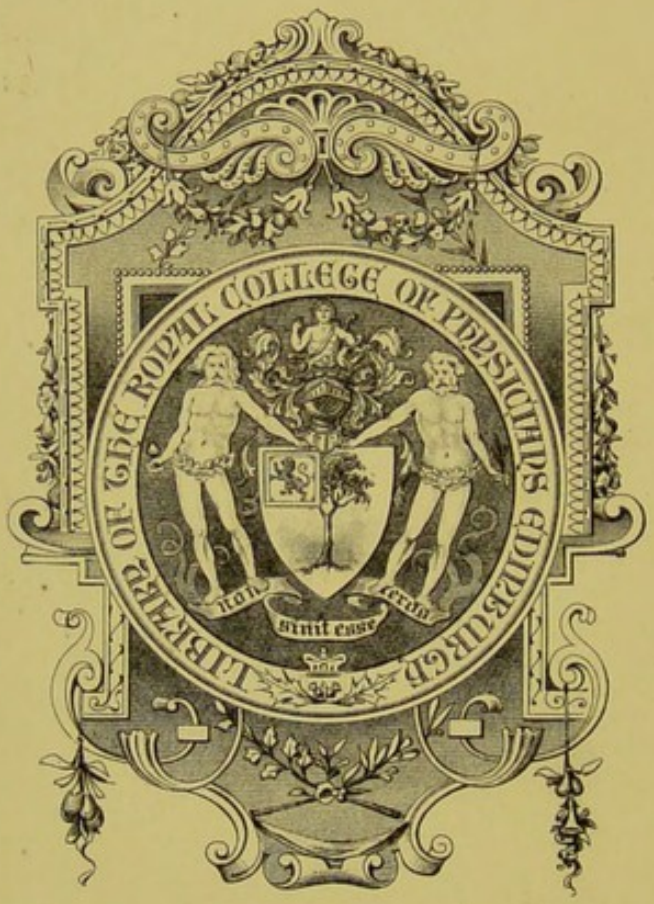
Pleurisy : including empyema and bronchiectatic conditions / by Alex. James.



PLEURISY

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INCLUDING

EMPYEMA AND BRONCHIECTATIC CONDITIONS

BY

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WITH ILLUSTRATIONS

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PREFACE

IN this book on the etiology, symptomatology, and treatment of Pleurisy, I have endeavoured to put into words a number of the ideas on the subject which I have been led to form during an experience of some thirty years of hospital and private practice, and a like period of systematic and clinical teaching in medicine. These ideas I have striven to describe as a series of pictures of the disease, in the various manifestations in which it has from time to time presented itself to me.

It may be at once remarked that any reader who considers full references to medical literature essential in a medical book need not read further, for it has been no part of my object to attempt a narration of all that has been placed on record on this subject during those years. Rather has it been my endeavour to hand on the broad general truths regarding this disease which the generation to which I belong has inherited, along with what little in the way of explanation, illustration, and augmentation my experience and reading seem to me to be able to add.

That the descriptions fall far short of what they ought to be as regards completeness, I am fully cognisant. That such as they are they may prove to have been efforts made towards approaching the truth, I earnestly hope time will show.

Before each individual the addition made by his own

generation to the ever-growing sum of medical knowledge naturally tends to loom unduly in prominence and in magnitude. Yet, keeping this source of error in perspective well before us, when we reflect that we in our times can chronicle the works of a Pasteur and a Lister, we can feel that in this respect our generation has reason indeed for genuine self-congratulation. But there is something further, of which we must never be forgetful. This is, that just as the sum of the knowledge of a past generation is overshadowed by that of the present, so shall the knowledge of the present be overshadowed by that of a future.

I say this in no carping spirit, but in all humility; because I feel that—possibly in verification of Auguste Comte's trenchant saying, that great men are resembled by their disciples in nothing but their errors—our generation has had, as regards micro-organisms, to witness the attribution to them as a whole, and to certain of them in particular, of powers and potentialities as regards disease which, *per se*, I hold they do not possess. I have always been one of those who maintain that whilst the detection of a germ in a disease process is important enough, the interpretation of the conditions which have permitted it to invade a tissue, and to carry out there its morbid effects, is immensely more so.

The story of this phase of disease causation has yet to be read; at present, though written indelibly by Nature's hands, it is undecipherable by us. That it will be read by those who follow is certain, and I feel equally assured that its reading and interpretation will proportionately benefit mankind. By bringing home to him more emphatically, as I feel sure it will, the recognition that in reality the germ is of God, and that it is only its virulence, or the susceptibility to it, which is of man, it will correspondingly bring home to him the realisation that this virulence or

susceptibility is really the all-wisely ordained consequence to mankind of neglect or violation as well as of ignorance of God's laws.

Lastly, in connection with this book, I have gratefully to acknowledge the debt I owe to the older writers—to Graves and to Stokes, to Louis and to Laennec, and to the more modern authors, Copeland, Trousseau, Rokitansky, Rindfleisch, Lebert, Ziemssen, Walsh, Wilson Fox, Eichhorst, Garland, Donaldson, Douglas Powell, Bouveret, and many others. To many writers of the present day I have endeavoured to express my indebtedness on the pages themselves.

ALEX. JAMES.

14 RANDOLPH CRESCENT,
EDINBURGH, *June* 1911.

CONTENTS

PLEURISY

CHAPTER I

ETIOLOGY: ANATOMICAL AND PHYSICAL CONSIDERATIONS

In pleurisy, whatever be the manner in which it has been induced, the main pathological changes in connection with the pleura and lung are—hyperæmia, with infiltration of the serous and subserous tissue, proliferation of the epithelium and of the protoplasmic connective tissue corpuscles, and effusion of inflammatory lymph; all resulting in the formation of an inflammatory false membrane on the pleural surface, and in the collection of an inflammatory fluid in the pleural cavity. As this fluid increases in amount, space for it is made mainly by the collapse of the corresponding lung, the inflamed pleural surfaces being thus separated. Sooner or later the inflammatory process terminates, and the exudation of inflammatory lymph ceases. Reabsorption of the fluid then occurs, in part through the blood-vessels, and in part through the lymphatics; the numerous entrances to which, in the form of stomata, exist upon the intercostal and also upon the pulmonary and diaphragmatic pleuræ. With the reabsorption of the fluid the lung expands. In favourable cases the pulmonary and parietal pleuræ are soon again in close contact, the thin, or perhaps rather the thinned, layer of false membrane between them becoming organised, and serving to form more or less firm adhesions.

In less favourable cases the processes of fluid absorption

and lung expansion do not work together so smoothly and uninterruptedly. As the result of delay in the fluid absorption process, permitting the lung to be held down by glued-together air cells and bands or layers of false membrane, the absorption of the fluid is not accompanied by a corresponding expansion of the lung. There results, therefore, a drawing together of the surrounding parts, viz., chest-wall, mediastinum, and diaphragm—a thickening and fibrosis of pleura and false membrane—and with a fibrosis of corresponding parts of the lung substance itself, a traction upon it outwards, so as to produce emphysema and dilatations of the bronchial tubes.

The degree to which all those morbid processes are manifested varies within very wide limits, as will be seen later on. What concerns us, however, at present, is as to how they can be explained anatomically and physically; and as the above epitome of the main pathological changes in pleurisy arranges itself naturally under three heads, viz., (*a*) pleural inflammation, (*b*) fluid effusion, and (*c*) fluid reabsorption, it will be most convenient to consider the explanatory data, which a study of the anatomy and physics of the lung afford in the same order.

(**a**) **Pleural Inflammation.**—The first point, therefore, which seems to require notice is the *locality* or *site* at which the pleuritic process is most prone to manifest itself. In the vast majority of cases of so-called idiopathic pleurisy this is in the lower and lateral region. As the patient says, the pain is in his side, and it is here that the characteristic friction sound is to be heard most distinctly and loudly. Of course, in secondary pleurisies the locality may be very different, but this can be easily explained. Thus, tubercular deposit at the lung apex readily involves the pleura and causes an apical pleurisy, and the great frequency of adhesions at the apices and posterior borders of the upper part of the lungs corresponds with the tendency of phthisical lung disease to manifest itself at the apices and extend along the

posterior borders. Farther, the virus from a tubercular peritonitis may pass through the diaphragm and cause a diaphragmatic pleurisy, and malignant disease of the mammary gland or axillary glands readily involves those parts of the pleura which are in closest proximity. At present, however, we have to do with so-called idiopathic pleurisy, and the question again is, Why should it be most common at the lower and lateral regions of the chest?

One explanation is, that it is here that the chest-wall is thinnest, and consequently the great irritant cold will here best be able to exercise its injurious effect on the underlying pleura. Certainly all of us who have daily opportunities of examining chests can understand the importance of this explanation, for we know that this is, even in not very well-nourished individuals, the only place where ribs and interspaces are not rendered invisible by overlying muscle. Against this theory, however, it may not unjustifiably be argued, that the effects of cold are not necessarily manifested at the locality exposed; that inflammation of another serous surface, the peritoneum, can seldom be ascribed to cold; and, that in women, in whom the acknowledged evils of low-bodied dresses have to be considered, pleurisies show no special tendency to affect the upper part of the chest. Still the theory is one which deserves notice.

But a consideration of the movement of the lung in the pleural cavity gives us more important information on this question of locality. Thus, if in a living animal pleural windows be made, *i.e.*, if small portions of skin, fascia, and intercostal muscle be removed at different parts of the chest, so that through the transparent costal pleura the movement of the pulmonary pleura can be demonstrated, it will be seen that the amount of this movement is greatest at the lower and lateral regions, and becomes gradually less and less as we pass upwards or backwards, so that the apex and posterior border of a lung are practically fixed points. Hence the part

of the pleura where symptoms and physical signs show pleurisy to be most common is that part of it where this movement of the pulmonary against the costal pleura is most extensive. It may be suggested, therefore, that the correspondingly great stretching and relaxing of the pleural membrane which this implies will render it at this locality specially liable to be affected by morbific influences.

What is probably, however, of as much importance in connection with the answer to the question why pleurisy is specially prevalent at this lower and lateral region of the chest is the fact that, as the result of the greatest movement being at this locality, a pleuritic process once originated will here have a special tendency to be intensified and to spread.

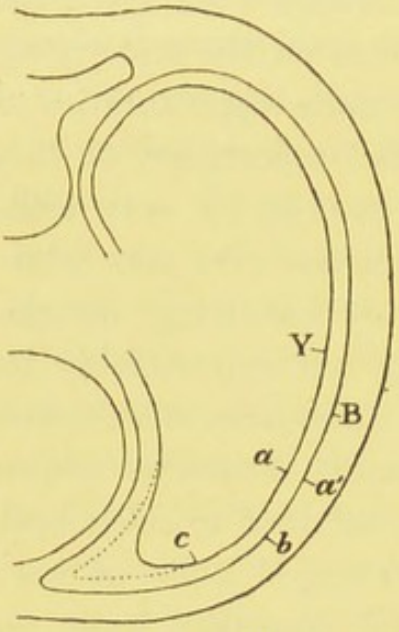


Diagram I.

This has been well shown by Rindfleisch,[1] and can easily be understood from Diagram I. This diagram, which represents a section through the chest about the level of the sixth rib, shows the lung in the position of expiration, the area included by the dotted line representing its increase in inspiration. Suppose that at the point *a* on the pulmonary pleura we have a focus of inflammation. During expiration it will be opposite a^1 on the costal pleura, and will infect it at that point, but during inspiration it will be moved forward, and will infect the costal pleura at *b*, and with the next expiration a portion of pulmonary pleura further forward will be opposite to *b*. This in turn will be infected, and with the next inspiration the costal pleura opposite *c* will be involved. In time the pulmonary pleura at *c* will become affected, and

[1] Rindfleisch, *Pathological Histology* (New Sydenham Society), vol. i., p. 307.

so with the respiratory movements the inflammatory process will be transmitted forwards. Similarly, it will be transmitted backwards, B, Y being with the respiratory gliding of pulmonary and costal pleuræ successively affected.

Seeing, then, that this movement spreads the pleuritic process, it follows that where it takes place to the greatest extent the pleuritic process must be most marked and severe. A small focus of pleural irritation, which at any part of the apex or posterior border of the lung would be able to induce but slight local irritation and inflammation, has thus much greater potency for harm if situated at any part of the lower and antero-lateral regions.

The great proneness, therefore, of pleurisy to affect the lower and lateral portions of the lung may be thus ascribed to the great amount of respiratory expansion and recoil rendering the parts there more vulnerable, and tending to a correspondingly great extent to spread and aggravate any morbid change in the pleura which may have occurred. Conversely, the fact that in a phthisical lung the pleuritic adhesions almost always present are so much limited to the parts most affected by tubercular disease, *i.e.*, to the apices and posterior borders of the upper part, is to be ascribed in great degree to the very slight amount of movement of lung in those regions. Further, it is justifiable to consider that the relative absence of symptoms and physical signs (pain and friction sounds), in such phthisical pleurisies, may be similarly explained.

(b) **Fluid Effusion.**—As fluid begins to collect in pleurisy it first fills up the so-called complemental space of the thorax, *i.e.*, the space between the diaphragm and costal wall into which the lung passes at each inspiratory increase. As it accumulates, more and more space is allowed by the progressive collapse of the lung. But now it is to be remembered that this collapse does not take place to an equal extent in all directions, but that it is most marked in the lower and

antero-lateral regions, where, as we have just seen, lung movement is greatest.

Thus in Diagram II., which represents the lung looked at (*a*) laterally and (*b*) from the front, the greatest amount of collapse is seen to be from the lateral, anterior, and lower portions inwards, backwards, and upwards towards the lung root. Hence, as a lung collapses from effusion in the pleural cavity, the space formed by the separation of the pulmonary

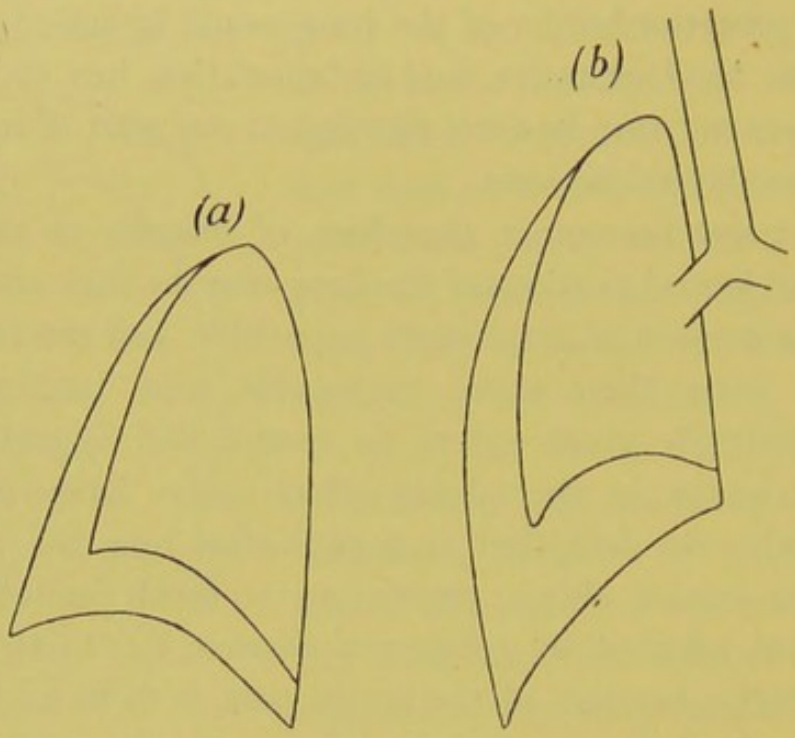


Diagram II.

from the costal and diaphragmatic pleuræ, is peculiarly shaped. Looked at in section from the front (Diagram III. (*b*)), its base is formed by the diaphragm, its outer border by the costal pleura, its apex by the junction of costal pleura and lung, and its inner, least regularly shaped border, by the lung and mediastinum. Looked at next from the lateral aspect (Diagram III. (*a*)), the upper limits of this space formed by the junction of pulmonary and costal pleuræ, is arched, the arch being highest laterally and lowest posteriorly. Hence then as fluid collects in the pleural cavity, its surface is not a level one, as that of fluid in a jar, but a curved one.

This "curved line" was well demonstrated by Garland of New York by injecting warm fluid cacao butter into the pleural cavities of animals and of man after death. On opening the pleura, after the cacao butter had cooled and solidified, he found its upper surface curved as described.

