A CASE
OF
CALCAREOUS METAMORPHOSIS OF THE
TRACHEAL LYMPHATIC GLANDS

FORMATION OF MEDIASTINAL ABSCESS AND ESTABLISHMENT
OF BRONCHIAL FISTULA; SUBSEQUENT PERFORATION
OF THE WALL OF THE ARCH OF THE AORTA; LONG-
CONTINUED REPEATED HÆMOPTYSIS;
DEATH FROM HEMIPLEGIA

BY
SIDNEY PHILLIPS, M.D.

PHYSICIAN TO ST. MARY'S HOSPITAL; SENIOR PHYSICIAN TO THE
LONDON FEVER HOSPITAL

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P. M—, aged 26 years, had always been delicate. His father and father's family were excessively gouty; his mother suffered from rheumatism. He had had no illnesses of any note, but at about the age of eighteen he suffered from frequent profuse epistaxis, for which he went on a sea voyage. He was very subject to attacks of tonsillitis. During the year 1885 he experienced a good deal of pain in the elbows, wrists, and finger-joints; the larger of these

joints contained little fluid, but on movement very rough grating could be felt within them. He went as a cadet to the Royal Military College at Sandhurst in 1886, and during the summer vacation of 1887 he went to Nuremberg; he complained much of the foul smells there, and was rendered quite ill by them: this was in August. He returned to Sandhurst fairly well at the end of that month, and while riding in the school there on September the 5th, 1887, he was suddenly attacked with very acute pain on the right side of the chest; this became more and more severe, and he consulted me in London on September the 11th. There was then intense pain round the right side of the thorax, almost limited to the third intercostal space, and with it extreme hyperæsthesia of the surface of this side of the chest as far down as the sixth rib; no tenderness existed over the spine; the temperature was normal; his general condition seemed good, and I could find no evidences of disease of any part of the lungs or the heart. I ordered him salicylates internally, and locally anodyne applications.

He returned to Sandhurst, but the pain grew more and more intense, confining him to bed, and morphia injections had to be administered by Brigade-Surgeon Clark, who attended him then and during the subsequent illness.

On September the 16th—eleven days after the pain had commenced—he had a severe rigor, and the temperature rose to 102° .

On the next day I saw him with Dr. Clark; his tongue was coated, his liver a little enlarged; the pulse was full and bounding, the lungs appeared to be absolutely healthy; there was a suspicion of a soft systolic murmur over the aortic cartilage. Beyond this there were no indications of disease anywhere, but his temperature was $104\cdot6^{\circ}$, and the pain was very severe.

During the next five days he improved a little, the temperature gradually falling to normal, and the pain lessening, but on September the 22nd—six days after the first rigor—rigors recurred, and again on each of the six

succeeding days, each time with a rise of temperature to 104° or more. Between the rigors the temperature fell to 99° .

On the 27th September I saw him again: he had just had a rigor with a temperature of 105° , accompanied by agonising pain in the fifth, sixth, and seventh left intercostal spaces just outside the cardiac area. The pain was intense, and he screamed with agony. There was a great reluctance to inspire, and he kept the painful side of the chest almost motionless, only a faint wheeze being audible as a breath-sound. There was now tenderness over the spines of the fourth and fifth dorsal vertebræ. There was a faint friction crackle just below the lower angle of the left scapula. The pulse was 145 a minute, very feeble. He had taken food well, but had lost flesh greatly. Morphia had to be repeatedly administered, and he took quinine at times.

On the 29th September he was seen by Sir William Broadbent, with Dr. Clark and myself; he then looked better, the pulse was 84 a minute, and the temperature 99° . No sign of disease anywhere could be detected, except the faint friction sound over a spot in the left back, and air did not enter so well on this side as on the right, though on deep inspiration it entered more freely, suggesting some pressure upon the left bronchus. There was no recurrence of rigors after the 27th September, but the profuse sweats and daily rises of temperature continued, with pain and loss of flesh and strength. The friction sound extended in area, and by the 8th October it was audible over the left back as high as the spine of the scapula, and round the axilla; cough was slight; heart-sounds normal.

