CLINICAL LECTURE

ON THE

RELATION BETWEEN LARYNGEAL AND PULMONARY DISEASE.

By GEORGE JOHNSON, M.D., F.R.S.,

Physician to King's College Hospital; Professor of Medicine in King's College.

History of a Case of Tertiary Syphilis.—Ulceration of the Larynx, followed by Acute Pneumonia.—Symptoms and Physical Signs.—Post Mortem Appearance.—Explanation of the Symptoms and of the Relation between the Laryngeal and the Pulmonary Disease.—Chronic Laryngitis a frequent Cause of Disease in the Lung.—Two Modes in which the Lung may be infected by a Primary Disease in the Larynx.—Importance of early and careful Treatment in Cases of Laryngeal Disease.

GENTLEMEN,—I wish to direct your attention to a case which has recently occurred in Craven Ward. I will give you a brief abstract of Mr. Brayn's full and complete notes of the case.

John B., aged 33, a labourer, about three years ago had two venereal sores on the prepuce at the same time, then a suppurating bubo in the left groin, and soon afterwards an eruption on the skin. The eruption appears soon to have assumed the form of rupia, and it left several circular scars about the size of a shilling on his arms, legs, and neck. About six months ago, a node formed on the centre of the forehead. Five weeks before his admission, his throat became sore for the first time; and with this he had cough, hoarseness, and some difficulty in breathing. For these symptoms, he was admitted on October 6th. He had a pallid, somewhat emaciated, and cachectic appearance. There was a swelling on the forehead about the size of half a hen's egg, hard at the margins, soft and elastic in the centre, not painful. There was a prominence—a bony node, not painful—on the front of the right tibia. There was cough, with some mucous expectoration; the voice was feeble and husky; no dyspnoea, or stridor, or dysphagia. Laryngoscopic examination showed a sharp-edged somewhat irregular ulcer, about the size of a pea, over the right arytenoid cartilage; the mucous membrane round the ulcer being red and swollen. The chest-sounds were normal; the urine was normal. He was ordered to take the following mixture.

R Potassii iodidi gr. v; liquor. hydrarg. perchloridi ʒi; tinct. cinchonæ comp. ʒi; aquæ ʒi—ter die post cibum.

On the evening of the 8th, he had a rigor; and at 7 P.M. the temperature rose to 103.2. Pulse 144.

On the 9th, the morning temperature was 102; pulse 144; respirations 24. He had some pain in the right side of the chest. In the evening, temperature 103.6; pulse 144. Up to this time, no abnormal sounds were heard over the lung.

On the morning of the 10th, there was some dullness on percussion and fine crepitation over the lower lobe of the right lung. Temperature 99.6; pulse 120. The sputum was somewhat viscid and slightly rusty. He had severe pain over the right side, for which a linseed poultice was applied. There was free perspiration.

On the 11th, he had slept fairly well; the pain was less severe; there was profuse perspiration; the dullness on percussion over the lower lobe of the lung had increased; and dry tubular breathing and bronchophony were heard over the dull space. The vocal fremitus was decidedly lessened over the hepatized lung. Temperature 99.6; pulse 144. He had cough, with muco-purulent expectoration.

October 12th. He was much the same in every respect.

October 13th. He had frequent cough, with rather copious expectoration and free perspiration. Temperature 100; pulse 128.

October 16th. There was no material change. The physical signs remained as before, except that the respiration was less distinctly tubular, and there was some loose crepitation over the dull space.

On the 23rd, the physical signs over the lower lobe of the right lung remained the same; but there was now decided dullness on percussion over the middle lobe. There was a copious frothy expectoration. He was ordered to have a quinine mixture three times a day, and six ounces of brandy in the twenty-four hours.

From this time, there was a rapid loss of flesh and strength, with an extension of disease in the right lung. On October 30th, there was dullness on percussion, with blowing respiration, over the upper lobe; so that the physical signs now indicated consolidation of the whole right lung, and these signs remained the same until he died on November 8th.