Garland expressly states that in his experiments "even with injections which filled one-half of the pleural cavity there was little or no intrusion of the fluid between the lung

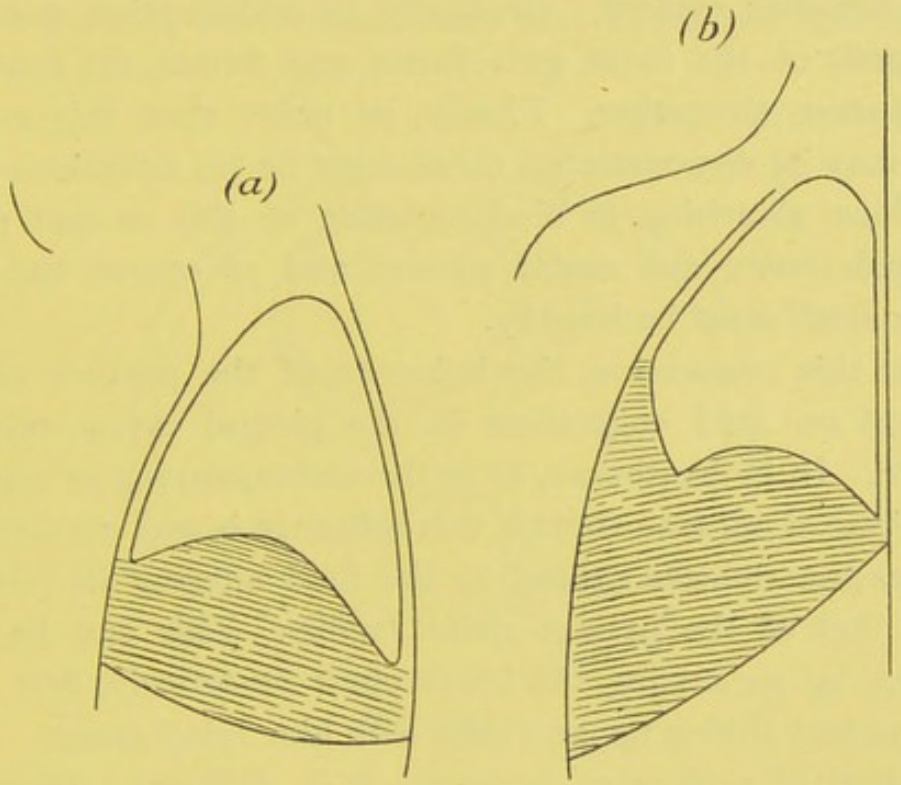


Diagram III.

and the chest-wall, but the model all lay between the lung and the diaphragm." It will be evident, however, that with very large effusions, separation of the lung from the costal pleura, even up to the apex, will occur, and a condition more like that depicted in Diagram III., where this intrusion is shown, will be met with.

This curved line can be demonstrated readily by percussion in pleurisy, whilst effusion is going on. It may be recognised, more easily however in hydrothorax, with the

patient in a sitting posture, but probably most easily of all in pleuritic effusion when reabsorption of the fluid has begun to take place (see p. 70). Of course for its manifestion, certain conditions must be fulfilled. Thus, a certain amount of fluid is required. If the pleural cavity is quite full, the lung is not only collapsed, but is airless and even compressed, and on percussion, dulness is met with all over. With reabsorption, however, resonance begins to show itself over the upper chest anteriorly and posteriorly, and the curved line becomes evident. Gradually as reabsorption goes on, the arch of the curve gets flatter and flatter, till finally it disappears altogether. Finally, in order that this curved line may be demonstrated there must be no adhesions from previous pleurisies to bind together, at this or that point, the pulmonary and costal pleuræ, and of course the lung tissue itself must be healthy.

In this connection, the influence of the posture of the patient on fluid collections in the pleural cavity, requires notice. In hydro-thorax, or in the accompanying or ensuing effusions of pneumo-thorax, this influence is well marked, the percussion dulness caused by the fluid altering its position with that of the patient quite readily. This can be well shown by percussing the lower chest posteriorly, first with the patient sitting up, and then with him lying prone. With the fluid of a pleurisy, however, it is different. Here the pulmonary and costal pleuræ are adherent wherever they are in contact, and although in the early stages when the fluid is increasing in amount, its position as ascertained by percussion may alter to some extent on altering the position of the patient, in the later stages, when reabsorption is occurring, the adhesions between costal and parietal pleuræ are so firm that this cannot take place. The precise position of the curved line, however (Diagram III. (*a*)), may vary to some extent according as the patient has been lying in bed, or moving about during his illness. In the former case, it is

apt, as might be expected, to show the highest portion of its arch more posteriorly than in the latter.

With the accumulation of fluid and collapse of the lung, other important changes are associated. These are, displacement of the mediastinum with the heart to the opposite side, and displacement of the diaphragm with the liver, stomach, etc., downwards. Ordinarily, all these displacements are described as being due to pressure of the fluid, but this requires qualification and elucidation.

As regards the mediastinum and heart, the conditions

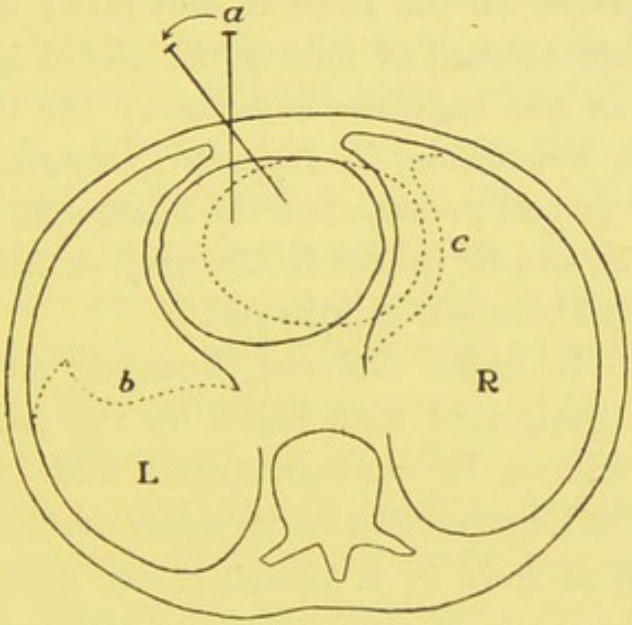


Diagram IV.

pertaining have been demonstrated very clearly by Douglas Powell. Suppose that on the body of a cadaver, we push a long needle (*a*) through the fourth left interspace so that its point enters the heart, as is shown in Diagram IV., which represents a section through the body, about the level of the fifth rib. Suppose next we make a free opening into the left pleural cavity. Air will enter, and collapse of the left lung will occur, as shown by the dotted line *b*. But what is of importance to notice is that with this the heart will be displaced to the right, as indicated by the tilting of the head of the needle to the left. Here nothing has pressed the

heart over, it has simply been drawn over by the elastic retraction of the right lung, as indicated by the dotted line *c*. Under ordinary circumstances, therefore, the heart and mediastinum are, as it were, held in position by the two lungs drawing them each to its own side; if this traction is abolished on one side by letting air in, as in Powell's experiment, or by fluid collecting as in pleurisy or hydro-thorax, the heart is displaced towards the other. It will thus be seen that for this heart displacement, pressure need not be existent at all, and indeed it will be evident that in pleural effusion, pressure on the heart cannot really occur to any extent until the amount of fluid in the pleura is sufficient to cause more or less complete collapse of the corresponding lung. Frank Donaldson, in *Pepper's Cyclopædia*, has stated that there is no real pressure on the heart until the pleura is two-thirds full, and he quotes Rokitansky as holding that to do this, it must be seven-eighths full.

The case is quite different, however, as regards the diaphragm. Supported from below by the pressure of the abdominal viscera, its normal arched form is practically independent of lung traction on its upper surface. Hence an accumulation of fluid in a pleural cavity does not tend to lower it as the result of abolishing lung traction, but does so only when the fluid is sufficiently great in amount by its weight, or by causing positive pressure to press it down. Clinically, the point of importance in connection with all this is, that whereas displacement of the heart and mediastinum will occur even with small accumulations of fluid, it is a common and an early sign of effusion; whilst displacement of the diaphragm, which can occur only when the effusion is considerable, is a less common and a later sign of the condition.

On the other hand, it is to be remembered that with the reabsorption of the pleural fluid, the return of heart and diaphragm to their normal positions takes place conversely.

Thus, supposing we have a chest completely full of fluid, and consequently presenting percussion dulness all over the side, we can usually note that when reabsorption has begun, the first sign of the diminution of the fluid is the rise of the diaphragm. On the left side this will be shown by the ascent of the tympanitic stomach note, as ascertained by percussion, and on the right by the ascent of the lower border of the liver, as ascertained by palpation and percussion. With this rise of the diaphragm, a return of percussion resonance at the corresponding chest apex will soon be associated. But it will be found that not until this resonance has become fairly extensive, and the lung correspondingly re-expanded, will the heart have returned to its normal position. All that has just been said, however, it must be remarked, applies only to cases in which no previous pleuritic attacks have produced adhesions. Where such are existent the whole sequence can of course be altered.

The precise position and relations of a heart displaced as the result of pleural effusion on one or other side, require notice. One might expect, *e.g.*, in left-sided effusions, when this displacement occurs towards the right, that the relatively mobile apical portion of the heart would be more tilted or twisted across than would be the more fixed base. This, however, is not so. Careful examination reveals that on whatever side the effusion may be, the heart with the mediastinum is mainly displaced *en bloc*, and that as a rule no bruit can be heard on auscultation to indicate any twisting of the organ on itself or on the large vessels.

(c) **Fluid Reabsorption.**—But now the subject of the reabsorption of fluid in the pleural cavity requires detailed consideration. In connection with it, the experiments of Dybkowsky[1] are, I believe, well worthy of note. That observer demonstrated, that if watery solutions of grape

[1] Dybkowsky, "Ueber Aufsaugung und Absonderung der Pleurawand," *Ludwig's Arbeiten*, 1866.

sugar or gum arabic, or of water containing in suspension minutely divided particles of Berlin blue, were introduced into the pleural cavity of an animal, these could in time be absorbed. But he also showed, that for the absorption of any of these, the performance of respiratory movements was necessary. Thus, if they were injected into the pleura after death, or into the pleura of a living, quietly breathing animal, little or none was absorbed. Injected, on the other hand, into the pleura of a dead animal, whilst powerful artificial respiratory movements were being induced, or into the pleura of an actively breathing living animal, absorption took place readily. Dybkowsky further showed that whatever increased the elastic traction of the lungs favoured absorption. Thus, when the trachea of the animal experimented upon was connected with an air space in which the pressure was lowered, absorption was increased, while if it were connected with a space under higher pressure, it was diminished or arrested.

In the case of the fluid solutions, this absorption seemed to take place as the result of filtration and osmosis through all the surrounding parts—costal, diaphragmatic, mediastinal, and pulmonary pleuræ—into the underlying blood-vessels and lymphatics. In the case of the solid particles of Berlin blue, however, it was different. In all his experiments he found that the absorption of these was limited to the costal pleura, and that there it took place through stomata into the underlying lymphatic vessels. These stomata and lymphatics, he further demonstrated, were limited to the intercostal spaces, none were to be found on the portions of pleura over the ribs themselves.

From his experiments, some deductions of practical importance can be drawn. In the first place, as regards the effects of the respiratory movements in pleurisy, we find a good example of what we often see in Nature, viz., that one and the same thing can act beneficially or harmfully according to circumstances. We have already seen how, in the

early stages of pleurisy, respiratory movements will tend to spread and intensify the pleuritic process; we can now see also that in the later stages they will tend markedly to promote the absorption of the effusion. In an early pleurisy, therefore, rest in bed, with a flannel bandage round the chest, will be as beneficial as will walking about, climbing stairs, dumb-bell exercise, etc., be in the later.

The fact further, that his experiments demonstrated that the absorption of minute solid particles from the pleural cavity took place mainly through the stomata and lymphatics of the interspaces of the costal pleura, is of practical importance. We can readily understand that if in a case of pleuritic effusion approximation of the ribs has occurred, these stomata and lymphatics must be more or less injured and rendered incapable of performing their function. Moreover, as in these circumstances the mobility of the side will be correspondingly impaired, the benefit of the respiratory movements will be to an equal extent awanting.

Lastly, his demonstration that reabsorption processes are favoured when the animal is made to breathe an attenuated atmosphere, is of importance in connection with the æro-therapeutics of pleuritic effusion.

What has been said as to the collecting of fluid in the pleural cavity, and corresponding collapse of the lung, enables us to understand that there may be large quantities of fluid in that cavity, and yet that that fluid is under little or no positive tension or pressure. Homolle[1] and others have shown this by connecting water or mercury manometers with the various intra-pleural fluids. In this way what was to be expected was demonstrated, viz., that with sero-fibrinous effusions, the tension was frequently negative, the amount of this negative tension being increased during inspiration and diminished during expiration. Homolle states that in

[1] Homolle, "De la tension intrathrocique dans les épanchement pleuraux," *Revue Mensuelle de Medicine et de la Chirurgie*, February 1879.

his cases of sero-fibrinous pleurisy the greatest negative tension which he obtained was 38 mm. of mercury (about 19 in. water) in inspiration, and 33 mm. mercury (about 16 in. water) in expiration. The greatest positive tension which he met with was 15 mm. mercury.

With empyemas, on the other hand, positive pressures are quite usual. This marked difference between those effusions is explained by the fact that whilst in the sero-fibrinous, any adhesions between the pulmonary and costal pleuræ are usually weak, in the purulent they are usually strong. In this way, with a sero-fibrinous effusion, the lung will progressively collapse with increase in the amount of the fluid, and a positive tension will practically only occur when this collapse is complete. With a purulent effusion, on the other hand, positive tension will occur practically as soon as the space—whatever be its size—hemmed in by adhesions and occupied by the pus, is quite filled. It will be obvious also that the higher the tension of the fluid, the less will it show respiratory oscillations, and *vice versâ*. The important practical point which all this enables us to understand, is that with even large quantities of fluid in a pleural cavity, we may yet find not only no enlargement of the side, nor obliteration or prominence of the interspaces, but even a somewhat retracted side, and drawn-in interspaces.

A further point of interest is as to the amount of fluid which can be contained in the pleural cavity. This must vary with the age, size, etc., of the individual. But in a general way it may be stated that in the case of the adult, the presence of 100 to 120 oz. will mean that a pleural cavity is full, that is to say, that on percussion, dulness will be found all over. With a quantity of about 60 oz., dulness will be met with in front up to the third rib or interspace, with 35 or 40 oz. up to the fifth rib or interspace. With 8 or 10 oz., percussion dulness will be just recognisable about the base or lower axillary region. In my practice, I have frequently

removed quantities of 100 or 120 oz., but the largest quantity, 145 oz., was curiously not in an adult, but in a boy of 13. The case was one of empyema of the left side, and in addition to the side being greatly enlarged, percussion dulness extended from the very apex above to the iliac region below, to which the diaphragm was depressed, whilst the heart was so pushed over, that an impulse could be felt in the right axillary line. With incision and drainage this patient made an excellent recovery. But while 145 oz. was the largest amount of truly pleural contents which I ever removed, still larger quantities have been reported. Wilson Fox[1] has given an interesting list of such cases. The greatest amounts which he quotes—58 pints or 928 oz.—were removed in two aspirations from a case of empyema necessitatis. These quantities of course represented more than the pleural collections, but of intra-pleural fluids he quotes one of empyema, in which 240 oz., and another of sero-fibrinous effusion in which 224 oz. were removed, each at one operation. Wilson Fox also alludes to the very large effusions, and specially purulent ones, which may be met with in children.

In dealing with large watery effusions, a possible source of error as regards the amounts must be contemplated. This is, that during the time occupied by the aspirating off of the fluid, there may be some further oozing into the pleural cavity, so that, even though this cavity cannot be completely emptied, the amount removed by the aspiration may be greater than that which it had actually contained. In cases, for example, of aspirations of quantities of 80 or 100 oz., in which the aspirating needle used is small, and in which the fluid is drawn off slowly, the fifteen or twenty minutes occupied by the operation may give time for this to occur.

A remarkable instance of huge pleural aspiration occurred in my own experience some years ago, and merits record

[1] Wilson Fox, *Diseases of the Lungs and Pleura*, edited by Sidney Coupland, p. 961.

here. It was that of a man, aged 44, a labourer, with a markedly alcoholic history, who was admitted into my ward with slight jaundice, enlarged liver, enlarged spleen, ascites, and effusion into the right pleura. Aspirations of both abdominal and pleural fluids were soon required, and during his five months' residence in hospital, the abdomen was tapped ten times, and the right pleura thirteen times. The average quantity removed from the abdominal cavity was 245 oz., with a maximum of about 300 oz., whilst from the pleural it was 250 oz., with a maximum of 446 oz. The very large quantities of pleural fluid which were being removed soon caused comment, and the interest in the case was intensified, when it became evident that the aspiration of the pleural fluid caused marked diminution in the amount of the abdominal. Indeed, it was found as time went on, that the abdominal cavity could be emptied more thoroughly by aspiration of the pleural, than it could be directly.