About the 15th October there became superadded to his troubles a most incessant, violent, and exhausting spasmodic dry cough, preventing rest or sleep. Laryngoscopic examination showed the larynx to be normal. The pyrexia now again increased, reaching 103.4° on October the 19th, when he was seen with us for a second time by

Sir W. Broadbent. The friction sound was now audible all over the left back, and the cough kept him constantly sitting up in bed, and he was exhausted to an extreme degree.

On October 28th the cough became more violent than ever; it was apparently due to some irritation of the laryngeal nerves. On this day there suddenly came on signs of rapid copious effusion into the left pleura. The apex of the heart became at once pushed to the right, and could be felt well outside the right nipple line; its action was greatly disturbed. Simultaneously there developed loud systolic murmurs over the base of the heart. I saw him that afternoon, and the orthopnoea being great we at once aspirated the left chest, withdrawing 56 ounces of clear fluid. No attempt was made to withdraw all the fluid in the chest. The aspiration gave considerable relief, respirations falling from 38 to 24 per minute, and the pulse improving.

By October 30th the fluid had re-accumulated, the respirations were 40, and the pulse 140 a minute. His distress being great, 20 ounces more fluid were removed by aspiration; more was not removed as the patient was under chloroform, and his condition was very critical; after this the respirations fell to 22 a minute, and he was much easier.

On the next day the temperature was only 101.2° , the respirations 32, and the pulse 140 a minute. During the night of November 1st he had an attack of syncope with complete unconsciousness and flickering pulse; this lasted three hours. He was thought to be dying, and his relatives were summoned; but he was treated by Dr. Clark with subcutaneous injections of ether, and gradually recovered consciousness.

On the next morning 10 ounces more fluid were removed by Dr. Clark with the aspirator. The temperature was 102° , and the cough incessant.

The same afternoon, November 2nd, he expectorated after violent cough about one drachm of pus slightly

tinged with blood and mixed with aërated mucus; this was the first expectoration of any sort throughout the illness.

On the next day the cough had become a little less, but there was still great pain over the third and fourth left intercostal spaces.

I saw him on that afternoon, and there was a great change in the physical signs on the left side of the chest; there were still all the signs of pleuritic effusion over the back, but in front of the chest there was now audible loud tubular breathing. This extended round to the axilla, and there were sibilant râles of a resounding character, as if in some large cavity; there was dulness on percussion over the area of tubular breathing; it appeared probable there was some air in the pleural cavity, but the physical signs were anomalous. The heart's apex was now just below the right nipple.

Although the cough had somewhat decreased in intensity the patient continued to lose ground, with daily rises of temperature to 102° , and considerable dyspnœa and pain.

On November 6th, the dyspnœa having rather increased, Dr. Clark removed eighteen more ounces of clear fluid by the aspirator.

On November 9th the temperature was a little lower, and the pain and cough were less; there seemed to be less serous effusion in the pleura, but there was a peculiar "crack-pot" sound on percussion just below the inferior angle of the scapula; the tubular breathing in front of the scapula had now given place to loud amphoric breathing, but no gong sound or succussion sounds were obtainable: the heart's apex was still to the right of the nipple line, and the murmurs, which had for a time disappeared, returned—a loud systolic murmur behind the sternum at the level of its junction with the second and third rib cartilages, and in the second and third intercostal spaces; there was also a very loud bruit at the angle of the left scapula: the patient's breathing was again greatly embarrassed, and his pulse very rapid and compressible.

On the next day, the 10th November, he had another syncopal attack of ten minutes' duration.

On November the 11th the temperature was $103\cdot6^{\circ}$, and aspiration in the sixth space failed to remove any fluid; on the same night he had another very severe syncopal attack.

