We have had in the wards lately a large number of cases of pleural effusion. I have before me the notes of no less than sixteen cases. It is obvious that I cannot enter into the details of all these, but can only take from them such points as I think may be useful and interesting to you.

It is always advisable, I think, to trace symptoms back as far as we can to their cause, and find out why a given occurrence takes place. A collection in the pleural cavity occurs just as a collection of fluid does elsewhere, for the very simple reason that more fluid is poured into it than passes out from it in a given time (Fig. 1, d). If a quantity of liquid be poured into a funnel, care being taken not to pour it in more quickly than it can run out, no fluid will accumulate (b); but if the fluid be poured in more quickly than it can run out (a), or if the rate at which the fluid can run out be lessened, accumulation will occur. An accumulation of fluid in a serous sac, such as the pleural cavity, may take place, either by an increase of the rapidity with which fluid is poured in, or a...
decrease of the rapidity with which fluid is absorbed, or by both at the same time (Fig. 2).

Now, as you know, the pleural cavity in healthy men is not a cavity at all. It is called a cavity, because it can be made into one, but it is not a cavity in the ordinary sense of the word, for the costal and the visceral pleurae come close together without leaving any space between them. Their surfaces are kept moist by a certain amount of serous fluid. In order to keep this serous fluid fresh, a little is poured out and a little is absorbed regularly; so that, although there is only a very small quantity, just enough to moisten the surface of the pleura, yet this is constantly renewed. The way in which it is renewed is this: The lymphatics take up from the pleura the fluid which is poured into them from the blood vessels. The lymphatics are perhaps more easily shown in the central tendon of the diaphragm than they are anywhere else; and a beautiful injection of the lymphatics in the central tendon of the diaphragm may be obtained by taking a dead rabbit, cutting it across about the middle of the abdomen, and suspending the upper

![Diagram](image)

Fig. 2.—Diagram of the relations of lymph spaces and blood vessels.

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Fig. 3.—Section of the central tendon of the diaphragm in the rabbit. a, peritoneum; b, tendinous fibres in cross-section; c, circular fibres; d, the pleura; e, peritoneum stretched over a full lymph space; f, peritoneum lying in an empty lymph space; g, blood vessels.—After Ludwig and Schweigger-Seidel.

part of the body in the way you see here, so that the diaphragm forms a sort of hollow cup, with its concavity directed upwards. Then, if some Berlin blue be poured in, and the movements of artificial respiration be kept up in the lungs, so that the diaphragm is kept constantly moving up and down for two or three hours, the Berlin blue becomes taken up, and passes into the lymph spaces;
so that a most lovely injection is frequently obtained. Our experiment this morning has not succeeded so well as many that I have tried; but you can see that some of the Berlin blue has been taken up. There may not unfrequently be seen in the central tendon a most lovely radiating structure, the blue representing the lymph channels; and it has been found that in the diaphragm there are certain stomata, or openings, very much resembling those found on the under surface of leaves (Fig. 4). Through these stomata the particles of Berlin blue are taken up, and the use of these particles is to show where the fluid is gone; without their presence we could not trace the fluid. By the same means we are able to trace the passage of fluids from the pleural cavity into the lymphatics, and it has been shown (Fig. 5) that in the interspaces between the ribs, on the inner side of the thoracic wall, the lymphatics are to be found in great abundance. And if a section be made of the pleura, the lymphatic spaces are seen to present very much the appearance which I show you in the diagram (Fig. 6). These lymph spaces are kept in a state of movement during ordinary respiration, and every breath that is drawn has a pumping action upon the pleura; so that the more a man breathes, and the more the fluid is poured out to keep the lungs well lubricated, the more rapidly is the fluid pumped up from the pleura into the lymphatics of the chest and so into the general circulation. Perhaps this fact does not bear so much upon the cases that we are going to deal with first; but it does bear to a very great extent upon one of the cases at least that we shall have to take into consideration later on, namely, the effusion of fluid into the pleural cavity in the later stages of heart disease.

