V. DISEASES OF THE PLEURA.

I. ACUTE PLEURISY.

Anatomically, the cases may be divided into dry or adhesive pleurisy and pleurisy with effusion. Another classification is into primary or secondary forms. According to the course of the disease, a division may be made into *acute* and *chronic* pleurisy, and as it is impossible, at present, to group the various forms etiologically, this is perhaps the most satisfactory division. The following forms of acute pleurisy may be considered:

I. Fibrinous or Plastic Pleurisy.

In this the pleural membrane is covered by a sheeting of lymph of variable thickness, which gives it a turbid, granular appearance, or the fibrin may exist in distinct layers. It occurs (1) as an independent affection, following cold or exposure. This form of acute plastic pleurisy without fluid exudate is not common in perfectly healthy individuals. Cases are met with, however, in which the disease sets in with the usual symptoms of pain in the side and slight fever, and there are the physical signs of pleurisy as indicated by the friction. After persisting for a few days, the friction murmur disappears and no exudation occurs. Union takes place between the membranes, and possibly the pleuritic adhesions which are found in such a large percentage of all bodies examined after death originate in these slight fibrinous pleurisies.

Fibrinous pleurisy occurs (2) as a secondary process in acute diseases of the lung, such as pneumonia, which is always accompanied by a certain amount of pleurisy, usually of this form. Cancer, abscess, and gangrene also cause plastic pleurisy when the surface of the lung becomes involved. This condition is specially associated in a large number of cases with tuberculosis. Pleural pain, stitch in the side, and a dry cough, with marked friction sounds on auscultation are the initial phenomena in many instances of phthisis. The signs are usually basic, but Burney Yeo has recently called attention to the frequency with which they occur at the apex.

II. Sero-fibrinous Pleurisy.

In a majority of cases of inflammation of the pleura there is, with the fibrin, a variable amount of fluid exudate, which produces the condition known as pleurisy with effusion.

Etiology.—For generations physicians have considered cold the potent factor in inducing pleurisy. This may be true in many cases, but modern views of serous inflammations scarcely recognize cold as anything more than a predisposing agent, which permits the action of various micro-organisms. We have not yet, however, brought all the acute pleurisies into the category of microbic affections, and the fact remains that pleurisy does follow with great rapidity a sudden wetting or a chill. Of late years an attempt has been made, particularly by French writers, to show that the majority of acute pleurisies are tuberculous. In this connection the following facts may be admitted: (1) In a limited number of cases of pleurisy coming on abruptly in healthy persons the disease has been shown—(*a*) by post-mortem, in cases of accidental or sudden death, (*b*) by the subsequent history—to be tuberculous; (2) in a larger proportion of those cases which come on insidiously in persons who have been in failing health or who are delicate the disease is tuberculous from the outset; (3) the acute pleurisy, which occurs as a secondary, often a terminal, event in chronic affections, such as cirrhosis of the liver, Bright's disease, and cancer, is very frequently tuberculous. I confess that the more carefully I have studied the question the larger does the proportion appear to be of primary pleurisies of tuberculous origin. The subsequent history of cases of acute pleurisy forces us to conclude that in at least two thirds of the cases it is a curable affection. This may well be so, according to our present ideas of local tuberculous disease. One of the most interesting contributions to this question has been made from the records of Henry I. Bowditch, of Boston, to whom we are indebted for so many important contributions to our knowledge of pleurisy.* Of 90 cases of acute pleurisy which had been under observation between 1849 and 1879, 32 died of or had phthisis—a percentage large enough to indicate what an important *rôle* tuberculosis plays in the etiology of this disease.

Morbid Anatomy.—In sero-fibrinous pleurisy the serous exudate is abundant and the fibrin is found on the pleural surfaces and scattered through the fluid in the form of flocculi. The proportion of these constituents varies a great deal. In some instances there is very little membranous fibrin; in others it forms thick, creamy layers and exists in the dependent part of the fluid as whitish, curd-like masses. The fluid of sero-fibrinous pleurisy is of a citron color, either clear or slightly turbid, depending on the number of formed elements. In some instances it has a dark-brown color. The microscopical examination of the fluid shows leucocytes, occasional swollen cells, which may possibly be derived from the pleural endothelium, shreds of fibrillated fibrin, and a variable number of red blood-corpuscles. On boiling, the fluid is found to be rich in albumen. Sometimes it coagulates spontaneously. Its composition closely resembles that of blood-serum. Cholesterin, uric acid, and sugar are occasionally found. The amount of the effusion varies from a half to four litres.

The lung in acute sero-fibrinous pleurisy is more or less compressed. If the exudation is limited the lower lobe alone is atelectatic; but in an extensive effusion which reaches to the clavicle the entire lung will be found

* Vincent Y. Bowditch, in Boston Medical and Surgical Journal, 1889.

lying close to the spine, dark and airless, or even bloodless—i. e., carnified.