The patient had now for a period of two months suffered from terrible pain, from protracted fever, and latterly from a most exhausting cough; his rest had been greatly interfered with; he was emaciated to a skeleton, and becoming weaker daily; he had been aspirated eleven times, and though the serous effusion in his chest did not re-accumulate to any extent, the heart remained displaced, the fever continued, and syncopal attacks were becoming more frequent,—he had had two in the last thirty-six hours; he was at times delirious, and it appeared certain that he might die at any moment from syncope, or would gradually sink from exhaustion unless relieved by treatment.

Mr. Godlee was therefore asked to see him, with the view of making a free incision into the chest wall.

On November the 12th A.C.E. mixture was administered by Surgeon-Captain Mansell, who had aided us in the care of the patient throughout the illness, and Mr. Godlee made an incision into the left axilla in the fifth intercostal space. About ten ounces of clear fluid ran out; the finger passed into the wound entered a very large cavity, the diaphragm being felt below, and the lung being greatly collapsed and retracted, with sticky lymph upon its surface. A very large drainage-tube was inserted and the wound dressed with salicylic wool.

After this improvement commenced, the temperature falling to $99\cdot5^{\circ}$ by the 30th November; the cavity, explored with a bougie, appeared to be of enormous size, and discharged very freely; at times drainage was difficult, and on October the 2nd the temperature rose to $102\cdot2^{\circ}$, the pulse-rate to 140 a minute, and the patient became almost delirious. The incision was enlarged a

little, and a good flow of pus resulted. The cough and pain were now but slight, but there was still some tenderness over the fifth and sixth dorsal spinous processes. By the end of December he was able to be got up, and he travelled to Bournemouth in an invalid carriage on January the 3rd, 1888, under the superintendence of Dr. Clark, to whose unremitting attentions his recovery had been mainly due.

At Bournemouth he was under the care of Dr. E. Hyla Greves, with whom I saw him on several occasions. The cavity continued to gradually decrease in size, but attempts to discontinue the drainage-tube were always followed by difficulties, and it had to be replaced. He became strong enough to walk a little, and came to London on March 20th, 1888; improvement continued, and he went to the country again at the end of May for a short stay, but quickly returned with pyrexia and retention of pus in the cavity; good drainage was soon re-established. About this time, while exploring the cavity with a long bougie, I once felt at the tip of it something feeling rough, like a small surface of carious bone. It appeared far back near the spinal column, but I was never able to hit upon it again, and it was not until the necropsy that this rough surface was shown to be one of the calcareous mediastinal glands.

Mr. Godlee saw the patient with me once or twice, and as the opening could not be closed, an anæsthetic was administered, and Mr. Godlee removed two or three inches of the fifth and sixth ribs, which had become united together by two cross-pieces of bone, forming a complete osseous ring around the drainage-tube: after this the chest wall fell in well, and by the middle of June the drainage-tube had been discontinued, and the opening in the chest completely closed—six and a half months after the incision into pleural cavity.

This terminates what I may call the first chapter of the patient's illness.

Then followed a period of eighteen months of quiescence

of symptoms, though never of perfect health. The patient gained flesh and strength, and was able to enjoy himself, but he always suffered from what he termed "asthma," a wheeziness which became very marked in damp or foggy weather; the voice, too, at times was a little husky, but it had not lost its power, and he was able to sing; there was no cough, dyspnœa, expectoration, or fever, and the only physical signs of disease within the chest during the beginning of this period were those due to the old pleurisy.

In November, 1888, he went to Australia, and came back early in 1889 much stronger, but there was still a little wheeziness and huskiness of voice. Shortly after he came home—in June, 1889, he brought up one day a little semi-purulent pink-stained matter, much resembling crushed strawberries and cream; there were no tubercle bacilli found in it. Below the right clavicle I noted on the next day that there was a slight rise of percussion pitch and a little weakening of air entry.

In November, 1889, he went to Cairo for the winter; he continued very well there till 18th January of 1890. On that day commenced the third epoch of the disease.