The explanation of this, which I was inclined to believe in, was that during the pleural aspirations fluid from the abdomen was being pumped through the diaphragmatic lymphatics into the pleural cavity, by the action of the respiratory movements in the manner of the well-known experiment of Ludwig. But although I believed this process to be at work, I came to the conclusion that there was also in this case some direct communication between the two cavities. Not only was the character of the peritoneal and pleural fluids the same, but by means of long glass tubes acting as manometers, I found that the pressure of the fluid in both cavities was the same. Further, on careful examination of the respiratory oscillations of pressure, it was found that the rises and falls of these were synchronous in both cavities. It will be remembered that normally the intra-pleural tension falls with inspiration, and rises with expiration, whilst the reverse holds as regards the intra-abdominal. The fact that in this case the rises occurred in both with inspira-

tion, and the falls with expiration, indicated some communication as existing between them.

In connection with the subject of the re-expansion of the lung after pleuritic effusion, it seems appropriate to consider here the forces which cause a lung to re-expand in cases in which, as after chest incision for empyema, there is an opening in the chest-wall. It will be evident that through such an opening the outer air can readily communicate with that in the pleural cavity. Hence the negative intra-pleural tension which in a closed pleura is established by the absorption of the effusion, and which, increased with every inspiration, is ever present and active in producing lung re-expansion in sero-fibrinous effusions, cannot exist to anything like the same extent. It will obviously be absolutely non-existent when the dressings are off the wound, and whilst it will be present during the times when the dressings are on, its amount will depend on the degree of air-tightness of the dressings, and on the capability of the pleural membrane to absorb what air has been allowed to pass in. It cannot therefore be regarded as playing anything like the important rôle which it does in an unopened chest. Of great relative importance, therefore, in opened empyemas, are expiratory movements, and especially forced expiratory movements. Coughing will manifestly act beneficially in this way, as will anything which causes the patient to perform expiratory movements against resistance. I remember well the first example of this which I ever saw. It occurred many years ago in the person of a patient who had had an empyema, the opening and draining of which had been rather long delayed. He was fretting at the length of time required for the lung to re-expand and the tube to be removed, and as he was a cornet player in a volunteer band, he asked one day if he might practise his music, in order, as he said, to kill time. Permission was granted him, and in a few days so complete was the lung expansion that the tube was no more required.

Bearing all this in mind, however, it will yet be evident that anything which tends to induce or increase negative tension in the affected pleural cavity, may be of use. For example, the more valve-like the arrangements of the dressings the better. The patient will then, with each expiration, and still more with each forced expiration or cough, drive a quantity of air out of the pleura, greater than will enter it at the next inspiration. Ordinarily in empyema, the dressings act more or less in this way, and usually efficiently enough. To increase this effect, mechanical contrivances, such as Duncan's[1] syphon and Hutton's valve, have long been known.

But now, what from the anatomical and physical standpoints is important, is the consideration of what occurs in sero-fibrinous or purulent pleurisies, when the disappearance of the fluid has been too long delayed. In such cases the lung, or at any rate its lower part, will have been so long in a state of collapse, that its air-cells have become more or less completely obliterated. Moreover, re-expansion will be still further hindered or prevented by the false membrane which, investing it all round, binds it down to the spine and posterior aspect of the chest. Although in time, therefore, disappearance of fluid may occur, re-expansion of lung cannot correspondingly follow. Nature abhors a vacuum, and so the filling-up of the space may be said to occur mainly in one or other or all of the following five ways :—

1. Ascent of the diaphragm.
2. Falling in of the chest-wall.
3. Thickening of the pleura.
4. Dragging out of the lung, so as to produce emphysema and bronchial dilatation.
5. Dragging over of the heart and mediastinum.

Before considering each of those in detail, it has to be pointed out that they are one or other or all to be expected

[1] *Transactions of the Medico-Chirurgical Society of Edinburgh*, Vol. VII. (new series), p. 154.

in a much more marked degree after long-standing empyemas than after long-standing sero-fibrinous pleurisies. In the latter, the normal physical conditions of the lung are much better preserved than in the former. Although sero-fibrinous fluid may have caused more or less complete lung collapse for weeks or months, the air-cells will often be found to have retained their integrity to a wonderful degree, and there is usually no very thick or dense false membrane covering the pleura, and apparently binding the lung down. In empyema, on the contrary, the inflammatory process is much more severe. Alike as regards the overlying pleura and the underlying pulmonary air-cell tissue, it is apt to be much more deeply spreading and destructive, so that the physical conditions induced in lung and pleura, militate against lung re-expansion much more than they do in sero-fibrinous pleurisies. Hence in empyema, these five compensating processes all tend to occur to a correspondingly greater extent.

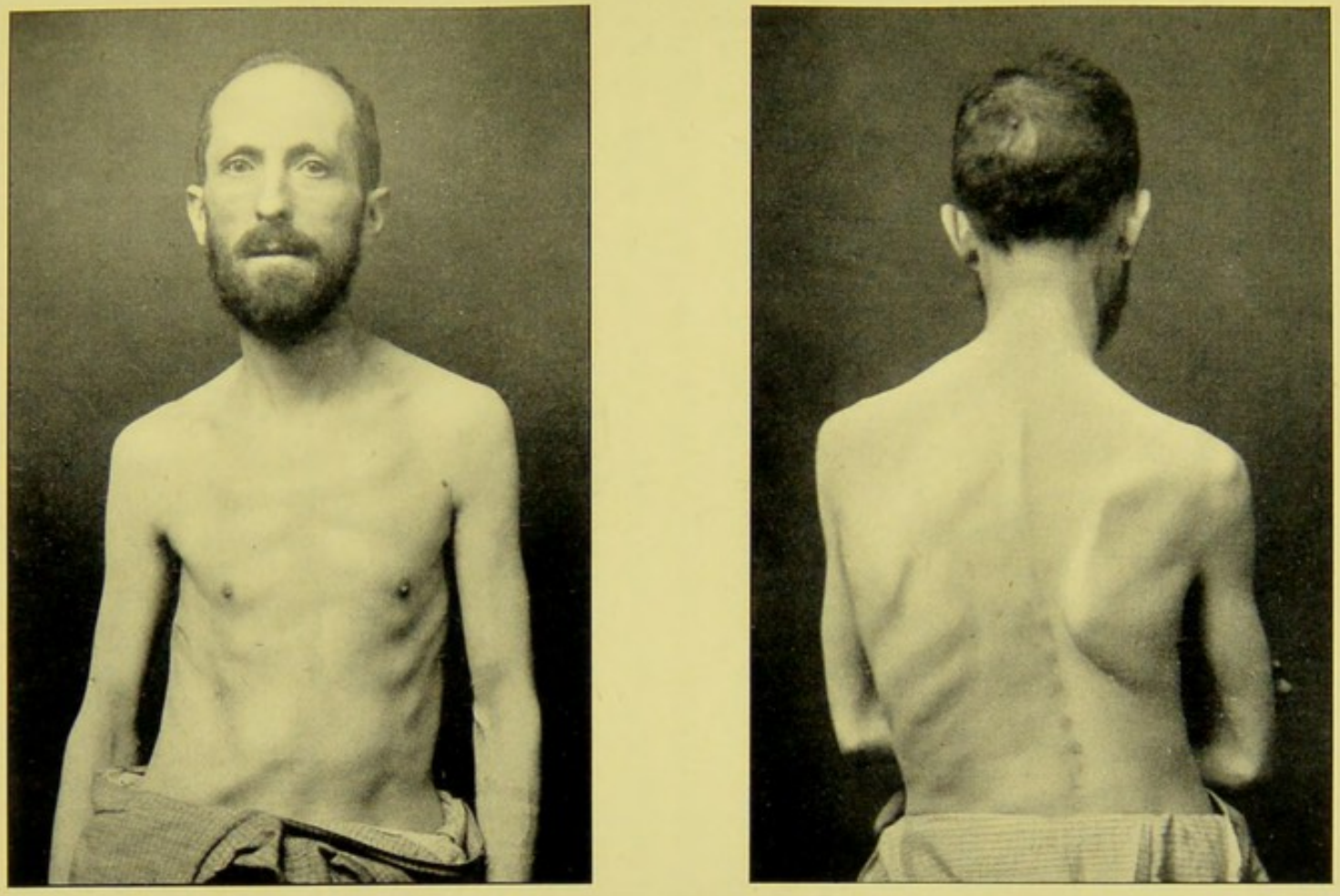
1. **Ascent of the Diaphragm.**—From what has been said at page 10, it will readily be understood that of the five processes mentioned, the first to occur, as a rule, is ascent of the diaphragm. On the left side there is drawn up with it the stomach, on the right the liver. Bearing this in mind, we can understand that on the right side, the ascent is not likely to be quite as readily accomplished as on the left, because on it the solid liver will tend to keep the lower ribs more extended, and in this way prevent its own ascent, in a way which on the left side the yielding stomach cannot do. On either side, however, diaphragmatic ascent may be as high as the level of the fourth interspace, a practical point here being, that in tapping or incising a chest through the lower interspaces posteriorly, it must always be borne in mind that the diaphragm may have risen, or may rise above the level of the opening.

2. **Falling in of the Chest-wall.**—This will occur on either

side of the chest, but for reasons just mentioned, more readily on the left side. As a process, it is associated with descent and approximation of the ribs to one another, with lowering of the shoulder, and with a varying amount of lateral curvature of the vertebral column. The appearance of a chest so deformed is very characteristic, as shown in the adjoining photographs. In such cases, the ribs are depressed and approximated, forming a rigid case for the lung, and the antero-posterior diameter of the chest on the affected side is much reduced, the resulting deformity of the side being rendered the more apparent by the compensating condition of hypertrophous emphysema of the sound lung. When such pathological changes have been caused by empyema, great hardening and thickening and osseous union of ribs, with much formation of new bone, may be found as well.

3. Thickening of the Pleura.—The amount of this also varies in different cases. It may be seen all over the surface of the lung, but for the most part, and for obvious reasons, it is met with mostly over the lower parts. Its presence is due in part, no doubt, to the solid pleuritic exudate; but it is equally certain that the same condition of negative intra-pleural tension which draws up the diaphragm and draws in the ribs, will tend also in large extent to its formation. Analogous in production to this are the cap-like coverings of thickened pleura over the apical part of the lung, caused by the shrinking of an apical cavity. I have seen one such, upwards of half an inch in thickness, covering the upper part of the lung like a dome, the healed cavity which had given it origin being in the very middle of the apex. Pathologically, thickening of the pleura is associated to a greater or less extent with increased growth of the white fibrous tissue of the lung, bands of which can be seen proceeding from the surface into its substance.

4. Dragging out of the Lung, so as to produce Emphysema and Bronchial Dilatation.—The amount of this will



FALLING IN OF THE CHEST-WALL AFTER PLEURISY.

[*Face p.* 20.

vary to some extent according to the length of time during which the lung has been collapsed, but more importantly, according to the amount of air-cell destruction and lung fibrosis induced in it, and according to the strength of the false membrane which prevents the affected part of the lung from expanding. For obvious reasons, it will vary inversely with the degree to which ascent of diaphragm and falling in of chest-wall can occur, and in this way it will be more likely to occur markedly on the right side than on the left. For reasons already stated, and for others referred to in Chapter VI., it does not as a rule occur to anything like the same degree after sero-fibrinous as after purulent effusions.

5. **Dragging over of the Heart and Mediastinum.**—The amount of this varies also in different cases, but there is no doubt that marked and extreme degrees of it occur more readily on the left side than on the right. On the right side, as we have seen, the ascending diaphragm carries with it a solid organ, the liver; and this, while keeping the lower ribs somewhat extended, at the same time mechanically prevents the heart from being dragged over. Moreover, it must not be forgotten, that as has already been mentioned, in reabsorbing effusions the drawing upwards of the liver precedes as regards time the drawing over of the heart. With pleurisies of the left side, however, it is otherwise. There is nothing there to prevent the heart being drawn over, and as the result of an old left pleurisy, an impulse beat felt in the left mid-axillary line is not uncommon.

At the risk of making a digression I would emphasise the fact that in my experience, marked dragging over of the heart to the right has never been the result of an old right-sided pleurisy, but has practically always been the result of apical disease, tubercular or otherwise. That this should be so, is explained when we remember that any shrinking of the apex and upper parts of the lung must exercise a dragging process on the more proximate heart and blood-

vessels, long before it can affect the more distant diaphragm. On one occasion I believed that I had met with an exception to this. It was in the case of a man who presented marked pulsation in the third and fourth right interspaces, with dulness on percussion there, reaching out as far as the right nipple line. This patient had suffered some years before from a right-sided pleurisy followed by impaired re-expansion of lung, so that a diagnosis of heart displacement due to old pleurisy seemed justifiable. Closer investigation showed, however, that the patient was really suffering from an aneurism of the ascending aorta, and I was led to regard the unusually low situation of this aneurism as having been the result of the mechanical dragging downwards of the aneurismal parts caused by the old pleurisy.

Finally, in connection with the anatomical and physical aspects of pleurisy, the vital condition of the lung left by an attack of the disease requires some comment.

In favourable cases of pleurisy with sero-fibrinous effusion timeous reabsorption of the fluid is followed, as has been seen, by re-expansion of the lung, so that pulmonary and parietal pleuræ are soon again in close contact, and except for the existence, in greater or less degree, of pleural adhesions, the patient is regarded as being in a condition of complete recovery. But even in these favourable cases, it may be questioned if the individual is as good as he was before from the vitality point of view.

We all know how frequently tubercular lung disease follows on in the subjects of old pleuritic attacks, and although at present we need not consider in detail the connection between pleurisy and tubercular disease, a reference to the possible influence of pleuritic adhesions in tubercular mischief is appropriate.

Early in this chapter we saw how the respiratory movements, and the presence of stomata and lymphatics over the pleura of the interspaces are important in connection with

reabsorption of fluid in the pleural cavity. It can hardly therefore be doubted, that if we are dealing with a rather extensive pleurisy, which, although the patient has recovered favourably enough, has yet left him with his pulmonary and costal pleuræ bound together by adhesions, we are dealing with an individual in whom lung function cannot be regarded as being normal. Inasmuch as impaired lung function, whatever be its cause, is to be regarded as being a factor in the production of tubercular lung disease, this impairment, induced by adhesions, may be regarded as having a share in the explanation of the frequency with which tubercular lung disease follows pleuritic attacks. Since the use of the X-ray screen has become prevalent in the examination of chest cases, a deficient respiratory movement in the lower chest and diaphragm in the phthisical has become a matter of common knowledge; and although this deficient movement may occur independently of pleuritic adhesions, it is yet probable that such adhesions may at times be its cause.

Still, in connection with tubercular development subsequent to pleuritic attacks, we must not ascribe too much to the mechanical effects of these adhesions. Empyemas are followed quite as markedly by adhesions as are sero-fibrinous pleurisies, and yet, although one at times sees empyema developing in a patient with tubercular lung disease, it will, I think, be admitted that to see an ordinary acute empyema followed months or years afterwards by phthisis, is just as rare as it is common to see this phthisical development occur months or years after a sero-fibrinous pleurisy. By those who look to the organism alone, it will of course be said that this is so because, whilst the majority of sero-fibrinous pleurisies are due to the bacillus tuberculosis, a very considerable proportion of the empyemas are due to the pneumococcus. My own idea is that, inasmuch as practically all of us have tubercle bacilli and pneumococci in our tissues more or less constantly, the differences as regards the

morbid effects produced by these organisms depend really on differences in the resistance power of our tissues. In those of us who are constitutionally strong, a temporary lowering of this resistance power may give a chance to the pneumococcus to assert itself, which it will have no difficulty in denying to the tubercle bacillus. In those of us who are constitutionally feeble, on the other hand, the tubercle bacillus can assert itself with the very minimum of lowering of the general health. It is consequently not difficult to understand why the man who has recovered from a pneumococcus empyema, should so often get fat and well, and remain so for the rest of his life, whilst the man who has suffered from a sero-fibrinous pleurisy should so often date from this permanent ill-health.

Still, sero-fibrinous pleurisies, which leave behind them adhesions, may be caused by or associated with other than tubercular organisms and toxins; and if they occur in an individual constitutionally below par, these adhesions may by their physical effect on lung function, act as a link in the vicious circle chain which prevents the lung tissue nutritive power being able to hold its own against the tubercle organism.

All this, however, requires further qualification. Remembering that anything which entails increased lung function and increased blood afflux may act salutarily in tubercular lung disease, it is quite conceivable that an individual, with a lung maimed not only by pleural adhesions, but by extensive fibrotic and other changes, may, as regards his sound lung, and as regards the unaffected part of his maimed one, be in a better position as regards tubercular development than he was before.

That these pleural and lung changes may possibly therefore present a salutary aspect as well, is quite conceivable (see Chapter on Phthisical Empyema).