Now, in ordinary acute pleurisy, it would appear that the cause of the effusion is rather that fluid is more quickly effused
than that absorption is diminished. In order to understand how this effusion of fluid takes place, we have to take into consideration the blood vessels from which the fluid passes out. You know that the capillaries consist of numerous cells, closely applied to one another so as to form a complete ring. These cells, however, have been stated to consist of two parts. A sort of ground substance is said to lie next to the lumen of the capillary; and the cells, when the capillaries are not dilated, are in constant contact with one another; but if the capillaries are dilated, they are drawn apart from one another; so that one can readily see why fluid should pass out quite easily from a dilated capillary. In fact, the puzzle is, if the capillaries are dilated, and the edges of the endothelial cells are drawn apart, why does not the whole of the liquid in the capillaries leak out? The reason alleged is that the outer part of the cell consists of protoplasm, and while the inner part of the cell may be drawn apart from its neighbouring cell, leaving a gap between, the protoplasm, being elastic, tends to stretch; so that a layer of protoplasm renders the wall of the capillary still continuous even during dilatation. We have, however, a difference in filtration through the capillary wall when it is in a state of contraction, and when it is in a state of dilatation. In contraction, we have a double layer of substances, the ground substance and the protoplasm in the capillary wall, through which the fluid must filter before it can get into the tissues below. But, in the state of dilatation, we have only got the protoplasmic layer acting as a filter; and therefore, as we can well imagine, not only is there likely to be a difference in the quantity of the fluid effused, but there is also likely to be a difference in the quality of the fluid effused from the capillaries into the tissues. Now, as you all know, one of the first circumstances that occur when the pleura is inflamed is that the capillaries become dilated; so that, if the pleura be examined shortly after an irritant has been applied—and this can be done readily in the case of experiments on animals—it is found that the capillaries are much dilated, and that the pleura itself assumes a much more ruddy colour than in the normal condition. At the same time, it loses its smooth polished surface
and becomes slightly rougher, so as to present an appearance, not like a piece of ordinary window glass, but rather like a piece of frosted glass, or glass upon which one has breathed. This process may not go on any further, and a retrogression may occur; so that the pleura may again return to its former condition without anything more happening. On the other hand, however, you may get the two surfaces of the pleurae becoming adherent without the effusion of any liquid; but in many cases liquid becomes effused, sometimes to a very considerable extent. The liquid frequently is serous, and generally contains fibrin also. The amount of fibrin varies in different cases; sometimes it is very considerable, in other cases it is only slight. This fibrin, however, is frequently deposited upon the surfaces of the pleurae, giving rise to a thick, rough, membranous-looking substance; and by and by, after the fluid has been absorbed, you may get a large thickened mass of fibrin between the two layers of the pleurae, and in the interstices of this a quantity of fluid is frequently enclosed.

Now, one of the first causes of pleurisy may be injury to the side; a blow, or something of that sort. In one case we had in Hospital, there was a certain suspicion of a blow having originated the pleurisy, although it was properly aided by the effect of a chill. In many other cases, however, we find that a chill gives rise to the pleurisy, and we are not able, in some of those cases at least, to trace the presence of any other factor.

Many means have been tried of late years to find out the presence of microbes in addition to the chill; for the presence of pleurisy has been looked upon as due to the microbes finding their way into the chest, and having their vitality increased, or rather, perhaps I should say, their power for mischief increased, by the vitality of the tissues of the pleura being lessened by exposure. I do not think, however, that this has certainly been proved, and I think in the meantime we may assume that pleurisy may be due simply to the effect of chill. The place where the chill is very likely to take effect is just the place where you would naturally imagine it, namely, that part of the chest that is not well covered by muscle, just between the latissimus dorsi in the back and the pectoral muscles in front. A chill may take effect much more readily here than in those parts which are better covered.