He had on the 18th January, 1890, got wet while out on a shooting expedition, and on the next day he suddenly, without effort or cough, brought up about seven ounces of bright red blood. He then came under the care of Dr. H. Sandwith of Cairo, who kindly communicated to me from time to time the progress of the case. The hæmoptysis recurred again and again at intervals of a few days; the longest interval was three weeks, and then it came on immediately after he had been carried to the roof of the hotel for fresh air; any exertion or a fit of laughter would bring on the hæmorrhage. Dr. Sandwith found over the apex of the right lung some wasting of the pectoral muscles, slight deficient percussion resonance, and increased vocal resonance, with an occasional moist sound.

In the middle of April, being somewhat better, he was brought homewards by sea to Marseilles, and thence by

rail to Cannes ; on the sea journey he had several attacks of hæmorrhage, and had to stop at Cannes, where he was seen by Dr. Battersby and Dr. Bright.

He arrived in London on May 21st, and came again under my care.

I found the same slight physical signs as Dr. Sandwith had noted over the apex of the right lung, the most marked abnormality being the increase of vocal resonance over the supra-spinous fossa. Tenderness still existed over the sixth dorsal spine.

The attacks of hæmorrhage continued ; the blood was always bright red, unmixed with air, sometimes partly clotted ; it sometimes came up in a sudden gulp when he was walking in the street, at other times it woke him up from sleep ; after it had been gulped up there was an end of it for the time, and no trace of blood came up till the next free hæmoptysis. The usual quantity of blood ejected was seven or eight ounces, sometimes as much as ten ounces, and at one time the bleeding was much greater over two successive days. The blood was repeatedly examined, but no tubercle bacilli could be found in it by Mr. Watson Cheyne or others. The hæmorrhage recurred at irregular intervals, on an average twice a week. It did not seem to be determined by exertion, and though any very active exercise was forbidden, it was thought best to let the patient take daily walking exercise.

At times soreness about the larynx was complained of, but Sir Felix Semon, who examined him, found the larynx normal. There was no expectoration, except that in the intervals of the hæmorrhages he coughed up frequently tube-like casts of the smaller bronchial tubes ; they were always quite white and unstained by blood ; they often excited considerable cough on ejection. Microscopically I found these casts to be tubular, non-laminated, and of homogeneous, apparently fibrinous composition.

In May, 1890, while at Eastbourne under the care of Dr. Downes, he once brought up some small pieces of whitish pellicle, looking very like hydatid membrane, but

the microscope did not show any lamellated structure, and this never recurred.

The physical signs remained the same as I have described, but there were slight differences on different occasions when I examined him, the dulness sometimes quite disappearing, and occasionally distant weak tubular breathing was audible. During this period of the illness he was seen once again by Sir William Broadbent ; also by Sir Richard Quain, and by Dr. James Pollock and by Mr. Godlee.

For fifteen months the hæmorrhages continued ; he certainly lost on an average 16 ounces of blood a week, and in all the quantity lost must have been nine or ten gallons of blood. He recuperated after each hæmorrhage with extraordinary rapidity, and showed great spirit and courage in making the best of his troubles ; but after about a year it became obvious that he was failing to make good the repeated drain of blood ; he became anæmic and sleepless, with a hectic flush and transparency of skin, and the pulse was very soft and compressible : there was never any fever, and he always ate well.

In the winter of 1890 he went to Cannes again, but soon returned, the hæmorrhage having become more profuse.

On the 26th of March, after two days of very free bleeding, he became hemiplegic on the left side ; unconsciousness supervened, and he died in this condition on April 2nd, 1891.