CHAPTER II

ETIOLOGY OF PLEURISY—PREDISPOSING CAUSES

Age.—Pleurisy may occur at any age. I have met with an empyema on post-mortem examination in an infant of two months. I have seen a sero-fibrinous pleurisy occur and end in tubercular lung disease in a woman of 82, and I have seen a fœtid empyema occur, and be recovered from, in a man of

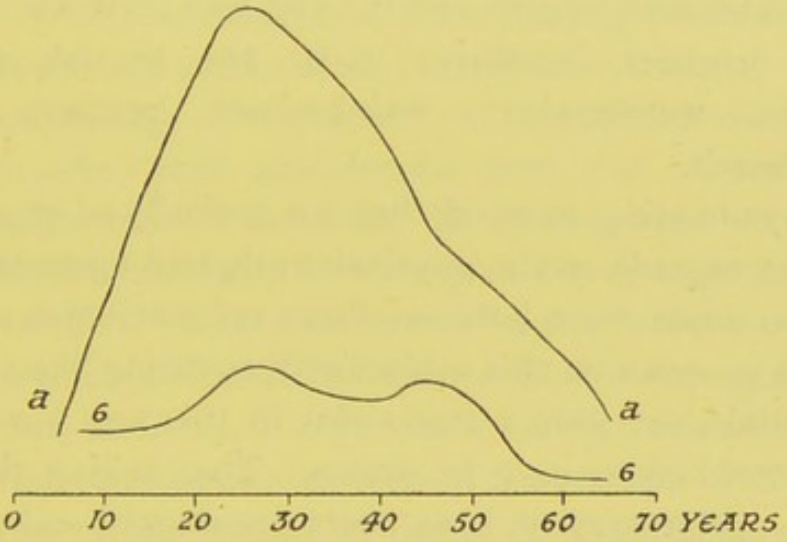


Diagram V.—Curves to show age incidence in hospital cases of (a) sero-fibrinous pleurisy and (b) of empyema.

84. The accompanying curves indicate the age incidence of my hospital cases of pleurisy and empyema. The noteworthy features which these curves present are:—

(1) That the great bulk of both pleurisies occur between the ages of 15 and 55.

(2) That the pleurisies of children are more likely to be purulent than those of adults.

(3) That in spite of this being so, purulent pleurisies yet tend to show themselves at later periods of life than do sero-fibrinous. Thus whilst 20 per cent. of the latter occurred after the fortieth year, 40 per cent. of the empyemas occurred after that period of life.

Sex.—As regards sex in pleurisy, the preponderance of the male cases among hospital patients is marked. Of my hospital cases of sero-fibrinous pleurisy, 75 per cent. were males, of empyema 85 per cent.

Occupation.—In order to consider whether or not occupation can influence this disease, a good plan is to divide occupations into two main divisions, viz., (1) those which entail physical work in the open air, or in open spaces; and (2) those which entail sedentary work indoors.

Under the first head come such occupations as shepherds, outdoor labourers, engine and railway men, cabmen, curriers, brewery workers, butchers; under the second, such as engravers, watchmakers, hairdressers, printers, clerks, factory hands.

Always bearing in mind that the majority of occupations occupy as regards strain, physical work, and openness of air a position somewhere between those two extremes, and that therefore to speak on this subject with anything like precision is impossible, yet from a study of it in this way some broad general conclusions may be drawn. Thus taking the three diseases—pulmonary phthisis, acute pneumonia, and pleurisy, and arranging them as regards occupations in this way, it will be found that whilst pulmonary phthisis shows itself markedly under the second head, viz., indoor and sedentary, acute pneumonia shows itself under the first, outdoor and straining, whilst pleurisy occupies a somewhat intermediary position. Moreover, it has to be added that whilst, as regards occupations, sero-fibrinous pleurisies tend to resemble phthisis, empyemas tend to resemble acute pneumonia. This latter is the more easily understood when we remember that the bulk

of the empyemas which we meet with follow pleuro-pneumonia.

But it must not be forgotten that occupation, as a factor in disease, can appear to have more importance than it really has. Apart from the fact that the same occupation may be followed under house and work surroundings the most diverse from the health point of view, it must always be borne in mind that a man's occupation is largely dependent on his physical and constitutional health. Thus the liability to phthisis or pleurisy of the watchmaker, the printer, or the clerk, as compared with the shepherd, the dock-labourer, or the engineer, may be due, not so much to the occupations, as to the physical and constitutional characteristics of the individual which determined him to follow this or that occupation. Still, inasmuch as it can strengthen by use or weaken by disuse, occupation must have a distinct influence on the resistance power of an individual to disease.

Constitution.—If the subject of occupation in disease must be approached with considerable care, that of constitution necessitates this still more so. In disease, what we mean by constitution is the nutritive or trophic power transmitted from parent to child. This nutritive or trophic power we define as the resistance power of the tissues to disease organisms or toxins, and we know, in a general way, that this power in the child must correspond with that of the parent. If this resistance power in the parents is feeble, this feebleness will show itself in the offspring, appearing either as the same or as some other disease. In the former case we call it heredity, in the latter innateness. Thus, phthisis, rheumatism, diabetes, or epilepsy in the offspring of parents with phthisis, rheumatism, diabetes, or epilepsy, constitutes heredity; whilst phthisis or rheumatism in the offspring of parents with diabetes or epilepsy, constitutes innateness. Much confusion has occurred from time to time in connection with this subject. For example, because a set of statistics may

have revealed that out of so many cases of phthisis, a history of phthisis in the parents is not specially marked ; it has been asserted that family history has nothing to do with phthisis. The error will be quite apparent from what has just been said. Indeed, if any conclusion at all can be drawn from such statistics, it will be one against the infectiousness of phthisis from person to person. But further, it must always be remembered that the resistance power transmitted from parents to children must vary within wide limits, at different ages, at different times, and according or not as disease, strain, misfortune, or accident have or have not made themselves manifest in the parents' lives. In this way, in the same family, we may meet with individuals presenting great differences as regards constitutional inheritance. We must not, on account of this, be led into minimising the importance of heredity, we must simply regard it as indicating our incapacity properly to identify, collect, or interpret its manifestations. Bearing all this in mind, we realise that on this subject we are probably nearest the truth when we treat it in a very broad and general way. I would, therefore, refer only to two points. The first is that the family history in pleurisy and in empyema cases is, on the whole, not so satisfactory in the former as in the latter. The second is that, as age statistics show that empyema, although common in childhood, yet tends to occur at a later period of life than sero-fibrinous pleurisy, to it again must be ascribed a proportionately better heredity. Constitutional vigour, or the want of it, is often denoted not so much by the disease which a person suffers from, as by the age at which he succumbs to it.

Previous Disease.—The main cause of an attack of pleurisy being want of power in the pleural tissue to hold its own against some infection or toxine present in the blood, a study of the diseases from which a patient with pleurisy has previously suffered, is extremely important.

It will, however, at once be evident that, if it is to be of any value, this study must be limited to those diseases only which appear to have a causal relationship to the pleuritic attack. I have endeavoured to do this with my hospital cases, and the following are the results :—

In simple sero-fibrinous pleurisies the previous diseases were—previous attacks of pleurisy, phthisis, pneumonia, rheumatism, bronchitis, heart disease, Bright's disease, liver disease, malignant disease, influenza, asthma, measles, malaria, syphilis, gland disease, bone disease, abdominal tubercle, puerperal and pelvic disease, alcoholism, purpura, otorrhœa. More will be said regarding many of these later on, but meanwhile it is to be observed that some of them, notably heart disease, kidney disease, and liver disease, are more frequent etiological factors than they appear in the records. Every hospital physician, in going round his wards, will in such chronic cases often detect slight pleuritic attacks, which present so little in the way of symptoms, that they would have passed undetected save for physical examination. Moreover, in our ward records, many instances of such pleurisies presenting distinct symptoms are yet not recorded among the records of pleurisy cases. Bearing all this in mind, we need not be surprised at the great frequency with which unsuspected old pleuritic adhesions are found on post-mortem examinations.

In empyemas the previous diseases were—previous attacks of pleurisy, phthisis, pneumonia, bronchitis, whooping-cough, bone disease, child-birth, pelvic and abdominal peritonitis, including inflammations of and about the abdominal viscera, septic wounds, scarlatina, malignant disease, alcoholism, influenza. Concerning some of these also, more will be said later on, but at present a rather interesting comparison can be made between them and the diseases preceding sero-fibrinous pleurisies. Thus, in the sero-fibrinous pleurisy cases, investigation of the previous health history gave

records of previous pleuritic attacks in 15 per cent., of phthisis in 15 per cent., of pneumonia in 4 per cent., of rheumatism in 8 per cent., of alcohol in 4 per cent., and of no previous disease in 32 per cent. A similar investigation in empyemas gave records of previous pleuritic attacks in 16 per cent., of phthisis in 2 per cent., of pneumonia in 26 per cent., of rheumatism in none, of alcohol in 2 per cent., and of no previous disease in 36 per cent. Here the noteworthy feature is the relative infrequency of phthisis and of rheumatism among the empyema cases.

But now a very important point falls for discussion, and this is the connection between pleurisy and phthisis, or perhaps rather the connection between pleurisy and the tubercle organism. Whatever view we may take on this subject from the pathological side, there is no doubt that from the clinical a classification of pleurisies into three main divisions can be recognised. These divisions are :—

1. Pleurisies associated clinically with more or less active lung disease.
2. Pleurisies which occur and are succeeded by tubercular lung disease, weeks, months, or years afterwards.
3. Pleurisies which occur and are not succeeded by tubercular disease, in the lungs or elsewhere.

Let us now consider those in detail.

1. Pleurisies associated clinically with more or less Active Lung Disease.—These are of course extremely common, and in them the resulting pleurisy may be induced either (*a*) by the direct extension of the tubercular process to the surface of the lung ; (*b*) by the transmission of tubercular products along lung lymphatics to its surface ; or, (*c*) as in the non-phthisical, by chill, exposure, or injury setting up a pleurisy which may be associated with the tubercle bacillus, but which is perhaps as frequently associated with some other organismal or toxic agency.

The pleurisies *a* and *b* induced directly by the tubercle

are most common at the apices—that is to say, at the parts where tubercular foci are most common. Owing, however, to the slight amount of movement of pulmonary and costal pleura at this part, such pleurisies cause little or no pain, are little liable to spread, and may be indeed to some extent salutary by permitting of more ready shrinking and cicatrisation of apex cavities, and it may be by preventing their rupture when superficial. The pleurisies *c*, on the other hand, more readily affect the pleura over the lower parts of the lung. Here the respiratory movements of pulmonary or costal pleura being great, chest pain is experienced, extensions of the pleuritic process occur, and the inflammation is frequently followed by effusion, sero-fibrinous, occasionally hemorrhagic, and sometimes purulent. In many instances of this kind, the pleuritic attack proves to be the first thing to draw attention to the fact that the patient has phthisical disease. The doctor is called to the case as one of pleurisy; but careful inquiry into the history elicits symptoms evidencing that tubercular lung mischief had existed beforehand, whilst the absorption, often tardy, of the fluid, corroborates this by revealing as it goes on the physical signs of tubercular apex disease.

2. Pleurisies which occur and are succeeded by Tubercular Lung Disease, Weeks, Months, or Years afterwards.—Clinical illustrations of these will be considered later on. What concerns us at present, however, is the special importance of the tubercle bacillus as a pathological agent in their production.

The recognition of the closeness of the relationship between pleurisy and tubercle is based on (1) the fact that so many cases of apparently primary pleurisy develop tubercle, weeks, months, or years afterwards; (2) the fact that though the pleuritic exudation may not show the tubercle organism, either by staining or by culture, yet inoculations of it into guinea-pigs demonstrate with extreme frequency its

tubercular nature; and (3) that in a large proportion of cases of primary pleurisy the tuberculin reaction, if tried, will be found to be present.

My own view is that inasmuch as the great majority of healthy men and women have the tubercular organism in their tissues, and inasmuch as any lowering of the tissue nutritive power may permit this organism to manifest its harmful effects, a lowered condition of health, with or without an exposure superadded, can readily induce a pleurisy in which the tubercle bacillus plays, of all organisms, the primary part. But in my opinion there are other pleurisies, and foremost amongst them I put the rheumatic, in which the part played by the tubercle organism is a secondary one. That is to say, there are pleurisies set up by non-tubercular organisms or toxins, which having occurred, so lower the trophic power of the pleural and lung tissue that the ubiquitous tubercle organism can then assert itself. True it may be, that though secondary in coming into action, the tubercle looms in our eyes as the all-important element. But this is precisely because in its action it illustrates the truth of what Beau said long ago, that tubercle does not so much begin the malady as it finishes it. As good an example of this as can well be obtained is furnished by tubercular endocarditis. Do we not, for example, every now and again meet with a patient who, after having suffered for years from heart valvular disease, the result of a rheumatic endocarditis, becomes phthisical? Do we not, in his or her downward progress, very frequently recognise evidence of fresh and spreading endocarditis, which proves on the post-mortem table to have been tubercular? In such a case no one will gainsay the assertion that the rheumatic poison has been the primary cause of the valve mischief, and that the resulting maimed condition of the valve tissue has made it vulnerable to the tubercle organism later on. Why, therefore, should it not be admitted that a pleura, similarly maimed by the rheu-

matic or some other poison, should not then be more vulnerable to the tubercle? True it is that although feeble resistance powers to the rheumatic and to the tubercle organism are frequently associated in individuals, yet healthy endocardial tissue appears to be as resistant to tubercle as healthy pleural tissue appears susceptible to it.[1] The difference, however, is only one of degree. Moreover, we know it to be the case that when a patient with confirmed phthisical lung disease develops a pleurisy or an empyema, other organisms than the tubercular are specially frequently associated with that complication.[2]

The fact, lastly, that tubercular pleurisies are so very common—apart from substantiating our view as to the ubiquitousness of the tubercle organism—has no special significance. If the conditions of organisms and soil which give rise to septicæmia were as common as are those which give rise to tubercular disease, septic pleurisies would assuredly be quite as common as tubercular are.

The following is a case of pleurisy which terminated in tubercular mischief, but which was, I believe, primarily due to a rheumatic poison, and associated with purpura.

D. A., æt. 25, a grocer, was admitted to the Infirmary on 11th April 1905, suffering from purpura rheumatica, of some seven weeks' duration. His family history was good, except that two sisters had died of heart disease. His surroundings at home and at work were comfortable, and beyond a slight attack of influenza a year before, he had had no previous illnesses. His present attack had, as stated, begun seven weeks before, with pains and stiffness in the legs, the knees having been specially painful and swollen. A few days after these rheumatic symptoms appeared, he noticed the purpuric rash all over the legs. With rest at home, all these symptoms

[1] Concerning tubercle and rheumatism and the so-called tubercular pseudo-rheumatism. See Dieulafoy, *Text-Book of Medicine*, vol. i., p. 269.

[2] Compare Lindsay, *Diseases of the Lungs*, 2nd ed., 1906, p. 166.

disappeared, and after three weeks he went back to work. After two weeks more, however, he was ill again exactly as at first; he had considerable stiffness and pain in the legs, with effusion into the knee joints, and reappearance of the rash. Finding that improvement was not showing itself very rapidly, he came to the Infirmary.

State on Admission.—Height 5 ft. 7 in., weight about 9 st. He stated that his normal weight was about 10 st. His face was pale; his muscularity was only fair. His temperature varied from normal to 99° F., his pulse about 80 per minute. His alimentary, circulatory, and respiratory systems were all normal. A blood count showed red blood corpuscles 4,000,000, whites about 11,000, h.b. 95 per cent. The blood film showed a normal count. He complained of stiffness in both knees, and there was still slight swelling there. All over the legs many petechiæ were seen. The urine contained a small amount of blood, and a corresponding amount of albumen. His condition rapidly showed improvement, the pulse and temperature kept normal, the petechial spots disappeared, and no fresh ones formed, and the condition of the knees improved. This improvement continued till 26th April, when after a rise of temperature to 100° F., a fresh outbreak of purpuric spots on both legs was observed. Otherwise, however, he was fairly well. On 30th April, however, the temperature rose again, reaching 101° F. This time there was no joint affection, nor petechial irruption, but he complained of pain in the back on both sides, especially the left, aggravated by breathing. He had also a short hacking cough, with no expectoration. On 11th May distinct effusion was noticed on the right side, while on the left pleuritic friction could be heard. On 20th May the right pleura was aspirated, and 25 oz. of hæmorrhagic fluid were drawn off. This fluid showed a large number of lymphocytes in the film; an attempt, however, to obtain from it a culture was not successful. He gradually improved, and on 25th

May he was allowed up. He had then no cough nor spit, but there was still a little fluid in his right side. Above the level of the fluid, some redux friction was met with.

As the days went by, the fluid at the right base showed signs of disappearing, but his general condition and weight were not improving, although his appetite and digestion kept fairly good. Just before his discharge on 23rd June, his opsonic reaction was tried, and found to be ·2 as compared with normal blood.

Shortly after his return home, his cough became worse, and accompanied by muco-purulent spit, which showed unmistakably the tubercle bacillus. Physical signs of apical mischief appeared and spread, and he died in the following February.

In the following case the tubercular supervention was revealed by post-mortem examination.