In large exudations the adjacent organs are displaced. In large right-sided pleurisies the liver is much depressed. Rather varying statements are made with reference to the position of the heart and as to whether or not it rotates on its axis. In a number of post-mortems I have carefully studied its position, both in pneumothorax and in large effusions, and can speak with some degree of certainty on the following points: (1) Even in the most extensive left-sided exudation there is no rotation of the apex of the heart, which in no case was to the right of the mid-sternal line; (2) the relative position of the apex and base is usually maintained; in some instances the apex is lifted, in others the whole heart lies more transversely; (3) the right chambers of the heart occupy the greater portion of the front, so that the displacement is rather a definite dislocation of the mediastinum, with the pericardium, to the right, than any special twisting of the heart itself; (4) the kink or twist in the inferior vena cava described by Bartels was not present in any of the cases.

Symptoms.—Prodromata are not uncommon, but the disease may set in abruptly with a chill, followed by fever and a severe pain in the side. It is remarkable, however, with what frequency the disease comes on insidiously. The pain in the side is the most distressing symptom, and is usually referred to the nipple or axillary regions. It must be remembered, however, that pleuritic pain may be felt in the abdomen or low down in the back, particularly when the diaphragmatic surface of the pleura is involved. It is lancinating, sharp, and severe, and is aggravated by cough. At this early stage, on auscultation, sometimes indeed on palpation, a dry friction rub can be detected. The fever rarely rises so rapidly as in pneumonia, and does not reach the same grade. A temperature of from 102° to 103° is an average pyrexia. It may drop to normal at the end of a week or ten days without the appearance of any definite change in the physical signs, or it may persist for several weeks. The temperature of the affected is higher than that of the sound side. Cough is an early symptom in acute pleurisy, but is rarely so distressing or so frequent as in pneumonia. There are instances in which it is absent. The expectoration is usually slight in amount, mucoid in character, and occasionally streaked with blood.

At the outset there may be dyspnœa, due partly to the fever and partly to the pain in the side. Later it results from the compression of the lung, particularly if the exudation has taken place rapidly. When, however, the fluid is effused slowly, one lung may be entirely compressed without inducing shortness of breath, except on exertion, and the patient will lie quietly in bed without evincing the slightest respiratory distress. When the effusion is large the patient usually prefers to lie upon the affected side.

Physical Signs.—*Inspection* shows some degree of immobility on the affected side, depending upon the amount of exudation, and in large effusions an increase in volume, which may appear to be much more than it really is as determined by mensuration. The intercostal spaces are obliterated. In right-sided effusions the apex beat may be lifted to the fourth interspace or be pushed beyond the left nipple, or may even be seen in the axilla. When the exudation is on the left side the heart's impulse may not be visible; but if the effusion is large it is seen in the third and fourth spaces on the right side, and sometimes as far out as the nipple, or even beyond it.

Palpation enables us more successfully to determine the deficient movements on the affected side, and the obliteration of the intercostal spaces, and more accurately to define the position of the heart's impulse. In simple sero-fibrinous effusion there is rarely any œdema of the chest walls. It is scarcely ever possible to obtain fluctuation. Tactile fremitus is greatly diminished or abolished. If the effusion is slight there may be only enfeeblement. The absence of the voice vibrations in effusions of any size constitutes one of the most valuable of physical signs. In children there may be much effusion with retention of fremitus. In rare cases the vibrations may be communicated to the chest walls through localized pleural adhesions.

Mensuration.—With the cyrtometer, if the effusion is excessive, a difference of from half an inch to an inch, or even, in large effusions, an inch and a half, may be found between the two sides. Allowance must be made for the fact that the right side is naturally larger than the left. With the saddle-tape the difference in expansion between the two sides can be conveniently measured.

Percussion.—Early in the disease, when the pain in the side is severe and the friction murmur evident, there may be no alteration, but with the gradual accumulation of the fluid the resonance becomes defective, and finally gives place to absolute dulness. From day to day the gradual increase in height of the fluid may be studied. In a pleuritic effusion rising to the fourth rib in front, the percussion signs are usually very suggestive. In the subclavicular region the attention is often aroused at once by a tympanitic note, the so-called Skoda's resonance, which is heard perhaps more commonly in this situation with pleural effusion than in any other condition. It shades insensibly into a flat note in the lower mammary and axillary regions. Skoda's resonance may be obtained also behind, just above the limit of effusion. The dulness has a peculiarly resistant, wooden quality, differing from that of pneumonia and readily recognized by skilled fingers. It has long been known that when the patient is in the erect posture the upper line of dulness is not horizontal, but is higher behind than it is in front, forming a parabola. Ellis and Garland, of Boston, who have made a careful study of this question, state that the line of dulness from behind forward may sometimes be represented by a curved line resembling the letter S. The condition is fully considered in Garland's exhaustive work on Pneumo-dynamics.