On inspection, the whole right lung was found in a state of red hepatization, and the lower lobe was adherent to the wall of the chest by recent lymph. The left lung was engorged and oedematous. There had been no excavation of the lung-tissue; nor was there any tuberculous deposit. There was an ulcer over the right arytenoid cartilage, exposing the cartilage, which was indurated by calcareous deposit, and apparently in a state of necrosis.

There are several points of interest in this case to which I wish to direct your attention. First, we had the opportunity of witnessing the onset of the pneumonia; and we saw, what may be seen in most cases of acute pneumonia, that rigors, high temperature, quick pulse and breathing, preceded the local symptoms in the chest. The febrile symptoms began on the evening of the 8th; and on the morning of the 10th, about thirty-six hours afterwards, the physical signs of pneumonia were first noted, though there had been pain over the right side on the previous day. Then we saw what I have frequently pointed out to you in similar cases, that, simultaneously with the exudation into the lung, the febrile symptoms abated. Between the evening of the 9th and the morning of the 10th, when the physical signs indicated inflammatory exudation into the lower lobe of the right lung, the temperature fell from 103.6 to 99.6, and the pulse from 144 to 120. It would seem that the febrile symptoms are associated with some morbid blood-changes; and that, with the exudation of the morbid products into the lung, the fever abates. The phenomena are strictly analogous to the rapid subsidence of the initiatory fever of small-pox with the first appearance of the variolous eruption on the skin. I therefore am in the habit of saying that a case of so-called idiopathic pneumonia may be looked upon as an acute febrile eruptive disease, the eruption occurring, not upon the skin—except in the form of herpes labialis, which so often accompanies it—but into the lung. In uncomplicated cases of pneumonia, there is no considerable rise of temperature again after the lung is consolidated; but, with secondary changes in the lung, softening, and suppuration, there may be febrile symptoms analogous to the secondary fever during the suppurative stage of small-pox.

We noticed in our patient what may usually be observed in cases of acute pleuro-pneumonia; namely, that the pleuritic pain which marks the onset of the local changes in the lung and pleura, is much mitigated when the physical signs indicate that the lung is hepatized. The probable explanation is, that the consolidation of the lung lessens the movement and friction of the opposed pleuritic surfaces; and, with diminished friction, there is diminution of pain. So we may often observe that during the stage of resolution, when the air re-enters the pulmonary cells and the lung expands more freely, the greater friction between the lung and the chest-wall is associated with a temporary increase of pain, and with a friction-sound and fremitus, which are sometimes erroneously supposed to indicate a renewal or an increase of the pleuritic inflammation. I believe the explanation of the phenomena to be that which I have given. The pleura over a hepatized lung is usually, as in this case, more or less inflamed and covered by exuded lymph; while the lung is solidified, there is little or no movement of the opposed surfaces, and therefore little pain, and no friction-sound or fremitus; but there is often a return of pain and friction-sound during the stage of resolution, and before the formation of adhesions between the pulmonary and costal pleura.

Another physical phenomenon to which I would direct your attention for a moment is the marked diminution of the vocal fremitus over the hepatized lung. You will often find it stated that an increase of the vocal fremitus is an almost constant phenomenon during the second stage of pneumonia, and that by this physical sign you may distinguish consolidation of the lung from liquid in the pleura. I warn you, however, that you cannot rely upon this means of diagnosis. You will find that in about half the cases of pneumonia, while other physical signs clearly indicate consolidation of the lung without liquid in the pleura (we have seen that in our patient's pleura there was no liquid effusion), the vocal fremitus over the hepatized lung is either unchanged or, as in this case, very decidedly less than on the sound side.