R. M., æt. 50, a blacksmith, was admitted to Ward 30, 24th November 1894, suffering from pleurisy, and stating that he had been ill for two months and a half. His family history showed no tendency to tubercular disease, and was indeed particularly good. His habits and general surroundings were unexceptionable, and although at his work as a blacksmith he had been exposed to the usual risks of strain and changes of temperature, he had never been conscious of having received harm thereby. He had had no previous illnesses, and gave no history whatever of accidents. His present illness had begun on the 5th of September previously, with shivering, headache, and general feeling of unwellness. On the following day, however, he had gone to his work; but having begun to sneeze, he had suffered from such a severe pain in his right side that he went home and took to his bed. He lay in bed, evidently feverish and ill, and on 19th September, the doctor attending him ascertained the presence of pleuritic effusion on his right side. During all this time he states that he had little or no cough, and only pain in the

side when he took a long breath. Lying in his bed, he did not complain of any shortness of breath, but he was sleepless at nights, and troubled then with sweatings. After about three weeks in bed, he improved so much that he was able to go about. He was taking his food well, and gaining strength, but he noticed that whenever he attempted to climb stairs, or move about more actively, shortness of breath showed itself. He came through to Edinburgh for a change, but still feeling this shortness of breath, he called upon, and was examined by Dr Stockman, who finding the right pleural cavity almost filled with fluid, recommended him to the Royal Infirmary.

State on Admission.—Height 5 ft. 8 in., weight 10 st. 4 lb. —used to weigh about 12 st. His development was good, but his muscularity had fallen away very much since his illness began. His temperature varied between 97·5° F. and 100° F., his pulse between 100 and 108. On physical examination of the chest, we found the following conditions:—There was dulness, more or less complete, with absent fremitus and absent breath sounds over the whole of the right chest, except at its upper part anteriorly. At this area the percussion note was tympanitic, the vocal fremitus was markedly increased. The breath sounds were loud and somewhat harsh, vesicular in character, and at times accompanied by crepitations, and the vocal resonance, like the fremitus, was very markedly increased. Over the left chest, with the exception of somewhat exaggerated breathing, nothing abnormal could be made out. The heart was displaced, so that the impulse beat was in the fourth interspace, about half an inch external to the left nipple line. The liver was displaced downwards about 2 in. below the costal margin, in the nipple line. Examination of the digestive, urinary, nervous, and other systems revealed nothing abnormal. Here a slight digression as regards the physical signs in this case seems appropriate. In it, as in most similar cases of

chronic pleural effusion, one had a great difficulty in being sure as to whether or not the lung apex was free from phthisical disease. The marked increase in the fremitus and resonance, the loud, hollow, and almost bronchial character of the breath sounds, and above all, the crepitations, all suggested this. But these signs, and with them the tympanitic percussion note, can all be brought about as the result of pleuritic effusion alone, and in this case they were so, for, as will be seen directly, post-mortem examination revealed that the lung itself was free from destructive disease (see pp. 76, 79). At the time, however, we, as we were bound to do, contemplated the possibility of there being there apical mischief of some standing.

We prescribed for this patient rest in bed, nourishing diet, counter-irritation in the form of blisters to the side, and a mixture of Potassium Iodid and the Syrup of Iodid of Iron. With a temperature varying between 97° and 100·5°, a pulse of about 104, and respirations about 20, we found at the end of three weeks that the condition of his chest was practically unchanged; we determined, therefore, to draw off the fluid, and bearing in mind the possibility of the presence of apical disease, we did this, not by taking off a large quantity of fluid all at once, but by tappings repeated every seven days, or thereabout, taking away quantities of 10 to 35 oz. at a time. The first tapping was performed on 19th December, and the tappings were continued till 9th February, the total number of tappings being ten, and the total quantity of fluid removed being about 130 oz. During all this period, however, no real improvement occurred, the temperature kept oscillating between 98° and 101°, the pulse between 104 and 108, the patient's weight keeping very much the same, and examinations of the chest indicating only a very slight diminution in the quantity of the fluid. About 15th February he made complaint for the first time of some pain on the left side posteriorly, and his temperature began to rise, oscillating

between 99° and 102°. Some impairment of the percussion note, with what might be either fine crepitation or friction, was made out to be present at the left base, and in a few days distinct effusion there was manifest, with the complaint of some dyspnœa. Although after a few days more the temperature fell to what it had been before, he was never again so well as he had been. He suffered from attacks of dyspnœa, and quantities of fluid, varying from 20 to 60 oz., were removed, at one time from the right pleural cavity, at another time from the left. In this state, gradually though surely losing ground, he continued till about 15th April, when his temperature was noticed to be rising again. On 23rd April, with a temperature varying from 99° to 101°, examination of his chest showed the greater amount of fluid to be in the left pleura. I then determined to have his left chest incised and drained, and this was done on 24th April, a piece of rib being resected, and about 15 oz. of sero-fibrinous fluid removed. Considering his weak state, he bore the operation wonderfully well, but he never gained strength, and his temperature falling to normal, he died on 27th April.

The following was the result of the post-mortem examination :—Left pleural sac obliterated by adhesions of some standing. The lung weighed 2 lb. 4 oz., and showed a chronic pleurisy. There was a small amount of fluid at that part of the pleural cavity corresponding to the lower lobe, which was collapsed. Miliary tubercles, in very great numbers, were found in the thickened pleural membranes and adhesions. The upper lobe was œdematous, and the lower collapsed, both showing scattered tubercles in great numbers. The right lung weighed 1 lb. 12 oz.; the pleural surface was thickened like that of the left lung, but it was more collapsed, a large quantity of fluid being present. The pleuritic membranes and adhesions and the lung itself were studded with miliary tubercles. The peritoneum, liver, spleen, and kidneys all showed miliary tubercles.

In this case, the sequence I believe to have been a right-sided rheumatic pleurisy with effusion, and formation of false membrane, little or no absorption of the fluid, and great increase correspondingly in the thickness of the false membrane. After some months, pleurisy of the left side with effusion, and with similar want of absorption power as regards it, and consequent thickening of the false membrane. In time, acute miliary tuberculosis, due to the weakened tissue nutritive power allowing the tubercle bacillus to take root, the miliary process showing itself first in the lowly vitalised tissue of the pleuritic membrane and spreading thence to the lung, and to the serous coverings of the liver, spleen, and kidney.

I am well aware that it will be argued in this case also that the tubercular element had been primary, and that the unfavourable course and termination had been all due to this. But I would point out that the patient's family and personal history were absolutely favourable, that he gave no history of the tubercle within him having ever been able to assert itself before, that the most careful examination of his spit never revealed bacilli, and that the post-mortem examination showed no evidence of old-standing tubercular lesion. I am therefore of opinion that though, like his neighbours, healthy or unhealthy, he had had tubercle bacilli in him for years, the sequence of events was as I have stated.

But a further piece of evidence in favour of this view is that cases of pleurisy presenting symptoms similar to the one just given, and running a similar course, may be met with, in which the post-mortem examination reveals no tubercle whatever, either recent or old. Of this the following is a very fair example.:—

J. H., æt. 47, worker in a brewery, was admitted 16th January, complaining of pleurisy. His family history was particularly good, his parents having died at the ages of 85 and 83. He had always been able to take care of himself,

and but for slight rheumatic pains, he had had no previous illnesses.

His present illness had begun on 1st December previously, with a chill and cough, and pain on the right side. He had kept in bed for about three weeks and then got up; but as he felt that he was troubled with breathlessness and was not improving, he sought admission to the Infirmary.

On admission he was found to be somewhat reduced in weight, his temperature varied between 98° and 101°, his pulse was usually about 104, his appetite was poor, he had thirst, and occasional sweatings.

As it is mainly the course of the case and the pathological appearances which are of interest, I shall now summarise these.

State of chest on admission, 16th January, after six weeks' illness. Marked dulness on the right side, extending as high as the third rib in front, and as the spine of the scapula behind, with absent fremitus, resonance, and breath sounds; a tympanitic note over the dull area in front; liver not apparently displaced; heart slightly displaced to the left.

On 19th January, right chest tapped, and 30 oz. of sero-fibrinous fluid withdrawn. On 30th January, no improvement in the state of his chest or in his general condition. On 1st February it was noted that he had had a slight shivering on the previous day, and that there was some pain and a pleuritic friction murmur at his left base. On 27th February, with a very slight diminution in the quantity of fluid in his right chest, it was noted that fluid had accumulated in the left chest, the dulness reaching as high as the seventh rib in the scapular line. As he was suffering from dyspnœa, the right chest was again tapped, and 25 oz. of sero-fibrinous fluid withdrawn. On 9th May examination revealed some diminution in the quantity of fluid in the right chest, with increase of that in the left; so that percussion of the chest posteriorly showed areas of dulness very much alike in extent on either side, the upper limits reaching as high as the sixth

rib in the scapular lines. As his dyspnœa was distressing, an effort to relieve it was made by tapping both sides of the chest, 16 oz. being thus removed from the right side, and 23 oz. from the left. On 23rd May he had a severe shivering, and his pulse and temperature rose rapidly. His cough became aggravated, and was accompanied by rusty, viscid, pneumonic spit. Becoming rapidly weaker, he died on 2nd June. On post-mortem examination the pleuræ of both lungs were found to be greatly thickened, the lungs were adherent to the chest-wall above, and collapsed below, owing to the pleuritic fluid. In both of them the greater part of the upper and middle lobes were found to be the seat of catarrhal pneumonic consolidation. There was no appearance of miliary or of old tubercle in the lungs or elsewhere.

3. Pleurisies which occur, and are not succeeded by Tubercular Disease in the Lungs or Elsewhere.—Under this head come the bulk of the pleurisies which are not only recovered from, but after which the individual enjoys the normal duration of life. Examples of these are fortunately frequent enough, but they are for the most part met with in private practice rather than among hospital patients. There is no doubt that with them the tubercle bacillus is the organism which may be primarily at work, but it is to my mind equally certain that they are often due to some other organism or toxin.

At this juncture a consideration of the whole subject of pleurisy and tubercle from the points of view of soil and germ would seem appropriate. It would be, however, too lengthy a digression, so I shall merely indicate in a general way some of the ideas which experience has led me to form on the subject.

In the first place, there seems to be no doubt that the tubercle bacillus is present potentially in practically every adult human being, and that the main reason why in a certain relatively small proportion it is able to become active

is that in that proportion what we call the resistance power of the tissues is below par.

This lowered resistance power of the tissues may be acquired as the result of a too straining life, of unhealthy surroundings, of other diseases, associated of course with other organisms, of injury or of excess, in all of which cases, provided that the lowering has not been too great, and provided that those various causes can be eliminated, recovery after weeks, months, or it may be years of treatment, can occur.

On the other hand, this lowered resistance power may be constitutional, that is to say, may be hereditary or innate, and then all treatment proves fruitless. We all know how helpless we are when we are faced with a phthisis, developing in one brought up from youth in the most perfect and favourable surroundings.

In practice, the bulk of the cases of tubercular disease of lungs and pleura that we meet with present to the physician faults both as regards surroundings and constitution; and the correctness of our prognosis, and in a way also, the value of our treatment in each individual case depends on our proper interpretation and appraising of the relative parts played in the disease by those two main fault factors.

But now it will be asked—What do we mean by this resistance power of the tissues? To answer this precisely is beyond us, but yet if we study Nature aright, we can obtain some information of value on the subject.

We can recognise, in the first place, that this power shows some relationship to tissue growth and development. The liability of tubercular disease to show itself specially in the brain in childhood, in the lung in adult life, and in the intestine during boyhood and girlhood, is explained when we remember that brain growth reaches its maximum about the fourth year, lung growth about the twenty-first year, and intestine growth somewhere between these two. Furthermore, the great infrequency of testicular and laryngeal

tubercle before puberty indicates that a tissue, so long as it possesses the exuberant vitality required for growth, is fairly safe, and that although there are infinite complexities, it is after this period of exuberant vitality is over that the risk becomes active.

In the second place, we can recognise that this resistance power is not necessarily associated with what we ordinarily mean by perfect physical health. Indeed the opposite is too often the case, for, although tubercular disease flourishes among the ill-grown, ill-nourished, and ill-formed denizens of the slums of our towns, it is yet often in the specially handsome and well-formed men and women that we see it to occur. This in itself is suggestive. Inasmuch as these types of humanity, when seen in our large towns, are yet not of them, but are largely the fresh product of country districts, where there is a plethora of sunlight and a minimum of air impurities and other deleterious agencies, it indicates that town life, though it may, after some generations, sacrifice the physique to some extent, yet has the effect of strengthening this resistance power. Not only may this be said of tubercle, but it applies to other organisms as well. Those of our profession who have experience of fever wards in large towns will, I think, bear me out in instancing the special frequency of serious and malignant cases of scarlet and other fevers among big and healthy-looking men and women who have been country bred.

But next, just as we know that this tissue resistance power varies enormously in different individuals, in the same individual at different times, and in the different tissues of an individual at different periods of life, so also great differences are to be recognised in the degree of virulence of the germ. In the report of the Royal Commission, this is shown very well; for not only did they find that even with the enormous doses of bacilli which they administered some animals showed very great resistance and others very little,

but they found also that some blends of the tubercle bacillus were virulent, and others hardly so at all. The inference to my mind from this is that the tubercle bacillus, like the pneumococcus, streptococcus, bacillus coli, etc., is in a non-virulent form a much more widely spread organism than we at present have any conception of. We know of many acid-fast bacilli, the Timothy grass, the butter bacillus, the smegma bacillus, Johnne's bacillus, etc., and although as yet experimental efforts have not succeeded in so increasing the virulence of any of these to such a degree as to demonstrate that any, or all of them, and the tubercle bacillus are of the same blend, yet the possibility of this being so cannot be denied. Personally, I am distinctly of opinion that something of this kind is really the case, and I am borne out in this opinion by the results which I obtained when comparing tuberculin with Timothein and Smegmine, as regards the cutaneous reactions which they induce in man. I willingly admit that in healthy and in tubercular adults these reactions are nothing like so strong, as a rule, as are tuberculin reactions, still they are distinctly similar in character, and I have met with certain instances in which their reactions were quite as marked as were those of the tuberculins.

Ever since the days of Adam, man has shown a specially strong inclination, when anything went wrong with him, to throw the blame on something outside himself. Adam blamed the woman, next it was the devil who was blamed, now it is the germ. We need not be surprised that it has been so. The idea acquits the individual himself, and in a way also his family, of any responsibility, and if it throws this responsibility on his fellows, individually or collectively, it does this so vaguely, as a rule, that the dissatisfaction caused thereby is relatively insignificant. Furthermore, this idea of the prime importance of infection in tubercular, and indeed, in all diseases, has been greatly exalted by the fact that it can be made of enormous service in furthering whole-

sale State assumptions of individual responsibilities, and so in promoting crude and unscientific socialistic legislation.

But in the sense in which infection is understood by the public, it is not true. I willingly admit that in households in which, as the result of poverty, neglect, or vice, everything that is cleanly and good is disregarded, the disease can become dangerous from person to person, just as pneumonia, scurvy, and many other diseases which are ordinarily considered not infectious can become. But to imbue the public mind with the idea that an individual with tubercular disease, who observes cleanliness himself, and who lives among people who do the same, is a source of danger to his neighbours, is as false as it is cruel.

On the other hand, there can be no doubt that milk from a cow affected with tubercular disease is a real source of danger. Here the bacilli, it may be in their most virulent form, are brought into direct contact with the mucous membranes of the mouth, pharynx, gullet, stomach, and intestine, and although at all of these parts, and especially in the stomach and intestine, there are processes and secretions, the function of which is to destroy by digestion and other means all such morbid agents, one cannot but admit that there may be times and circumstances when these will be able to escape this destruction, and to effect a lodgment in the tissues. Having got there, resistance power of the tissues may in its turn be powerless, and so an individual, who otherwise might easily have escaped tubercular disease, may be overcome.

For this milk infection the remedy must be, not alone the killing off of the affected animal, but the improvement of the conditions under which these animals live. Indeed, when we reflect that milk is not a food only, but is in addition and in reality an animal juice, it will suggest to us that the milk from a cow living under the conditions which give the tubercle germ its virulence, cannot but be a source

of risk. The belief that this is so must, for its acceptance, appeal to common sense and knowledge; but the statements of Courmont and Cade,[1] that agglutinins can be transferred to an infant through the milk of a wet nurse, and of Salge,[2] that diphtheria antitoxin may be so transmitted and absorbed by the infant, seem to afford it a scientific corroboration.

But now it must be remembered as regards the milk supply that the conditions of life of the dairy cow—absolute rest in the stall, warm atmosphere, nature of food—which tend in time to make it become tubercular, are precisely those which make it yield the largest supply of milk to the dairyman, and *vice versâ*. Moreover, there are grounds for considering that in accordance with the law that nutrition and reproduction are antagonistic processes, the breed of cow which is most robust, and so least liable to tubercular disease, is not the breed which can be made to yield the largest supply of milk, and again *vice versâ*.[3]

Improvement in milk in this respect, therefore, must simply mean addition to its price; and in the poorer districts of our large towns, there must be instances in which, either temporarily or continuously, this increase cannot be afforded. In these circumstances, I am one of those who hold that it would be better in the rearing of children to follow Nature more closely, and with the appearance of the teeth, to feed the child in larger proportion on a diet more approaching that of the adult.