The symptoms that we get after exposure are that the man gets a rigor, or shiver, but instead of my detailing the symptoms to you in a general way, I may perhaps simply read a case to you, because I think you will find the symptoms all beautifully given in it. We will take the case, for example, of John R. He was admitted on 19th February 1896; his age was 35, and he was a furniture porter. The history of the case is, that he was well till fourteen days before admission; then one day, while at his work lifting furniture, he became very hot. When he got home he felt very
chilly and began to shiver, but there was no very distinct rigor. The same evening he was seized with pain in the right side, and a cough. The pain was very much worse whenever he took a breath, or when he coughed. Notwithstanding this pain and the cough, he continued to work for two days, and then he stayed at home for five or six days, not going to bed, but simply sitting about the house; and he attended at the surgery here for a week. The pain which seized him so violently at first had almost gone for the last three or four days before admission. When he was admitted, his respirations were 36. He lay upon the right side, the side in which he had had pain, but from which the pain had now gone. He had a troublesome cough, but no expectoration. On examination of the chest, the movements of the right side were deficient; the vocal vibration was nearly absent, and, behind, the vibration was absent also. The breath sounds were very much weaker on the right side; there was slight bronchial breathing at the angle of the right scapula. The percussion note was nowhere resonant, and there was absolute dulness all over the region below the third rib in front, and below the spine of the scapula behind. The pulse was 100 and regular, the apex beat was in the fifth space; but whereas it ought to have been about half an inch to the inside of the nipple line, it was in the nipple line; it was therefore displaced outwards. I should also mention that aegophony was found opposite to the angle of the scapula on the right side.

In this case, then, we had a history of pleurisy. There was the cough, the pain in the side, and also the raised temperature. The pain had disappeared, and we found on admission dulness on percussion—dulness of an absolute nature—over a great part of the right side; the breath sounds almost absent, and the vocal fremitus almost, or entirely gone. These, then, were the signs of pleural effusion. The disappearance of pain pointed to effusion, for at the beginning of the disease the two surfaces of the pleurae had rubbed against one another, giving rise to pain, but as the fluid became effused and the surfaces became separated from one another, so that there was no longer any friction, the pain had gone. An exploring needle was inserted in the post-axillary line, in the seventh space on the right side; clear fluid was drawn off, and so an aspirating needle was put in at the same spot, and 13 oz. of slightly opalescent fluid were removed. After the removal of this quantity of fluid, the dulness quite disappeared, the voice and breath sounds came back, and the vocal vibrations could again be felt all over the right side; the temperature still remained up, but the respiration went down to 18, and both sides of the chest moved well. No evidence of reaccumulation occurred, and the man was discharged on April 22, quite well. After the fluid had been removed there was a certain amount of impairment of percussion over the side of the
Pleural Effusion.

Chest, and there was also a slight increase in the vocal resonance, and the breath sounds were of a slightly bronchial character. The reason of that simply was this, that probably the pleura was still somewhat thickened, and that the lung had not quite recovered its normal elasticity, or its normal spongy character, after the compression to which it had been subjected. In this case, then, we get an example of the signs of pleural effusion and the clearing up of the effusion after the great portion of the liquid had been removed by aspiration.

In another case, that of Walter E., we have also a typical example of pleurisy with effusion, but here there were one or two points that might be interesting to notice. He was well till 5th March; then he got pain on respiration; he was short of breath. On 9th March he went to bed, and he was admitted on 12th March with respiration, 32; pulse, 96; temperature, 99°. All over the front of the chest could be noted a rough friction sound, and what was also more interesting was, that over an area stretching about an inch and a half to the right of the sternum, a rough to-and-fro friction could be heard with every beat of the heart. So that at first sight it might have seemed as if the man had pericarditis as well; but when the lung diminished in size during expiration, this friction completely disappeared, again commencing at the beginning of inspiration. The reason simply was, as you can readily understand, that as the lung with its roughened pleura came across the heart during inspiration, the heart caused a rubbing on the pleura, and so gave rise to the rough to-and-fro friction. Sometimes it is rather difficult to make certain as to whether these frictions are pleural or pericardial, but if that friction had been pericardial, it would have continued whether the lung were inflated or not. In this case aspiration removed 3½ pints of clear fluid, and as the side still remained dull after this quantity had been taken away, the chest was again tapped, and rather more than 3½ pints were removed; so that, altogether, 7½ pints were removed from the chest.