Now there arises the interesting question, What was the exciting cause of the pneumonia in this case? I cannot give you an infallibly certain answer to this question; but I suspect, and I venture to suggest, that the pneumonia was the result of a blood-infection through the absorption of morbid products from the ulcer in the larynx. The pneumonia came on after the patient had been for some days in bed, and without exposure to cold or other obvious external exciting cause. We may be quite certain that every so-called idiopathic disease has a physical exciting cause, whether we can discover it or not. It is always scientifically interesting, and not rarely it is of the highest practical importance, to discover the determining cause of any malady that we are called upon to treat. Not long since, I saw an acute pneumonia occur under the following circumstances. A boy about fourteen years of age

had his membrana tympani punctured for the relief of deafness. The operation was attended with considerable pain, and followed by a discharge from the external meatus. In the course of two or three days, the boy began to complain of chilliness and languor; there was some stiffness in the neck; and the lymphatic glands below the damaged ear were enlarged. When I saw him on the eighth day after the operation, he was in bed, with a temperature of 103 deg.; there was some dullness on percussion, and fine crepitation over the lower lobe of the left lung. On the following day, the dullness over the lower lobe had increased, and there was bronchial breathing and bronchophony, while the temperature had fallen to 99. The consolidation of the lung rapidly passed away, and in a few days he was convalescent. In that case, the sequence of events was sufficiently obvious. Some morbid infecting product from the wounded ear passed through the lymphatics in the neck to the general circulation. The blood-infection was indicated by general *malaise* and a febrile elevation of temperature; and this again subsided with the occurrence of exudation into the lung. In that case, I have no doubt that the wound of the membrana tympani was the primary cause of the pneumonia. In the case which we are now discussing, it is not so certain, but it is, I think, highly probable, that the ulcer in the larynx was, through a secondary blood-infection, the determining cause of the pulmonary inflammation which supervened, and which proved fatal in consequence of the very enfeebled and cachectic condition to which the patient had been reduced by a long train of syphilitic symptoms.

There is good reason to believe that disease beginning in the larynx is a frequent determining and exciting cause of disease in the lungs. During the last ten years, since I have learnt to use the laryngoscope, I have seen, both in hospital and in private practice, a large number of cases in which chronic disease beginning in the larynx has been followed by tubercular disease of the lungs. These cases come under the designation "laryngeal phthisis". This painful and fatal malady often begins with a neglected cold. The sore-throat and hoarseness excited by exposure to cold and wet remain for weeks and months; there is cough, with mucous expectoration; perhaps some pain and difficulty in swallowing; and there may be more or less stridor on deep inspiration. Looking at the larynx with the mirror, we see swelling and perhaps ulceration of the epiglottis, or of the mucous membrane within the larynx, the false cords, and the interarytenoid fold. In some cases, the mucous membrane is injected and red; but the most characteristic appearance consists of a pale, doughy, oedematous thickening, with or without ulceration. In some cases, the swelling of the mucous membrane remains for months without the occurrence of ulceration. In the earlier stages of this laryngeal affection, there is no evidence of pulmonary disease. The chest-movements and the percussion-sound are normal; and, when auscultation is not interfered with, as it often is, by laryngeal obstruction or stridor, the natural respiratory sounds may be plainly heard. Sooner or later, however, in a large proportion of cases, the physical signs of pulmonary disease are discoverable, and the patient dies with tuberculous lungs. Having closely observed a large number of these cases, I have gradually arrived at the conclusion that the pulmonary disease is a more or less direct result of the pre-existing laryngeal affection. There are two modes in which it is conceivable that a disease in the larynx may be a source of infection, and so a cause of secondary disease of the lungs. It is possible that morbid infecting materials from an ulcer in the larynx may be carried directly by the inspiratory current of air into the interior of the lungs, and there set up disease. This supposed mode of infection would be analogous to that which occurs in cases of diphtheria. The air-borne diphtheritic poison first infects those parts of the throat with which it comes into direct contact. Then the morbid process set up in the fauces acts as a new source of infection, from which the poison is conveyed by the inspired air into the larynx, trachea, and bronchi, with results usually fatal. I refer to diphtheria only as an illustration of my meaning. Obviously, the infective qualities of an ordinary laryngeal ulcer must be far less than those of a diphtheritic exudation in the fauces. Again, it is highly probable that a swollen and ulcerated larynx may, through the lymphatics or the blood-vessels, prove a source of infection and of a secondary tuberculous disease in the lungs—a result analogous to that obtained by Drs. Sanderson, Wilson Fox, and others, who, by exciting suppurative beneath the skin of the guinea-pig, have determined the formation of a kind of tuberculous deposit in the lungs and other organs of the animal.