At the present time, great concern is being caused by the prevalence of enlarged tonsils, adenoids, narrow palates, and ill-formed teeth, with mouth-breathing as their immediate, and lung and other troubles largely tubercular, as their ultimate result. For this unfortunate state of affairs, there

[1] Quoted in *Serums, Vaccines, and Toxines*, by Bosanquet and Eyre, 2nd ed., 1909.

[2] *Ibid.*

[3] See Dr Watson's article on "Tuberculosis," *Scottish Farmer*, 19th Nov. 1910.

are doubtless many factors to be held accountable, but I am strongly of opinion that whilst one of these is the result of the public having been imbued with the notion that cold is not a fruitful source of disease, another is the result of faulty milk and too soft feeding. I believe that as children are passing into the age of boyhood and girlhood, it would in many cases be well to substitute some other beverage for milk at certain meals, and to give with this some more solid and harder food. In this way the risk of troubles from occasionally faulty milk would be materially lessened, and with a larger proportion of such food, insalivation—which means digestion in the mouth—would be favoured, the teeth would get their destined work to do, and the palate would be broadened out.

CHAPTER III

ETIOLOGY OF PLEURISY—EXCITING CAUSES

By the exciting causes of pleurisy is meant that condition which so acts on the pleural tissue as to directly lower its resistance power to disease organisms. In a large proportion of cases this exciting cause is **exposure to cold.** It will be at once evident that the severity of the exposure required to produce a pleurisy will vary much in different cases. When the natural resistance power is great, the exposure must be a severe one; when it is small, a slight exposure will suffice. The truth of this can again be aptly shown by a comparison of sero-fibrinous pleurisies with empyemas. Thus, out of 142 patients with sero-fibrinous pleurisy, 42, or roughly 25 per cent., indicated exposure as the exciting cause, whilst in 40 cases of empyema, exposure was indicated in 24, or 60 per cent. This can be best interpreted on the theory that, as a large proportion of the sero-fibrinous cases were in delicate patients, and as a large proportion of the empyemas were in patients constitutionally more vigorous, in the latter a more severe exposure was required to produce the disease than in the former.

But it must not be forgotten that a similar variation as regards the amount of exposure required to produce inflammation of the pleura may be brought about by variations in the virulence of the acting micro-organism or toxin. It is evident that the more virulent this is the less need be the exposure required to allow it to manifest its

evil effects. On this point statistics are not easily obtained; still, in evidence of its general truth, one has only to call to mind the latent and apparently causeless onset of empyema in pyemic and septic cases, and in a way also in such a disease as scarlatina.

In connection with exposure as an exciting cause, it has been suggested that the fact that most acute pleurisies begin at the lower and antero-lateral part of the lung, can be explained on the ground that there the chest parietes are specially thin. But this is certainly not the explanation. Were it so, peritonitis caused by cold would be as common as it is rare. The explanation of the frequency with which pleurisy begins at this part of the lung is, as pointed out at p. 3, a physiological rather than an anatomical one.

The natural tendency in man to lay the blame for any misfortune which may occur to him upon something outside himself, and which has led to infection, and especially as we have seen tubercular infection being blamed for what constitutional or acquired feebleness is really to be held accountable, has acted also in exactly the same manner as regards **traumatism**. Moreover, since the Employers' Liability Acts have come in, the search for a discovery of injury as the etiological factor in pleurisy and other diseases has been increasing, and bids fair to go on doing so. The subject of traumatic pleurisy is, however, a very important one, and merits detailed consideration.

There is no doubt that a pleurisy, like a pneumonia, can be set up by injury to the side with or without the simultaneous occurrence of fracture of a rib, and although to a fall, a blow, or a strain, a pleurisy is often ascribed erroneously, yet genuine examples of traumatic or contusion pleurisy occur from time to time. Of 163 hospital cases of ordinary sero-fibrinous pleurisy, 7 gave a history of injury or strain sufficiently definite to warrant a causal relationship of a more or less distinct kind to be admitted. To illustrate this rela-

tionship, a brief account of each will best suffice. As will be seen, they all occurred in males:—

I.—W., æt. 24, a vanman, with a good family and personal history, was knocked over by a runaway horse and sustained a fracture of several of his lower ribs on the right side. He was unconscious for some minutes, and on regaining consciousness, he had a slight hemoptysis. He was at once conveyed to a surgical ward in the Infirmary, where he lay for one month, and was treated for fracture of the seventh and eighth ribs.

He then went home, but was not fit for work; and after two weeks at home, symptoms of right-sided pleurisy with effusion showed themselves. He was then sent to my ward in the Infirmary, and his chest was found to be about half full of hæmorrhagic effusion. He was aspirated three times, and after some ten weeks' treatment in hospital he was discharged quite recovered.

II.—A., æt. 34, a grocer, with a delicate family history, and a history of specific disease ten years previously, slipped his feet and fell against the edge of a box, striking it with his left side. He did not think it necessary to see a doctor at the time, as he believed he had simply strained his side; it was tender, but caused him no very great pain. As time went on, he noticed that a "lump" was forming on his chest-wall at the injured spot. After two weeks this lump had increased and become tender, so that he saw a doctor, who told him that he had fractured his seventh and probably also his eighth rib, and that this lump was due to callus. The doctor advised a blister, and afterwards plasters. He kept going about, but in another month (that is to say, three months after the accident) the pain became very severe. It was of a gripping character, very severe at night, but not bad during the day. It shot round to the back when it was very

severe, but at that time it was not aggravated by drawing a long breath or sneezing. The doctor told him that the pain was due to the callus pressing on a nerve. After some two months, however, the pain became worse during the day as well, and he noticed it to be increased by deep breathing or movement, so that as he was doing a good deal of work at the time, he had to stop it. He remained at home, applying plasters and ointments for about two months longer, but as he was not improving, he came to hospital. He was admitted, therefore, about eight months after his accident.

On his admission, he was found to be suffering from pleurisy with slightly blood-stained effusion. This was not great in amount, so that aspiration was never required. After some two months' treatment with iodides, mercurials, and rest, he was discharged, recovered of his pleurisy, but with the lump still present. It was oval in shape, 2 ins. long by 1½ ins. broad. It felt hard like cartilage, and was firmly adherent to the costal tissue. In this case we believed that the injury had set up a specific costal periostitis, and that this had induced the pleurisy, as a peripleuritis syphilitica.

III.—C., æt. 60, a gardener, fairly healthy, but liable to bronchitis, fell at his work and fractured his ninth right rib. He was conveyed to the surgical ward two days afterwards, but as, after three days' treatment there, indications of pleurisy and bronchitis supervened, he was removed to my ward. In this case no effusion occurred, physical examination showing only bronchitis and pleuritic friction. After five weeks' treatment he was discharged, recovered.

It will be noticed that in Cases I. and III. fractured rib had actually occurred. In Case II. it was doubtful. In the following cases no fracture was found :—

IV.—M., æt. 32, a worker in a sawmill, with a good family history, and a good previous health record, received a

severe blow from a log of wood on his right side. He was taken to the surgical hospital at once, and a bruise but no fracture was diagnosed. After being under treatment in bed for three days, he got up. He then noticed the characteristic stabbing pleuritic pain in the right side. This got worse, and soon dyspnœa showed itself, so that seven days after his admission he was tapped, and 35 oz. of hæmorrhagic fluid were removed. He was then transferred to the medical ward, where he was treated for eight weeks, and from which, after several tappings, he was discharged recovered.

V.—N., æt. 50, a clerk, with a history of specific disease thirty years ago, and of alcohol recently, stumbled and struck his right side against a marble table. The pain in the side was so great that he had to lie up at once, and seven days afterwards he came into the Infirmary. On admission, no fracture could be found, but a dry right-sided pleurisy was distinctly present. He had also symptoms of alcoholic neuritis. After some nine weeks' treatment, he was discharged recovered.

VI.—A., a schoolboy, æt. 12, with an apparently good family history, and a good record as to previous health, sustained a blow from a stretcher on his right side. The pain was so severe that he went to bed and was poulticed. At the end of two weeks he got up, but was able only to sit at the fireside. After three weeks longer, he came to my ward in the Infirmary, and a large right-sided effusion was found, which on exploration was found to be hæmorrhagic. Shortly after his admission the dyspnœa became so severe that he was tapped, and 40 oz. of this hæmorrhagic fluid were removed. This, on microscopic examination, showed of course red blood corpuscles, but also a large number of endothelial and round cells. As time went on, distinct indications of malignant disease of pleura and lung showed themselves,

and the boy died two months after his admission. No sectio was obtainable.

In the following case the pleurisy, like the lung disease to which it was secondary, must be traced to the trauma; but the connection between the two is still more complex than in the one just noted.

VII.—L., æt. 20, who as a boy had been always somewhat delicate, was at work as a miner, and about the beginning of June 1902 sustained a knock over the upper third of his right tibia. On the following day he noticed a lump at the place. It was painted with iodine, and he still continued to go about. As the weeks passed by, however, it was noticed to be becoming more painful and larger, and some four months after the accident he came to the Infirmary, where a sarcoma of the bone was diagnosed, and the limb was amputated above the knee. He made a good recovery, and changing his occupation for one above ground, he kept well till November 1903. He then suffered from pain at the back of the right thigh, which became so great that he had to keep the house. About a week after this he strained his right side on rising, and felt a pain in it, which was stabbing in character, and aggravated by breathing or coughing. He then applied for readmission to the Infirmary, and he entered my ward on 21st December 1903. He had cough and slight mucous expectoration. He was suffering from severe pain, both in the chest and right thigh. He gave a history of recent great loss of weight. His temperature varied from 99° to 101°, pulse 84 to 116, and his respirations 24 to 30 per minute. His blood count showed red blood corpuscles 3,600,000; whites, 11,000, going up to 19,000; h.b. 90 per cent. On examination of his chest distinct friction was heard in the right mammary region, and the upper half of the right lung was found to be solid, evidently from malignant growth. As time went on, he rapidly

became worse. Increase in the amount of lung involvement was noted, and enlarged glands made themselves evident in his right iliac region. Becoming rapidly weaker, he died on 7th February 1904. No sectio was obtainable.

In the following case, the pleurisy was ascribed to a strain :—

VIII.—A., a painter, æt. 50, with a history of previous good health, though somewhat liable to coughs, strained his left side on lifting a cask of white lead. Severe pain and cough supervened that night, and continued, so that in a week he came to the Infirmary. On admission he was found to be suffering from bronchitis and dry pleurisy of the left side. He rapidly recovered.

In all these cases there was a genuine history of injury or strain, but it will be at once evident that in some of them, notably in Cases VI., VII., and II., the precise appraising of the relative importance of the injury or strain would be a difficult matter. With these the following case can be appropriately detailed :—

IX.—C., æt. 32, a gasworks labourer, healthy, but probably alcoholic, was stabbed just below the middle of the left clavicle. He was at once brought to a surgical ward in the Infirmary, and there he was kept for eight days, by which time the stab wound was almost healed. By that time, however, he was complaining of pleuritic pain in the side, so that he was transferred to my ward. On admission there, the chest was found to be nearly half full of fluid, and on exploration the fluid was found to present the appearance of dilute blood. Most of the white cells were found microscopically to be small uninucleated leucocytes. With rest and treatment he rapidly recovered, and was discharged after three weeks.

Of empyemas caused by injury, I have notes of three examples. One was that of a man who had been shot through the chest by a Mauser bullet, the second was that of a boy who had been run over by a van. In both cases hæmothorax had first occurred, and had been followed by empyema. They made good recoveries, although the illnesses lasted many months, and the lungs remained considerably contracted. The third case was that of a man who had received a lance wound in the left side some thirty years before. He had recovered, and remained well for all those years, but at the end of that time some necrotic process had occurred at the site of the injury, and this had led to an empyema.

Lastly, among exciting causes of pleurisy must be placed direct **extension from surrounding parts.** Thus in ordinary acute pneumonia, a pleurisy is practically always present, although effusion, if present at all, is for obvious reasons limited in amount. A similar direct extension can be traced in phthisis, in malignant disease of the lung, and in lung embolism—*e.g.*, following on a phlebitis, whether it leads to gangrenous changes or not. Pleurisy may result also from malignant disease of the gullet. This in my experience has been very rare indeed. I have only seen one case of it, and in this effusion had occurred; and this when aspirated caused so much air to bubble from the gullet into the pleural cavity, that the aspiration process had to be stopped. Pleurisy also follows diseases, malignant or otherwise, of the costal wall.

Apparently from direct extension through the diaphragm one meets with many cases of pleurisy. In the abdominal tubercular disease of children and young people, this occurs, as might be expected, with special frequency. As an example of this, I quote in the first place the following:—

Felina C., æt. 11 years, born in Italy, but residing in Edinburgh, was admitted to the Infirmary on 13th November 1906, complaining of chest pain and weakness.

Her illness was stated to have come on about a month

before admission, when she complained of pain in the left side, and was noticed to be coughing. For a considerable time before this, however, her abdomen had been noticed to be swollen, and she was observed to be getting thinner. Notwithstanding all this, we were told that she had been walking about the streets until two days before her admission to hospital. There was no history of any previous illness.

State on Admission.—She appeared a fairly well-grown and developed girl for her age, but she was distinctly thin. Some fever was present. Her temperature was 101·6°, pulse 120, and respirations 28 per minute.

Respiratory System.—She complained of pain on the left side, and cough, but there was no expectoration. The left chest was noticed to be less mobile than the right, and on palpation, distinct friction fremitus was noticed on the right side in the mammary region. On the left side, dulness was found all over, from the fourth rib downwards. This dulness was absolute, indicating the presence of fluid. On auscultation all over this dull area, the breath sounds were absent—above this they were enfeebled. Over the whole of the right side the percussion note was good, the respiratory sounds were distinct, and in the mammary region distinct friction sounds were audible.

Circulatory System.—Except that the heart was slightly displaced to the right, this was normal.

Alimentary System.—Her appetite was variable, and she had been complaining occasionally of slight indigestion, but there was absolutely no history of abdominal pain nor diarrhœa. On examination of the abdomen, marked general enlargement was made out, and the veins of the abdominal wall were all somewhat distended. On palpation the prominent abdomen felt tense, and yielded to percussion a tympanitic note all over. No definite evidence of fluid could be ascertained, and no enlarged glands could be felt.

It should be mentioned here, however, that some weeks after her admission, and after some improvement had occurred, enlarged mesenteric glands were palpable.

Hæmopoietic System.—The spleen appeared normal in size; blood examination showed red blood corpuscles 4,800,000, h.b. 60 per cent., white corpuscles 8000. A differential count showed polymorphs 72 per cent., lymphocytes 28 per cent., eosinophils and basophils not found. The opsonic index was ·80.

The integumentary and urinary systems were practically normal.

When this girl came into the ward, the physical signs on the left chest were such as suggested a large quantity of fluid. Exploratory punctures were made at different parts of the chest, but only at one point was a small quantity of very clear fluid obtained. In this case, what we supposed to have happened was as follows:—Primary abdominal tubercle, which had, however, set up no intestinal ulceration nor enteritis, but had caused enlargement of the mesenteric glands with chronic peritonitis. This had led to a gluing together of the coils of the intestine, and probably also to a gluing of those coils to the parietes of the peritoneal cavity, so that there was little or no room for ascites. This process in the abdomen had probably been going on, in the remarkably latent way in which it often does, for a long time. Then by direct transmission through the diaphragm, pleuritic processes had been started. These had, on the left side, led to pleural adhesions at many parts of the surface of the lung, and lastly to effusion, which, however, as the result of the adhesions, was largely loculated. On the right side, a similar pleuritic process was going on, but to a much less extent.

With rest, good food, good air, and iodoform suppositories, this girl made great improvement. The chest condition cleared, the abdomen became less distended, and she gained in weight and in health generally. Her discharge from

hospital, however, meant a return to her old circumstances and environment, so that after some months the condition relapsed, and she died.

I have also a vivid remembrance of having witnessed this tubercular transmission through the diaphragm in each one of a family of three sons, the youngest being, at the time I knew them first, about 18 years of age. Within a period of five or six years, all three died of basal phthisis, ushered in by a pleurisy of the left side, and preceded by symptoms of intestinal tubercle. In this instance, the father was robust, but the mother was tall and distinctly delicate, and the order in which the sons succumbed to the disease was the usual one, viz., the youngest first, and the oldest last.

Lastly, to direct transmission through the diaphragm can be traced the pleurisies which one sees following inflammatory and suppurative changes associated with gastric ulcer, appendicitis, liver abscess, kidney abscess, etc. These, however, are for the most part purulent pleurisies, and examples of them will be detailed later on. As regards sero-fibrinous pleurisies excited in this way, the commonest are those which are associated with liver troubles, specially cirrhosis. But with large abdominal tumours of a simple kind, and with which ascites is associated, pleurisies not infrequently supervene. Although it is, perhaps, not justifiable to describe these as cases of pleurisy caused by extension through the diaphragm, the two following examples of such cases are fairly typical, and thus worthy of record:—

Mrs F., æt. 45, was admitted into the Infirmary, 30th November 1906. Her illness was a large ovarian tumour and a pleurisy of the right chest. Her family history was very good, and her home surroundings had always been comfortable.