On the right side the dulness passes without change into that of the liver. On the left side in the nipple line it extends to and may obliterate Traube's semilunar space. If the effusion is moderate, the phenomenon of movable dulness may be obtained by marking carefully, in the sitting posture, the upper limit in the mammary region, and then in the recumbent posture, noting the change in the height of dulness. This infallible sign of fluid cannot always be obtained. In very copious exudation the dulness may reach the clavicle and even extend beyond the sternal margin of the opposite side.

Auscultation.—Early in the disease a friction rub can usually be heard, which disappears as the fluid accumulates. It is a to-and-fro dry rub, close to the ear, and has a leathery, creaking character. There is another pleural friction sound which closely resembles, and is scarcely to be distinguished from, the fine crackling crepitus of pneumonia. This may be heard at the commencement of the disease, and also, as pointed out in 1844 by MacDonnell, Sr., of Montreal, when the effusion has receded and the pleural layers come together again.

With even a slight exudation there is weakened or distant breathing. Often inspiration and expiration are distinctly audible, though distant, and have a tubular quality. Sometimes only a puffing tubular expiration is heard, which may have a metallic or amphoric quality. Loud resonant râles accompanying this may forcibly suggest a cavity. These pseudo-cavernous signs are met with more frequently in children, and often lead to error in diagnosis. Above the line of dulness the breath-sounds are usually harsh and exaggerated, and may have a tubular quality.

The vocal resonance is usually diminished or absent. The whispered voice is said to be transmitted through a serous and not through a purulent exudate (Baccelli's sign). There may, however, be intensification—bronchophony. The voice sometimes has a curious nasal, squeaking character, which was termed by Laennec *ægophony*, from its supposed resemblance to the bleating of a goat. In typical form this is not common, but it is by no means rare to hear a curious twang-like quality in the voice, particularly at the outer angle of the scapula.

In the examination of the heart in cases of pleuritic effusion it is well to bear in mind that when the apex of the heart lies beneath the sternum there may be no impulse. The determination of the situation of the organ may rest with the position of maximum loudness of the sounds. In the displaced organ a systolic murmur may be heard. When the lappet of lung over the pericardium is involved on either side there may be a pleuro-pericardial friction.

The *course* of acute sero-fibrinous pleurisy is very variable. After persisting for a week or ten days the fever subsides, the cough and pain disappear, and a slight effusion may be quickly absorbed. In cases in which the effusion reaches as high as the fourth rib recovery is usually slower. Many instances come under observation for the first time, after two or

three weeks' indisposition, with the fluid at a level with the clavicle. The fever may last from ten to twenty days without exciting anxiety, though, as a rule, in ordinary pleurisy from cold, as we say, the temperature in cases of moderate severity is normal within eight or ten days. Left to itself the natural tendency is to resorption; but this may take place very slowly. Even after it has persisted for months a sero-fibrinous exudate may completely disappear. With the absorption of the fluid there is a redux-friction crepitus, either leathery and creaking or crackling and râle-like, and for months, or even longer, the defective resonance and feeble breathing are heard at the base.

A sero-fibrinous exudate may persist for months without change, particularly in tuberculous cases, and will sometimes reaccumulate after aspiration and resist all treatment. The change of the exudate into pus will be spoken of in connection with empyema. Death is a rare termination of sero-fibrinous effusion. When one pleura is full and the heart is greatly dislocated the condition, although in a majority of cases producing remarkably little disturbance, is not without risk. *Sudden death* may occur, and its possibility under these circumstances should always be considered. I have seen two instances—one in right and the other in left sided effusion—both due, apparently, to syncope following slight exertion, such as getting out of bed. In neither case, however, was the amount of fluid excessive. Weil, who has studied carefully this accident, concludes as follows: (1) That it may be due to thrombosis or embolism of the heart or pulmonary artery, œdema of the opposite lung, or degeneration of the heart muscle; (2) such alleged causes as mechanical impediment to the circulation, owing to dislocation of the heart or twisting of the great vessels, require further investigation. It occurs more frequently in right than in left pleurisies, and the effusion is usually serous. Death may occur without any premonitory symptoms, usually during some movement or effort.

III. Purulent Pleurisy (*Empyema*).

Etiology.—Pus in the pleura is met with under the following conditions: (*a*) As a sequence of acute sero-fibrinous pleurisy. It is not always easy to say why, in certain cases, the exudate becomes purulent. It rarely does so in the acute pleurisies of healthy individuals. In children many cases are probably purulent from the outset. Aspiration, which is said to favor the occurrence of empyema, in my experience does so very rarely. (*b*) Purulent pleurisy is common as a secondary inflammation in various infectious diseases, among which scarlet fever takes the first place. It has long been known that the pleurisy supervening in the convalescence of this disease is almost always purulent. It should be remembered that it is latent in its onset, and that there may be no pulmonary symptoms. The pleurisy following typhoid fever is also usually purulent. Other infectious diseases—measles and whooping-cough