Then we have got one case to which I may refer, of empyema, in which a child 22 months was admitted into “Elizabeth” Ward, and in this case the whole of the chest on the left side was completely dull. But not only was there dulness over the whole of the left side, there was a dulness where no dulness ought to have been—a dulness extending not quite to the right nipple, but still quite to the parasternal line. In this case the heart had been displaced very considerably to the right. An exploring needle showed that pus was present, and as pus is not readily removed by simple aspiration, especially in children, in whom the intercostal spaces are so small, a part of the rib was excised, and 16 oz. of pus were removed, which is a considerable quantity for a child 22 months old. A drainage tube was then put in. The child did very well, excepting now and again when
the exit of pus seemed to be interfered with, and then the temperature rose at once; but whenever the pus got free exit again, the temperature came down. It would seem from this case that pus, if retained at all, has the power of raising the temperature to an extraordinary extent. What the exact constituent of the pus is, I do not think we know at present, but there can be little or no doubt that pus does raise the temperature. The opening in the chest wall gradually healed up, and the child, which was admitted on 14th November with this large amount of pus in the chest, was dismissed on 27th January quite well.

In another case of empyema we found that, instead of our having to empty the chest artificially, the chest had emptied itself, because in the case of Edward B., aet. 35, he brought up nearly a quart of pus on 12th April, about a fortnight before admission. On admission, there was dulness over a considerable area in front; the dulness reached to about the same height behind, and I thought very likely that some pus was still present, but on putting in an exploring needle no pus was to be found. Probably there was no longer any free pus present, but only thickened pleurae with thick fibrous adhesions, and although there might be a little pus in the interstices of the fibrinous matters, yet no pus was free, and none could be removed.

Then there was another case of a boy, aet. 8, who on 7th April got his feet wet and had a rigor. On 15th May he brought up a pint of pus, and on the 16th he brought up a cupful more. There was no definite sign at all in this boy's lung, excepting that there seemed to be a little dulness just below the angle of the right scapula. I thought possibly there might still be some pus there. A needle was put in, but nothing came out, and we have not been able to find any indications of any pus remaining behind, so that in this case the empyema seems to have cured itself by the whole of the pus being ejected through the lung. Then we have some other cases in which the effusion into the pleural cavity has not come on in healthy individuals as in those already discussed, but in persons suffering from other diseases. In the case of Edward S., salesman, the pleural effusion came on in the course of cirrhosis. He had been subject to a cough during the winter for several years. Three weeks before admission he got a pain in the left breast, and the pain was much worse whenever he coughed. On examining the chest, it was found that there was dulness at the left back up to the sixth rib. There was no cardiac murmur, but the liver was enlarged, firm and hard, and there was a cloud of albumin in the urine. He was admitted on 19th March, and on 20th March the pleural cavity was aspirated, and 31 oz. of serum were drawn off. This eased the condition to a certain extent, but it did not cure him in the same way it cured the others, in whom the disease was uncomplicated. More oedema of the legs set in, the surfaces
became cold; there was some cyanosis, and on 3rd April the patient died; death being caused, not by the pleural effusion, but by the dropsy due to the liver and to the condition of the kidneys, as well as to a feeble heart.