Previous Illnesses.—Nineteen years before she had

been a patient in the Infirmary, under the care of the late Dr Thomas Keith. At that time she had had an ovarian tumour, and Dr Keith had operated. It would appear, however, that owing to adhesions he could not remove it. He had simply tapped it, and left it in the abdomen. Since that time, up to two years ago, it had given her little or no trouble. She had married, and had had six children and one miscarriage. About eighteen months or two years ago, however, she noticed her abdomen becoming larger. She has never had any pain, but this enlargement had gradually been increasing.

About three months ago she caught cold whilst doing washing work. A cough developed, and though she never noticed any sharp pains, she had had to complain of a feeling of soreness in front of, and across the chest. For the last week she had noticed herself distinctly short of breath.

State on Admission.—Height 5 ft. 1¼ in.; weight 7 st. 12½ lb. She is spare in build, but the muscularity is fair. There is no fever.

Digestive System.—Her tongue is clean, and she has complained lately of some discomfort after food, flatulence and occasional vomiting. On examination of the abdomen the great distension is found to be caused by a large tumour, which reaches from the epigastric region downwards, and is evidently more on the left side than on the right. About 2 in. below the umbilicus a large cicatrix is seen with puckered edges, and to this cicatrix the tumour is found to be adherent. With the tumour, some free fluid can be made out in the abdominal cavity. The tumour is nowhere painful nor tender. The liver is normal in size and position, but its upper border cannot be distinguished, owing to the pleural effusion on the right side.

Circulatory System.—Except that the impulse beat is displaced slightly to the left, nothing abnormal can be made out.

Respiratory System. — Her respirations are somewhat rapid, and she has a cough, with slight mucous expectoration. On inspection of the chest, no enlargement of the right side can be made out, but its lessened mobility is obvious. Percussion and auscultation show that the right chest is about half full of fluid.

The integumentary and urinary systems are quite normal.

Hæmopoietic System.—There is no evidence of enlargement of spleen or lymphatic glands anywhere. Examination of the blood reveals red blood corpuscles, 2,800,000, h.b. 50 per cent., white corpuscles 18,000. A differential count gives polymorphs 81 per cent., lymphocytes 16 per cent., eosinophils 2 per cent. This patient was examined by Dr Brewis, who reported that the abdominal tumour was ovarian, in part probably cystic, and that he would be prepared to remove it if the patient's general condition and the condition of her chest permitted of the operation. On 18th November the chest was tapped, and 45 oz. of the ordinary yellow pleuritic fluid was removed. With further treatment for the pleurisy, she gradually improved, so that the operation was performed, and the patient was discharged from hospital recovered on 12th January 1907.

Mrs M., æt. 48, was admitted to the gynæcological ward under Dr Brewis' care on 27th October 1906. She complained of a tumour in the abdomen, which had been troubling her for eighteen months, and of dyspnœa and pleurisy which had been troubling her for nine months. Her family history was fairly good, as also were her home surroundings. She has had eight children, of whom seven are alive. Her present illness began as stated about eighteen months ago, when she noticed a tumour on the right side of the lower abdomen about the size of a hen's egg. She consulted a doctor, who told her that it was a tumour of the womb, but

that as she was about the climacteric, it was to be hoped that it might not cause any great trouble. She tells us, however, that it has been gradually increasing in size. Then some nine months ago she caught cold, and developed a cough with some chest pain, but not very great. Rapidly, however, she became so short of breath, that she was confined to bed, where she had to sit up. After being in bed for about two months, she began to feel better, and was able to go about again. She has always, however, been distinctly short of breath, and has had a feeling of tightness in the chest. She has taken her food fairly well all through her illness, but she has been losing weight of late.

State on Admission.—Height 5 ft. 4 in.; weight 8 st. 9½ lb. She used to weigh 10 st. She generally lies on her right side, the breathlessness being then less troublesome. She has practically no fever.

Digestive System. — Her tongue is rather furred, her appetite poor, bowels regular. On examination of the abdomen, great enlargement is seen, and the tumour is felt, rounded, hard, and somewhat nodulated. In front it rises as high as the umbilicus, and it is rather on the right side. The presence of free fluid in the abdominal cavity can be made out distinctly.

Hæmopoietic System.—A few enlarged lymphatics, about the size of peas, are to be found in the neck. The spleen is normal in size. Examination of the blood reveals red blood corpuscles 3,000,000, h.b. 50 per cent., white corpuscles 11,000. A differential count showed polymorphs 68 per cent., lymphocytes 22 per cent., eosinophils 6 per cent.

As regards the circulatory system, all that need be said is that the heart is displaced towards the left.

Respiratory System.—Practically no chest pain is complained of, and she has very little cough. The respirations are about 24 per minute, and she has dyspnœa on the slightest exertion. On inspection, deficient movement is seen on the

whole right chest, and in size it is distinctly less than the left. On palpation, percussion, and auscultation, fluid, filling about two-thirds of the chest, is ascertained to be present.

The integumentary, urinary, and nervous systems are practically normal.

Dr Brewis diagnosed the tumour as a uterine-myoma, and sent her to my ward, telling me that he would be willing to remove it, provided that the state of the chest and of the patient generally would permit of the operation. On 13th November we aspirated, and withdrew 80 oz. of limpid, slightly coloured, pleuritic fluid. After this, her dyspnœa and general condition greatly improved, and the greater part of her chest, previously dull, became resonant, whilst the heart returned to its normal position. She went home intending to return to Dr Brewis, so as to get the tumour removed ; but she felt herself so well, both as regards the chest and tumour, that she determined to postpone the operation. It should be added that in both of those cases, careful examination of the pleural fluid revealed nothing to indicate any malignant disease.

CHAPTER IV

SYMPTOMS AND PHYSICAL SIGNS OF PLEURISY IN GENERAL AND IN DETAIL

I PROPOSE in this chapter to discuss these as they manifest themselves in both the sero-fibrinous and purulent forms of the disease. In Chapter VI. will be found as supplementary, special reference to some of them as they occur in empyema.

In an ordinary case of pleurisy, the symptoms of the invasion stage are—sharp pain in the side, aggravated by breathing and movement, a cough which is short, hacking, painful, and dry, some respiratory hurry, and a greater or less amount of fever. This fever has its usual associates, anorexia, thirst, furred tongue, confined bowels, scanty urine, with perhaps diminished chlorides; headache and general malaise.

Physical examination in the invasion stage reveals tenderness on pressure over the chest-wall at the part affected, little or no impairment of the percussion note there, but friction sounds, more or less distinctly marked, on auscultation.

As fluid accumulates in the pleural cavity, some lessening in the chest pain and cough may be observed. Dyspnœa, however, is then apt to show itself, gradually increasing in degree, with increase in the amount of the fluid. Physical examination in the effusion stage reveals on inspection lessened respiratory mobility, and possibly enlargement

of the affected side, flattening of interspaces, and some displacement of the impulse beat of the heart toward the sound side. On palpation, corroboration of the results obtained by inspection is recognised, and in addition absence of vocal fremitus over the part of the chest-wall where the fluid is. On percussion, dulness of a peculiarly toneless character is met with up to the level of the fluid above, and extending downwards, so as to merge with the liver dulness on the right side, and obliterate the stomach resonance on the left, whilst on auscultation over this dull area, absence of respiratory murmur and of vocal fremitus is found to be more or less complete.

The pleuritic effusion having occurred and having lasted some time, is followed in due course, in the great majority of cases, by its reabsorption, and by the recovery of the patient. This change is indicated by lessening of the irritation and trouble caused by the chest pain and cough, by improvement in the breathing, by subsidence of the fever, and by return of appetite and digestive power. Sweating to some extent is also apt to be an associate of the reabsorption. Gradually the lung re-expands, the physical signs of this being the return of percussion resonance and breath sounds, and for a time the reoccurrence of friction sounds (redux friction). The heart and diaphragm are then restored to their normal positions, the diaphragm, as explaincd at p. 11, being of the two the first to return.

Even in favourable cases of pleurisy with effusion it is some time before complete removal of fluid and re-expansion of lung is effected. A certain amount of dulness about the lower scapular region, with weakened respiratory murmur, may remain for many months; and indeed in certain cases of what is practically complete recovery, such traces of the disease in slight degree may be permanent.

So much for the symptoms and signs in a general way. These must now be considered in more detail.

Pain.—The pain in pleurisy is caused by the rubbing against one another of the inflamed tender surfaces of pulmonary and costal pleura, as the result of the to and fro respiratory movement. It is therefore greatest at the lower and lateral aspects of the chest, where the respiratory excursion is greatest. As regards degree, it is specially marked in purulent pleurisies, and indeed it may be said that if the pain of a pleurisy or pleuro-pneumonia is so intense as to require the ice-bag or morphia hypodermically, the process is fairly certain to be a purulent one.

On the other hand, it is to be remembered that a pleurisy may be wonderfully painless. We have already referred to the frequent occurrence of practically painless pleurisies at the apices in phthisis, and explained it as being due to the fact that the respiratory excursion at the lung apex is slight. But at the lower part of the lung one often meets with pleurisy indicated by loud friction sounds in individuals unassociated with pain; and this is specially so in the transient and apparently causeless pleurisies occurring in old-standing heart, kidney, and liver disease cases. Further, after reabsorption of the fluid in a pleurisy, when the pulmonary and costal pleuræ again in contact are rubbing against one another forcibly, it is usual for little pain to be complained of. This is why redux friction is usually much more easily heard than the friction occurring at the commencement of a pleurisy.

For obvious reasons, the pain of pleurisy is intensified by breathing, coughing, sneezing, movement of the body, or by pressure on the costal surface with the hand, or with the stethoscope. The site of the pain in pleurisy usually corresponds with the area of pleural inflammation; but an important point is that instead of it being there, it may be referred to the peripheral terminations of the intercostal nerves. In this way, just as irritation of the ulnar nerve at the inner elbow may set up pain at the

points of the fingers, pleuritic irritation of the intercostal nerves at the lower and lateral aspects of the chest, may set up pain in the abdomen. Thus a pleurisy may closely simulate a peritonitis or an appendicitis.

Cough.—The cough of pleurisy is also due mainly to pleural irritation. It is therefore dry, that is to say, accompanied by little or no bronchial expectoration; and because painful, it is suppressed, short, and hacking. That pleural irritation alone has the effect on the respiratory centre of setting up cough has been shown by experiment on animals, the pleura being a so-called cough-spot. In man, one often sees this demonstrated. If, for example, during the operation for empyema, and when the pus is being removed, the finger be pushed through the chest-wall opening so as to touch the pulmonary pleura, a powerful cough is almost certain to be brought on.

In the effusion stage, and especially when the effusion is a purulent one, the cough is often characteristic. Affleck used to describe it as presenting a metallic character. Donaldson refers to it as being like the cough of mediastinal tumour or aneurism. To be recognised, it requires to be heard rather than described; but its peculiarity appears to result from the patient's wishing to cough, but having very little air to do it with, making up for this by specially violent expiratory effort, with specially tight closure of the glottis, so that the coughing act is accompanied by slight phonation.

Fever.—The fever in simple sero-fibrinous pleurisy is usually a moderate one. The temperature varies in most cases from 99° to 101°, and in the adult it rarely exceeds 102° F. In the pleurisy of young subjects, higher temperatures, 102·5° to 103°, may be met with, and notably also is this the case in the pleurisies associated with tubercular disease, both in children and adults. In empyemas, high temperatures, 102·5° to 104° or over, are quite common.

Dyspnœa, as indicated by respiratory hurry, is brought

about in the early stage of pleurisy, in part by the fever, but certainly also by the pain. This, as it entails upon the part of the patient limitation in the amount of the respiratory change of air, must have for its consequence increased respiratory frequency. Later on, during the effusion stage, a more real dyspnœa, caused by the lung collapse lessening the ærating surface, ensues. When the effusion is very large, so that along with complete collapse of lung there is much heart and diaphragm displacement, the dyspnœa may be extreme; and the patient, panting for breath and with pale or livid face, will be seen either sitting up in bed or reclining on the affected side, so as to give the sound lung free play. On the other hand, it is no uncommon thing to meet with a patient with a chest absolutely full of fluid, moving about on his feet. In such a case, the effusion will often be found to have collected slowly, so that the patient has, as it were, had time to get accustomed to it.

There is no doubt also that, apart from this as an explanation, individual patients differ much in their manifestation of dyspnœa. One man will complain of intense dyspnœa, and call urgently for relief with a chest not half full; whilst another, with his side dull from apex to base, will express himself as feeling quite comfortable, unless he moves about too much.

These cardinal symptoms of pleurisy, pain, cough, fever, and dyspnœa—varying of course according to the precise position and severity of the process—are to be looked for at whatever part of the pleura the inflammation is present. When the diaphragmatic pleura is the part specially involved, the diagnosis is sometimes puzzling, and a peritonitis rather than a pleurisy may be suspected. The pain then is referred more to the base of the thorax, and it and the tenderness are often extreme, so that the patient suffers intensely, and cannot let himself be moved in bed. Hiccough,

nausea, and vomiting have also been described as occurring then, and if the right pleura is the one affected, possibly jaundice. In the course of time, however, the inflammation is apt to extend to the adjoining costal surface, and this, and the occurrence of effusion, makes the diagnosis less difficult.

Course and Termination.—As has already been stated, recovery is the usual termination of a sero-fibrinous pleurisy. It may occur at any stage. Thus, it may occur after the disease has lasted a matter, it may be, only of hours. It may occur also either without the occurrence of effusion at all (pleuritis sicca), or with an effusion which is so small as to be hardly recognisable by physical examination. On the other hand, when marked effusion occurs, it may be weeks before we can feel sure that recovery as a process has begun. In most of my hospital cases it was within a week; but I have several charts showing that distinct fever had continued for two and even three weeks before the indications of recovery and reabsorption could be recognised, and in some of these, moreover, the betterment had evidently been brought about by an aspiration. It may be said that the longer lasting the effusion and fever the greater danger is there of the tubercular element making itself manifest; and also that relapses of pleurisy on the same side, or extensions of it to the other side (bilateral pleurisies) have always a similar significance. Of prolonged cases of pleurisy I have already at pp. 35 and 39 detailed two examples. In the one, R. M., the conditions lasted some eight months, and terminated in acute miliary tuberculosis; in the other, J. H., it lasted some seven months, and terminated by an acute catarrhal pneumonic supervention. In the following case the pleurisy, which was probably a tubercular one from the start, lasted over four months and terminated in recovery:—

J. B., æt. 55, a labourer, was admitted to Ward 30, 30th April 1895, suffering from pleurisy with effusion.

His family history indicated a slight tendency to heart disease and phthisis.

His social history was always good. He had always been temperate, and his home had been comfortable.

Previous Illnesses.—Had influenza in Melbourne five years ago. Was ill about a fortnight, but completely recovered. Four years ago he fell about 20 ft., landing on his chest. He stated that his chest was badly bruised, and he was laid up for two months, but did not then see a doctor.

Present Illness. — This seems to have begun in the beginning of March last, with severe pain in the left side of the chest, aggravated by breathing and coughing. He stated also that at the beginning he had severe headache, and was feverish. He was treated by rest in bed, and by poultices and mustard plasters applied to the chest. In spite of this, however, he had been feeling no improvement, the pain was as bad as ever, and he had shortness of breath. He therefore sought admission to the Infirmary.

On admission his face was noticed to bear an anxious expression ; his lips were dry and rather cracked, the alæ nasi moved freely in respiration. His temperature was—morning 97·5°, evening 100°. Pulse varied between 80 and 90; respirations about 30 per minute. There was loss of appetite, thirst, slight cough, and night sweats. The small amount of opaque mucous spit showed no tubercle bacilli.

On examination of the chest the right side was seen to move freely, the left hardly at all, and the condition on percussion was as shown in the Diagrams VI. and VII. Over the whole of the dull area there was absent fremitus, resonance, and breath sounds; and below, the dulness in front could be made out to extend distinctly below the lower margin of the ribs. Above the dull area, especially in front, the vocal fremitus and resonance were increased. The cardiac impulse could be felt in the fourth right interspace, and

cardiac dulness could be made out to extend 1½ in. to the right of the right border of the sternum. The heart sounds

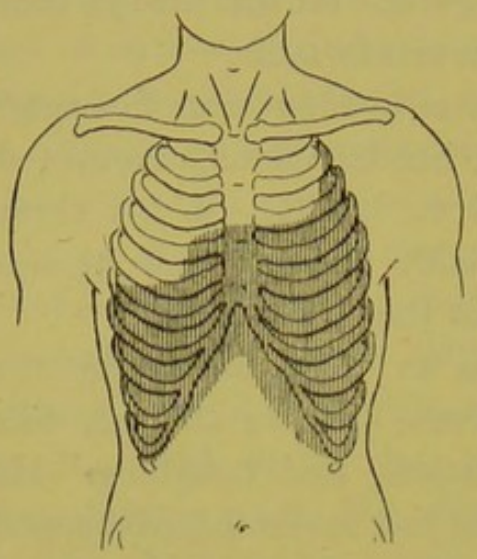


Diagram VI.