The *post-mortem examination* made by Dr. L. Guthrie and myself showed that all the organs were healthy. Nothing abnormal was found in the heart or pericardium ; there was little to denote the previous existence of the opening in the left chest except fibrous adhesions of the lung. The apex of the right lung appeared slightly shrunken, and felt a little tougher than the apex of the left ; there were no tubercles anywhere in the body. In the tracheal bifurcation was an ovoid mass the size of a walnut, adherent by connective tissue to the bronchi and the trachea, and con-

sisting of a group of lymphatic glands which had become calcareous and brittle, but which were still distinct in outline. They were united together by tough areolar tissue, leaving a small irregular cavity in the centre of the mass they formed. On slitting up the air-tubes the larynx was healthy, and a small orifice was seen in the inner aspect of the right bronchus, half an inch from its commencement. A fine probe introduced through the orifice passed into the cavity in the centre of the calcareous mass; the under surface of the commencement of the transverse part of the arch of the aorta touched the upper part of the rough calcareous glands, and at this spot there was a small perforation through the wall of the aorta into its lumen; a probe could be passed through this orifice in the aorta into the centre of the gland mass, just as it could be from the opening in the bronchus; the cavity in the calcareous mass was thus in communication on the one side with the interior of the aortic arch, on the other side with the interior of the right bronchus; it was in this way that the hæmoptysis had occurred. The ascending aorta was extremely small in circumference, resembling that of a child,—no doubt from the diminished volume of blood in the body which resulted from the long-continued losses of blood. Two or three isolated bronchial glands had undergone cretaceous changes.

Remarks.—Briefly, the course of events in this case appears to have been—

I. Calcareous metamorphosis and matting together of a group of lymphatic glands, forming a rigid tumour attached to the tracheal bifurcation.

II. Acute inflammation and suppuration in the connective tissue of this tumour, forming an encysted abscess.

III. Perforation of the right bronchus by pus from this abscess, leaving a permanent bronchial fistula.

IV. Subsequent erosion and perforation of the wall of the arch of the aorta by pressure of the calcareous glands.

V. Recurrent hæmoptysis from the aorta into the empty abscess cavity, and thence through the bronchial fistula into the trachea.

VI. Death from hemiplegia, the result probably of thrombosis from the effect of the repeated hæmorrhages.

Whether or not this was the actual sequence of events, the calcareous changes in the glands, the suppuration and formation of a bronchial fistula, the perforation of the aorta, and the repeated hæmorrhages from it are beyond question.

It is to these points I wish to draw attention, but it would be impossible within the limits of this paper to make any attempt to review the literature bearing on the many interesting clinical and pathological conditions which presented themselves during the three and a half years' duration of the case.

Calcification of the Mediastinal Lymphatic Glands.

Calcareous bronchial glands are occasionally found at post-mortem examinations of old persons, or in those in whom exist old tubercular foci in the lungs. In the present instance, however, a whole group of glands underwent this calcareous metamorphosis, and became united together by the periglandular areolar tissue into a hard unyielding mass situate in the bifurcation of the trachea, and adherent to it by fibrous tissue. Medical literature affords but scanty information as to the causes which lead to calcareous changes in the lymphatic glands, but they are probably the same as lead to similar changes in the walls of arteries and of other tissues. The patient whose case I record this evening was young, and was not tubercular, but he had shown a great tendency to arthritis with rough grating in the joints; and it seems probable that the calcareous changes were due to the rheumatic or gouty diathesis, the deposit being determined in the lymphatic glands of the chest, owing to some idiosyncrasy on the part of the patient. The changes in the glands and

the formation of the tumour must have been very gradual, and no symptoms arose until acute inflammation chanced to be set up.

I have not found any record of a similar condition ; the only case in any way similar to it that I know of is one alluded to by Dr. Henry, in the 'Transactions' of this Society for the year 1819, in which the autopsy of a girl of fifteen, who had suffered from much cough, revealed a concretion the size of a musket ball embedded in the left bronchus. But though this case has a superficial resemblance to the present one, it appears to have been really of quite a different nature ; for, as far as can be judged from the report of Dr. Henry's case, the mass was not of glandular origin, and was formed within the bronchial tube, and did not invade it from without.

Formation of Mediastinal Abscess.