The probability that a chronic laryngeal inflammation may, in addition to the local suffering which it often occasions, prove a source of blood-contamination, and ultimately of fatal pulmonary disease, affords a powerful argument, if such were needed, in favour of the early and careful treatment of all cases of laryngeal inflammation. Prompt and persevering treatment is obviously most urgently demanded in the case

of those whose family history or constitutional diathesis indicates a more than ordinary proneness to disease of a strumous or tuberculous character.

ADDENDUM TO A LECTURE ON THE TREATMENT OF TAPEWORM.

By T. SPENCER COBBOLD, M.D., F.R.S.,

Lecturer on Parasitic Diseases at the Middlesex Hospital Medical College.

IN reply to numerous letters, the substance of which has been communicated to me, I beg to state that one main object of my lecture was to insist very strongly on the necessity of persistent and careful personal search by the medical attendant of the stools of his patient, until he was satisfied that the neck and entire head were found. To some practitioners, this seems apparently a trivial and easy thing to do. On this score, however, I have reason to know that it is not easy, and that the duty is frequently, in many cases habitually, omitted; and hence we have one important cause of failure to cure tapeworm.

No portion of the *feces* should be left uninvestigated. On many occasions I have remonstrated with patients and attendants, who, from motives of delicacy, have, prior to my visit for the express purpose of ascertaining results, carefully removed all floating matters of a non-faecal character. I have obtained proof that the head of the worm is liable to adhere to such light materials.

Of course, in conducting these researches, one is apt to encounter rather unpleasant experiences. Some of these I recounted to my class with the view of impressing my hearers with the necessity of performing this duty in a thorough manner. The examination should extend to every stool while the patient is kept under treatment. As I invariably employ disinfectants, the disagreeableness of the task is not so marked as many might suppose; nevertheless I have sometimes been attacked with nausea, consequently upon several hours' search in a stooping posture.

By way of illustrating the proceedings necessary in some cases coming within my experience, I narrated in the lecture, which appeared in these columns in an abbreviated form, the following case. A practitioner of repute having repeatedly attempted and failed to cure his daughter, solicited my advice, by letter, in the first instance. I prescribed male-fern extract in the ordinary way, and recommended a search for the head after the exhibition of the *taniafuge*. As, however, at varying intervals, the worm continued to reappear, I at length advised him to send the patient up to town, which he did. I again prescribed the oil of male-fern. The body of the worm came away, as usual. After removing from the stools some ten or eleven feet of the body, including many loose proglottides, I continued the search for about two hours, during which I obtained some fragments of the neck, which were so fine from the part near the head that the transverse lines of segmentation were barely discernible. Ordering a small dose of castor-oil, with plenty of weak tea and warm milk, I promised to renew the search next day. I felt confident the head must have been dislodged from the upper bowel. When I called next day, the fresh evacuations had been retained in accordance with my instructions, and the investigation was renewed. Greatly advantaged by the strong light coming through the large hotel window, it was not many minutes before I noticed in the *feces* four minute dots of uniform size. As these mere specks were placed side by side in a symmetrical manner, I concluded that they were the suckers of the head of the tapeworm.

a. The four dots seen in the *feces*. b. The same on removal. c. One of the separated suckers (enlarged). d. Diagram of the head of the tapeworm, in which the shaded portion represents what remained of the head. The central falsely so-called supplementary sucker was entirely obliterated.