In the last case that I shall take up to-day, the effusion into the pleura occurred as a complication of heart disease. This is the case of Daniel M. He had a double aortic murmur, and also mitral regurgitation. He had been well until the beginning of March, when his feet began to swell. He then got shortness of breath, and went steadily downhill. He had a large amount of dropsy in the legs, and no less than 32 oz. of fluid were removed from the legs by the introduction of Southey’s trocars. This eased him a good deal, but he went on getting worse, and finally died. On post-mortem it was found that there was a good deal of fluid in the chest, and this leads one to note that not unfrequently, on post-mortem examinations, fluid is found in the chest when no fluid is noted as having been present during life. Now, you can readily see why this should be the case. A patient is dying of heart disease, and you find that, in spite of everything that you can do for him, he seems to be steadily getting worse and worse. You do not feel inclined to trouble him very much by making him sit up, so that you may auscultate and percuss the back. Moreover, in private practice more especially, you may find that not only are you unwilling, but the patient himself objects and the friends object; and they say, “The doctor is making the patient sit up and doing him harm, simply for the purpose of satisfying his own curiosity.” There is therefore a great temptation to allow the patients to lie quiet and not disturb them when they are dying of heart disease; and yet perhaps, in many instances, we ought to make them sit up, or at least to lie round on the side; so that one could examine the chest, because even although one might not be able to save them, one might be able to prolong their lives by aspirating the chest and removing the fluid which was accumulating.

I mentioned at the beginning of my lecture that the bearing of the experiment that I wanted to show you upon the accumulation of fluid in the pleural cavity had more to do with the accumulation consequent on heart disease, than on the accumulation which occurs after an acute attack of pleurisy. For you can easily understand that if you have got a heart failing, the valves of the right ventricle becoming incompetent, and backward pressure upon the capillaries everywhere, you are likely to get an accumulation of fluid, not only in the lungs, but in the pleural cavity. You are likely to get a larger secretion of fluid, and at the same time a smaller absorption of fluid. Moreover, the very illness of the patient which makes him lie quiet on his back, preventing him from breathing deeply, tends to stop those very movements of the chest wall, which, as Ludwig and Dybkowski have shown, tend to cause an aspiration of liquid from the pleural cavity; and
in this way you are apt to have the last stages of heart disease accompanied by effusion into the pleural cavity. In another case that occurred in "Elizabeth" Ward, there was a small quantity of fluid effusion from the left chest. This also tends to show that one must be very careful in examining, as far as possible, the chest in cases of heart disease. You will remember that in most of the text-books it is stated that very many cases of heart disease in the end have their course quickened by the accumulation of fluid. Now, there are one or two points that will not take more than a minute or two to bring before you, and they may possibly be useful.

In most of these cases, where the pain is great, you will find that putting a few leeches on the surface tends to ease the pain, but not invariably. One of the cases I have alluded to was not relieved, but in many the pain was rapidly relieved by the application of half a dozen leeches. Half a dozen, more or less, is an average number, but if the pain is severe, a dozen will lessen it, one might say, in almost a miraculous way. I remember seeing one private case, in which every breath that the patient took terminated in a shriek. The pain was so excessive, that each time that the breath was taken it was not passed out again as ordinary expiration, but simply came out as a shriek, and the scene was a most painful one to behold. After the application of twelve leeches the pain disappeared completely. I daresay some of you have had occasion to watch the disappearance of pain in the Hospital, under the application of leeches. I do not know exactly how leeches do act, whether it is simply by abstraction of blood or not, but that they do relieve there can be no doubt.

As far as medicine is concerned, in most of the cases we have used very little medicine. The chief medicine was the very old-fashioned drug, acetate of ammonia, which has the power of lessening the temperature by increasing the sweat; but it does not pull down the temperature in the same way that the more modern drugs, antipyrin, phenacetin, and antifebrin do, and so there is little or no risk of its having any effect in causing collapse. Ammonia, per se, is to a certain extent a respiratory stimulant, although its effect as such is not so well seen in the acetate as it is in the carbonate. In cases where the respiration tends at all to fail, then you may give the carbonate of ammonia; and you may even proceed to give such a drug as strychnine, but in most of the cases of simple effusion we have not used this. In the cases of effusion in cardiac disease, you have recourse to a great number of drugs. You may give strychnine and caffeine and digitalis, or other cardiac stimulants and tonics, and it may be necessary also to remove the fluid from the chest as well.