Mediastinal abscesses usually have their origin in disease of the vertebræ or in scrofulous lymphatic glands. In the present case there was no such cause, and it seems probable that while the patient was riding a sudden movement or strain lacerated or tore some of the areolar tissue which matted the glands together, or even fractured one of the brittle glands, setting up an acute inflammatory process which went on to suppuration. Suppuration in the mediastina is usually diffuse, but one remarkable point in the present case is that the formation of pus took place within a pathological cavity with rigid walls which encysted it, until it eventually made an exit for itself by perforating the wall of the right bronchus to which the tumour was adherent.

The symptoms which occurred during the patient's illness up to the date when this occurred may be conveniently considered here.

The first symptom was the *pain* around the wall of the chest ; it was at first felt on the right side, afterwards also on the left. It was accompanied by extreme hyper-

æsthesia of the surface of the chest wall, and by tenderness over the fifth and sixth dorsal spinous processes. The pain itself was most intense, and was insupportable without repeated injections of morphia; it lasted for many weeks, and until the tension of the abscess was relieved by the discharge of pus into the interior of the bronchus. There was considerable tenderness over the vertebral spines, but the pain could not be attributed to disease of the vertebræ, for there was free movement of the spinal column, and no rigidity of the muscles of the back.

So long as the pain was the *only* symptom its cause was quite unascertainable, but I think that the post-mortem appearances prove that it was a pain referred to the chest wall from the seat of disease in the mediastinum, and was set up there by the tension of the pus imprisoned within the rigid walls of the calcareous tumour. Pain around the chest wall is similarly complained of in some cases of aneurism of the arch of the aorta when it is not possible that it can result from any actual pressure upon the intercostal nerves. The tenderness over the vertebral spines was at the same horizontal level as the mass of calcareous glands, and was probably dependent upon it. This transference of pain to the intercostal spaces and the tenderness over the spinous processes from a lesion in the centre of the chest cavity may possibly be of service in the diagnosis of future obscure cases of mediastinal disease.

After the pain had persisted for days there occurred repeated rigors with intermittent severe pyrexia, and it became evident that there was organic disease somewhere within the thoracic cavity. Of lung disease there was still no sign; the absence of murmurs (except the slight systolic murmur heard at the base of the heart on the first day), the intensity of the pain, and the tranquility of the heart's action militated against ulcerative endocarditis or endarteritis, and we came to the conclusion that the fever and rigors were only to be attributed to pus formation, and that the pus was probably in the mediastinum; its exact locality remained a mystery

until the supervention of the incessant and harassing spasmodic cough, which, in the absence of any laryngoscopic sign of disease, pointed to something close to the air-passages, and causing irritation of the laryngeal nerves. It was still, however, inexplicable that we could find no other pressure signs or other indications of the exact locality of the pus. The necropsy showed that the abscess was encysted within the calcareous tumour. It was this imprisonment of the pus in a previously formed pathological space, the existence of which had given no symptoms, that made the exact diagnosis so impossible. No doubt the incessant cough was set up by the gradual perforation of the bronchial wall by the abscess.

The *attack of pleurisy* commenced apparently over the lower part of the back of the left lung, for it was here that localised pleuritic friction sound was audible for some days. At the time it was difficult to explain why the pleurisy occurred, and why it started at a distance from the local disease in the mediastinum; and even with the knowledge subsequently acquired from the post-mortem examination the reasons for the pleuritic attack remain unexplained.

After the dry pleurisy had lasted for some days there suddenly succeeded a very rapid and copious serous effusion; the heart became at once greatly displaced, and loud murmurs became simultaneously audible; the patient's life was in imminent danger, and death was certainly averted by the first aspiration; after repeated aspirations the effusion gradually became less and less.

