to the amount of half a pint, the last time five weeks before entering the hospital. Considerable loss of weight. Had suffered from diarrhœa.

On Nov. 20th he began to suffer from ascites, and the

urine was found to contain a large quantity of albumen, sp. gr. 1015, 50 ounces in quantity.

He continued to suffer from diarrhœa, and occasionally vomiting during the rest of his lifetime, but there was no return of hæmoptysis during the time he was under observation. He died on Dec. 28th.

Synopsis.—No hereditary predisposition. Hæmoptysis to the amount of half a pint twice; the last time being nearly four months before death.

Sectio cadaveris.—Dec. 22nd.—A small depressed cicatrix of old sore on the penis.

Right lung very adherent, adhesions universal and thick. The pulmonary tissue was generally very much congested, soft, and dark, containing a considerable quantity of blood. In the middle of the upper lobe and middle lobe were found two irregular patches of mortar-like matter, and surrounded by thick, black, pigmented tissue; the lower patch having a number of small, rounded, mortar-like masses, the size of mustard seed, outlying and similarly surrounded by thickened pigmented tissue. No tubercle was detected.

Left lung.—Adherent behind, with long fibrinous bands of attachment in front. Three irregular patches of mortar-like matter, similar to those in the right lung, were found occupying the lower and posterior portion of the upper lobe. A circular well-defined cavity, one inch in diameter, was found in the upper lobe. This cavity was close to the periphery, and was pigmented at its margin; the lining membrane had here contracted and become depressed; it communicated with a bronchial tube of some size (quarter of an inch). It was evidently the result of a complete clearing out of a mortar patch similar to those already described. These mortar patches were found to contain small masses of bright yellow pigment, evidently colouring matter from old blood. The lower part of the lower lobe was found full of semi-decoloured blood surrounded with blood stain and congestion.

Liver was covered with recent lymph, and was found in a very advanced amyloid condition. The peritonæum was

also covered with thick fibrinous lymph, and the kidneys and intestines were found to be amyloid. The large intestine was studded with a number of old thickened ulcers, which were probably of dysenteric origin. (Plate XV, fig. 1, and Plate XVI.)

CASE 5.—Charles B—, æt. 29, groom, admitted October 18th, 1877, under Dr. Powell.

Family predisposition.—Nil. Father rheumatic.

Personal history.—In 1864 he began to suffer from cough and hæmoptysis. He was admitted into the hospital in 1865, and left much improved. Went to Malta and India as an officer's servant until 1870. He had ague. In the winter of 1873 he was attacked with very copious hæmoptysis for several days without exertion. This returned five months before admission, but it did not recur again.

When admitted he was much emaciated; suffered severely from cough, expectoration being very copious. Towards the end of November he was attacked with epigastric pain and vomiting, and ultimately he became jaundiced, which disease proved fatal on the 9th of January, 1878.

Synopsis.—No hereditary predisposition. Copious hæmoptysis in 1873, and again eight months before death.

Sectio cadaveris.—Both lungs found to be very adherent at the upper lobe behind.

Left lung large. The upper lobe contained two cavities as big as a walnut (an inch and a half across), filled with glairy, yellow, puriform fluid. The tissue surrounding these cavities was found deeply pigmented and thickened. In the neighbourhood at the back were found some smaller, well-defined, oval cavities, filled with the same yellow matter. Below in the lower lobe were found two or three long irregular patches surrounded with pigmented tissue, and with ragged pigmented tissue in their centre, with here and there a curdy fibrinous mass adherent. These patches were about two inches long, and ran across the lung from side to side. Along the periphery of the lung, in the region of the anterior axillary portion, ran a band of pulmonary tissue half an

inch in depth, which was formed of small terminal bronchioles and alveoli choked with white, fibrinous material, apparently the result of blood inhaled into this portion. The lung-tissue at base was much congested.

Right lung.—Much the same appearance was found in the right lung, only it was less affected; the back of the apex of the upper lobe was riddled with small oval, circumscribed cavities, filled with yellow matter liquid and glairy. A similar cavity to that in the left lung was also found in this part of the lung, and irregular pigmented patches. At the upper part of the lower lobe were small rounded cavities, and a few racemose groups of tubercle tissue much congested generally.

The liver was found in an advanced condition of cirrhosis. The kidneys were small and hard.

DESCRIPTION OF PLATES XV AND XVI.

Pathological Traces of Pulmonary Hæmorrhage (Reg. E. Thompson, M.D.).

PLATE XV.

FIG. 1.—Inhaled blood: early condition showing blood clots losing colour; base of left lung. Case 4.

FIGS. 2, 3.—Inhaled blood: late condition; base of lung. Case 3.

PLATE XVI.

Mortar-like patches, result of old hæmorrhage; upper part of left lung. Case 4.

Fig. 1.

Fig. 2.

Fig. 3.