Pneumothorax.—After the serous effusion had gradually lessened till its quantity was but small, the heart still remained displaced, and the syncopal attacks became more frequent. At about this time there were indications of free air in the thoracic cavity, but the amphoric breathing which denoted this was limited to the front of the left chest, while there was serous effusion at the back on the same side, as shown by the physical signs and by

the aspirator. Again, though there was air and there was liquid in the left side of the thorax, no succussion sound could ever be obtained. The physical signs, therefore, were somewhat anomalous, and no air passed out through the aspirator when it was introduced into the region of the serous effusion.

At the time I thought that air must have entered the chest during the aspirations. With the light thrown upon the case by the necropsy, it seems more probable that it was sucked in with each inspiratory act through the fistulous opening in the right bronchus, and accumulated until it came to exercise considerable pressure upon the pericardium and produced the later syncopal attacks.

Again, I am at a loss to account for the presence of air on the *left* side of the chest, while the opening was in the *right* bronchus; but it must be remembered that the relative position of organs was greatly altered by the extreme displacement of the heart to the right by the serous effusion before the perforation in the bronchus had occurred.

Is it possible that the air which entered through the opening in the bronchus did not pass into the pleural sac, but into the mediastinal space, and forced its way between the left edge of the pericardial sac and the left pleura which had become adherent? This might account for the anomalous physical signs of pneumothorax to which I have above directed attention. Be this as it may, I think the repeated syncopal attacks and the physical signs I have described are of interest as resulting from pressure of air within the chest cavity, after the quantity of serous pleural effusion had become insignificant.

During the second and third periods of the patient's illness there existed a *fistulous opening through the right bronchus*. The only symptoms it gave rise to were a little wheeziness on breathing and a slight huskiness of voice, both much increased by damp or foggy weather; the small tree-like casts of the bronchial tubes were not expectorated until after the communication with the interior of

the aorta had been established, and they cannot be attributed solely to the presence of the bronchial perforation. It was not till several months after this perforation had been produced that there was any deviation from the normal detectable in the physical signs, but eventually there was a slight flattening of the upper part of the right chest and a rise of pitch in the percussion note, with a marked increase in the vocal resonance.

When the hæmoptysis became associated with these signs the case greatly simulated one of phthisis; the post-mortem examination, however, showed no trace of phthisis, and proved that the physical signs were produced by the slight shrinking of the apex of the lungs due probably to its not being for a long period fully distended on inspiration, in consequence of the perforation in the bronchus. The clinical simulation of phthisis by the bronchial fistula is one of the interesting features in this case.

The hæmorrhage from the aorta.—From the preparation (1148, St. Mary's Hospital Museum) which I show this evening it will be seen that the perforation of the wall of the aorta is situate in the beginning of the transverse part of the arch of the aorta.

No doubt it resulted from gradual erosion of the wall of the artery by its contact with the calcareous glands, a result which must have been materially assisted by the respiratory movements of the trachea to which the calcareous mass was attached, and by the repeated distension of the aorta itself with each systole of the heart. The opening into the aorta led into the interior of the calcareous tumour, whence the blood passed into the air-passages through the perforated bronchus. The amount of blood lost by the patient during the fifteen months of recurring hæmorrhages from the aorta must have amounted to not less than nine or ten gallons; but more extraordinary perhaps than the amount of blood lost was that the patient continued to live with a free

opening into the aorta distant only three inches from the heart itself.

The *diagnosis* at this period was replete with difficulties. As already pointed out, there were certain physical signs at the apex of the right lung which, taken with the hæmoptysis, suggested tubercular phthisis, but as time went on these physical signs in no way increased, no tubercle bacilli were ever discoverable in the blood ejected, and the amount of blood lost was far more than was consistent with the slight phthisis that was present if there was any phthisis at all. In addition there was no fever, and the patient picked up strength immediately after each hæmorrhage with a rapidity which made it certain that it was not merely a case of phthisis to deal with.

At one time some small pieces of membrane were expelled, and this with the repeated hæmorrhages and slight physical signs gave the case a resemblance to one of hydatids of the lung, but no more membrane was afterwards brought up that might have been microscopically examined.

Aneurysm was excluded by the absence of bruit or of pressure signs, except that I on one occasion heard over the right supra-spinous fossa a faint distant sound like a venous hum. The exact condition remained a mystery until the autopsy revealed the remarkable coincidence of openings into the aorta and bronchus which it would have been quite impossible to foretell.

The repeated expectoration of the *bronchial casts* is the only other point on which I need comment; they were always preceded by hæmoptysis, usually by a few hours only, but they were never in the least blood-stained. They showed no laminated structure under the microscope, such as is seen in some cases of plastic bronchitis, and the only supposition I can form as to their origin is that they resulted from coagulation of the fibrin of some of the blood which escaped into the smaller bronchial tubes.

I know of no case at all resembling the present case except one in the 'Transactions' of this Society for 1846, reported by Mr. Busk.

A woman aged 35 had a small hard lump in the mesial line of the neck, close above the upper edge of the sternum. This gave no pain or inconvenience for thirteen and a half years; the swelling then suppurated and opened spontaneously, discharging a quantity of "white matter." Five months later bleeding came on suddenly from the fistulous opening; this was arrested by a compress, but recurred the next morning. On removal of the compress the blood was ejected in a jet which rose above the surgeon's head. The bleeding continued, and the woman died in forty-eight hours from the commencement of the hæmorrhage.

The autopsy showed that the ulcerated opening communicated with an old abscess cavity containing a pound of coagulum occupying the point of the neck below the thyroid cartilage, being bounded posteriorly by the trachea, in front by the fascia and integuments. The cavity extended downwards and backwards between the right bronchus and the arteria innominata, behind the root of the right lung to the front and right sides of the bodies of two or three upper dorsal vertebræ, and inferiorly the abscess cavity reached the right side of the ascending part of the arch of the aorta, and for about two inches below the origin of the innominate artery, the external cellular tunic of that vessel as well as the greater part of the external side of the arteria innominata was completely removed. The middle coat of the aorta was quite bare, and there was a small opening or fissure about one eighth of an inch long in the middle of the denuded portion opening into the main trunk of the vessel.

This case resembles that I have recorded this evening in affording, to use Mr. Busk's words, "an unequivocal instance of a communication between the cavity of an abscess and the aorta, in consequence of an ulcerative process set up from without."

But there are many points of difference between the two cases ; and what makes the present case I believe a unique one is the formation of the calcareous tumour which subsequently became the medium of communication between an opening into the bronchus and an opening into the main trunk of the aorta, so that there eventually resulted the unique condition of an old abscess cavity communicating on the one side with the aorta, on the other side with the bronchus, the blood being thus afforded a free exit into the trachea.

Mr. Busk remarks on the length of time—forty-eight hours—his patient survived after hæmorrhage had commenced from the ascending aorta, but in this particular the present case is far more extraordinary, for a similar hæmorrhage here continued for no less than fifteen months.

With regard to the treatment, it was directed to the relief of the intense pain by the use of morphia and other anodynes. When it became evident that there was pus to be evacuated we were on the watch for any local indications of its presence, and it was eventually evacuated spontaneously. Withdrawal of liquid from the pleura was performed when there was great orthopnoea or tendency to syncope. The terrible cough was very difficult to alleviate, but an oro-nasal inhaler with eucalyptus gave most relief.

The free opening was made into the chest cavity, though there was no sign of the liquid in it being purulent, only after very careful consideration and with the certain conviction on the part of those who had watched the case from the commencement that the patient would very soon die unless it were done. Its results were most satisfactory, for it resulted in the cure of the disease so far as it had then gone.

During the period of recurrent hæmorrhages the only treatment possible was directed towards controlling it by rest and by drugs, of which far the most efficacious proved to be the tincture of hamamelis.

Beyond this, in the obscurity which rested upon the nature of the case, nothing could be attempted, and it is certain that even had we known of the exact conditions present no further treatment would have been feasible.