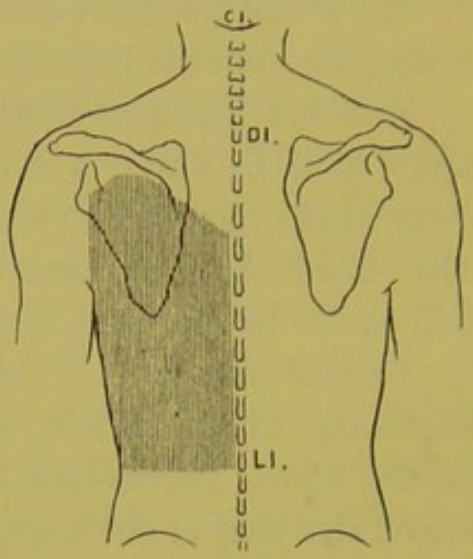


Diagram VII.

were normal. The liver was in its normal position, except that its left lobe was slightly depressed.

The examination of the chest posteriorly revealed dulness

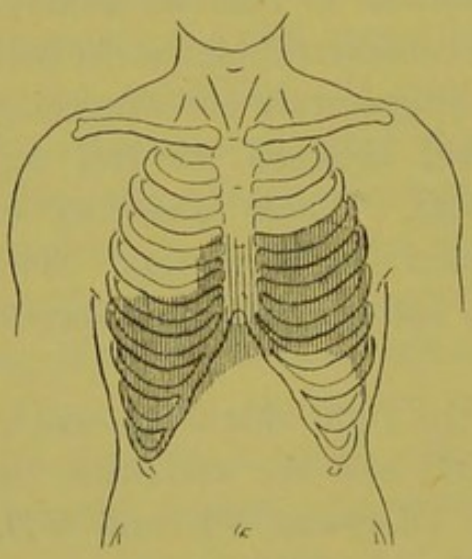


Diagram VIII.

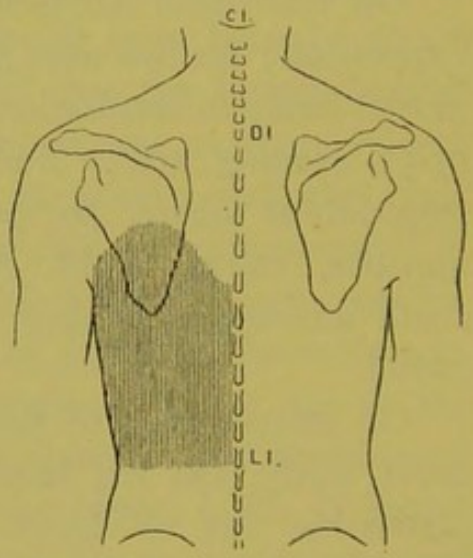


Diagram IX.

on percussion on the left side, extending as high as the spine of the scapula in the scapular line. Nearer the vertebral column the dulness was not so high, so that, as was remarked, the "curved line" was just beginning to show itself. The

other organs and systems were normal. He was treated by rest in bed, and a mixture of potassium iodide, syrup of the iodide of iron, and tincture of digitalis, with half an ounce of brandy every four hours. As the dyspnœa was troubling him, as he was very feeble, and as counter-irritants had been employed before his admission without effect, it was determined to withdraw some of the fluid. The chest was accordingly aspirated on 31st May, 19 oz. of distinctly hæmorrhagic fluid being drawn off.

The blood-stained character of the effusion corroborated

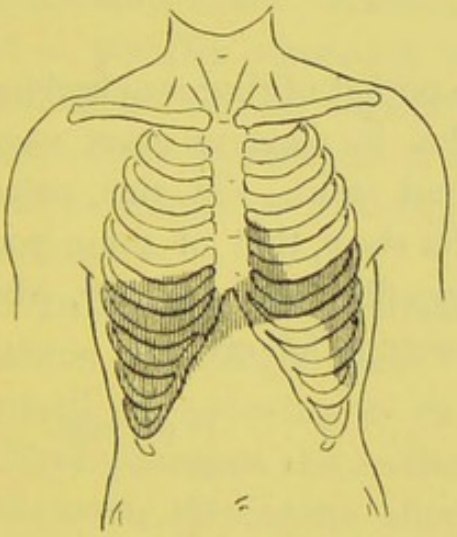


Diagram X.

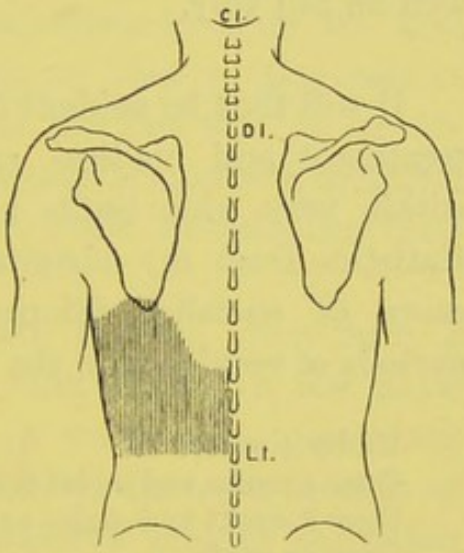


Diagram XI.

what we had been suspecting, viz., that this, if not a tubercular pleurisy, was very likely to become one in time; and so we determined to treat it by tapping at short intervals. This was accordingly done on 2nd June and 7th June—10 oz. being removed on the first, and 25 oz. on the second occasion. On both of those occasions more could have been drawn off, but the occurrence of pain warned us that it was better to desist. After the tapping on 7th June, distinct improvement showed itself, and on 11th June the condition of the chest was as shown in the Diagrams VIII. and IX., a distinct improvement also showing itself in the pulse, temperature, and respirations. Still there was fluid in his

chest, and on 14th June I aspirated again, drawing off 22 oz. It was noted that the fluid on this occasion, though still blood-stained, was lighter coloured than it had been previously. On 18th June the improvement in the condition of the chest was very marked; the tympanitic stomach note was no higher than before, but the heart had returned to its normal position, and the curve of the curved line was flattening out (Diagrams X. and XI.). He was allowed to get up and encouraged to move about. His progress after this was uninterrupted, and he was discharged practically well on 8th July.

It will thus be evident that the period of time required for treatment and medical supervision in pleurisy must vary within very wide limits in different cases. On this point statistics from my hospital results show, that of some 200 cases of so-called idiopathic sero-fibrinous pleurisy, the periods of residence in the wards were as follows:—

Under 4 weeks	51 per cent.
Over 4 weeks and under 8 weeks . .	32 „
Over 8 weeks and under 12 weeks . .	12 „
Over 12 weeks	4 „

In connection with these results it may be said that, as a rule, the shorter the period of residence in hospital, the more complete and enduring was the recovery, and *vice versâ;* and that in the prolonged cases the prolongation was associated in the vast majority of instances with manifestations of tubercular supervention.

A fatal termination in pleurisy, as the result of the disease itself, is rare. Indeed Louis went so far as to declare that pleurisy never caused death, but this requires qualification.

In the first place, as the direct result of the effusion, the heart's action may be so embarrassed that sudden and fatal

syncope may occur. This is specially frequent in the pleurisies of the elderly. Moreover, it is to be remembered that sudden death sometimes occurs during the operation of aspiration in the sero-fibrinous, and of chest opening in empyema; and whilst syncope is recognised as the pathological cause in such cases also, some other condition, *e.g.*, cardiac thrombosis or pulmonary œdema, may be operative.

Brain affections may supervene and cause death. In the course of an empyema, cerebral abscess may form, most frequently it would appear in the occipital and tempero-sphenoidal lobes. Hemiplegia may show itself in the case of a pleurisy somewhat suddenly, so that a diagnosis of embolism may be suggested. But the fact that in certain cases these hemiplegias prove to be only temporary, indicates that we must look at times to some toxic condition for their production.

In tubercular pleurisies, I have several times seen a sudden hemiplegia occur, which, after lasting a few days, passed off, ushering in its wake a tubercular meningitis. I have witnessed also in these pleurisies, the supervention of tubercular meningitis after the patient's mental condition had shown for some days distinct derangement.

Bearing out the view that a cerebral toxemia is a frequent factor in producing those conditions, is the not infrequent occurrence of forms of toxic peripheral neuritis after pleurisy. Paralysis of the shoulder and arm muscles of the affected side is met with every now and again in empyema, and although these conditions have not, in my experience, occurred so frequently in sero-fibrinous pleurisies, yet the wasting of the chest and shoulder muscles which is often seen after such, may justifiably enough be ascribed to disturbed nerve function. Garland aptly compares the wasting of these muscles after a pleurisy to the atrophy of the muscles around a joint which so often follows a synovitis.

Waxy disease affecting kidney, intestine, spleen, and liver

is another cause of death in long-standing empyemas; but with the more ready recognition and efficient treatment of this disease, it is much less frequently met with now than it used to be.

But now, a consideration of the physical signs of pleurisy in more detail is necessary.

We have previously referred to the fact that in the early stage of the disease, when the chest pain is acute, and when the friction sound is practically the only sign of the condition, this sound is often difficult to hear. This is simply because its production, viz., the rubbing together of pulmonary and costal pleura, is so painful that the patient, even when commanded to take a long breath, tries to do so, but does not do so. He inspires and expires, but the bulk of the air goes elsewhere than to the part of the lung corresponding to the affected pleura. It is quite otherwise, however, when there is no pain, as in some painless intercurrent pleurisies, and in the so-called redux friction conditions. Then the patient by breathing can rub the rough surfaces together without any discomfort, and the coarse, scratching, or rasping to-and-fro sounds can be heard, and often indeed felt.

In the effusion stage, the results obtained by inspection, palpation, percussion, and auscultation all merit consideration.

Inspection.—Under this fall to be considered alterations in the size and shape of the affected side, alterations in its mobility, and alterations brought about by pathological changes in connection with the intercostal muscles, the vascular supply, and the displacement of the heart and diaphragm.

First, then, as regards the size of the affected side. When a chest is full of fluid from apex to base, and when with this the lung is collapsed and the heart and diaphragm are both displaced, some enlargement will be recognised by the eye and verified by the tape measure. If

now we look for alteration in the shape, and this we can best do by the cyrtometer, we find that whilst the normal side is in shape like a half ellipse, the affected side has approached that of a half circle. The meaning of this is that by such an alteration of shape alone, more space is afforded to the contents of the chest.

But it is important to remember that very considerable quantities of fluid may be present, although the affected side is found, on inspection and mensuration, to be not only not larger, but even to be smaller than the healthy one. This, as has already been referred to at p. 9, is because the lung collapse has left quite sufficient space for the fluid to fill. In such instances the cyrtometer tracing may resemble a semi-ellipse, with a smaller antero-posterior diameter than that found on the normal side.

Whether there is side enlargement or not, immobility of the affected chest is to a greater or less degree always a noteworthy sign, and this will be revealed alike by inspection and palpation.

When we reflect on what has just been said, and on the fact that, as pointed out at p. 13, the intrapleural tension in sero-fibrinous effusions is rarely positive, we can understand that in sero-fibrinous cases, flattening or prominence of the interspaces is a relatively rare condition. It is otherwise, however, with purulent effusions. In these the intrapleural tension at the part is not only positive, as a rule, but, as already shown, the more violent inflammatory process may cause the intercostal muscles to become sodden with inflammatory œdema, and in a way paralysed. In this way, these muscles readily yield to any underlying positive pressure. In addition to all this, however, the inflammatory œdema of an empyema readily extends from the intercostal muscles to the subcutaneous tissues of the side. Hence, a localised œdematous condition of the chest-wall, with distended veins, is a specially frequent indication of an empyema.

These local changes in the chest-wall may be associated with a large empyema, and then in addition to their being present, the whole side is found to be enlarged. But it must not be forgotten that they may also be existent with smaller quantities of pus, and a side which, as compared with the healthy one, is distinctly contracted. Further, it is in empyema rather than in sero-fibrinous pleurisies that one sees a chest, very much contracted in size, but yet containing a considerable quantity of fluid. This is because with purulent effusions the space left for the pus to occupy is frequently the result not of lung collapse alone, but of actual cicatricial shrinking of lung following destructive changes in its tissues.

Palpation.—Under this head we need only consider in detail the vocal fremitus conditions found in pleurisy.

As the result of the presence of fluid, the vocal vibrations of the air in the large tubes at the root of the lung are prevented from getting through to the costal wall. Hence, vocal fremitus, and it may here be stated also vocal resonance, are absent at those parts where lung and chest-wall are separated by fluid. Where, on the other hand, lung and chest-wall are still in contact—as for example at the upper parts in cases where the chest is one-half or two-thirds full—vocal fremitus and resonance are, as a natural consequence, present, although perhaps not to the same extent as usual. But it is important to bear in mind that in many cases where the chest is found to be one-half or two-thirds full, the vocal fremitus and resonance are, at the upper parts and specially anteriorly, often very greatly increased. In my experience, the best examples of this are met with in cases where the chest has been quite full, and where reabsorption has gone on to this extent. I have repeatedly seen patients, in whom such notable increase of fremitus and resonance has been produced in this way, that a diagnosis of pre-existing apical phthisis has been confidently expressed. Moreover, this

diagnosis may be further strengthened by the presence there of some crepitations, see p. 37. In such cases, however, the percussion note over the parts will be found to be tympanitic, and often very low-pitched tympanitic, and this will keep the diagnosis right.

And now the question will be asked, What is the explanation of this increased fremitus and resonance so often met with over the upper part of the lung in pleural effusions? The fact that with it is associated a tympanitic percussion note suggests at any rate one factor in its causation, and this is that it may be due to so-called relaxed lung tissue. That is to say, that at the apices, the air cells, although open, have yet their walls less tensely stretched than in the normal condition, so that thus the air in them can vibrate without any interference from co-incident vibration of air-cell walls (Skoda). Hence to the percussing finger is yielded a very resonant tympanitic note, and to the vocal vibrations in the large tubes a very ready transmission to the chest-wall.

But I have always thought and taught that there may be another factor in the production of this fremitus and resonance increase over those parts, and this is concentration. In Diagrams VI. and VII., or VIII. and IX., p. 70, for example, it will be evident, that whilst on the right side vocal vibrations of the air in the large tubes at the root of the lung will readily diffuse and reach the entire surface of the chest-wall, the vibrations in the large tubes at the root of the left lung will reach the chest-wall only at the upper part—some in a direct line, and some reflected, as it were, from the fluid below. In this way the number of vibrations reaching the upper part of the chest-wall will be greater than normal, and so therefore will be the vocal fremitus and resonance met with there. Corresponding with the progress of the reabsorption of fluid and re-expansion of lung, more and more of the pulmonary pleura

will reach again the chest-wall, and *pari passu*, tympanitic note, increased fremitus, and increased resonance will gradually become less marked, till with complete recovery the normal condition of fremitus and resonance is attained.

Percussion.—As to percussion in pleurisy with effusion, there is little that need be said. When fluid is present, the note is dull, and the dulness is peculiarly absolute. With this is associated a feeling to the percussing finger of marked resistance.

As stated at p. 7, the upper border of the dull area can usually be shown to form more or less distinctly a curved line, the highest part of the curve being about the outer scapular region. Above this dull area lung resonance will be met with, and as already mentioned, the characteristic tympanitic note will often be found over the upper part of the chest anteriorly.

Grocco's triangle is the name given to a narrow strip of dulness; broadest below, which may be found alongside of the vertebral column, on the unaffected side, in pleuritic effusion. It is most easily demonstrated when the amount of effusion is great, and it disappears progressively with its reabsorption. For its explanation, we have to look, I believe, to the slightly collapsed condition which, as shown at p. 9, occurs in the unaffected lung as the result of the abolition of the elastic traction of its affected neighbour. A glance at the diagram on p. 9, and a consideration of the physical condition there depicted, suggests that in this condition the air-cells about the posterior border of the lung will be relatively unfilled. As might be expected, this Grocco's triangle dulness may be brought about also, though not in so marked a degree, by a pneumonia.

Auscultation in the effusion stage reveals an enfeeblement of the respiratory murmur, and of the vocal resonance, corresponding in degree to the quantity of the fluid and to the associated lung collapse. The physical explanation

of this enfeeblement is (1) that the affected lung allows little air to pass in and out; and (2) that the fluid blocks back the sound vibrations, just as we have seen it does in the case of the vocal fremitus. Inasmuch, however, as the collapse of the lung takes place mainly backwards, inwards, and upwards towards its root, the posterior surface of a collapsed lung lies usually more or less in contact with the vertebral column, and with the posterior aspects of the middle and upper ribs. In this way then, although a chest may be full from base to apex, and may show all over the front and sides absent breath sounds and absent vocal resonance, yet in and about the inner scapular region feeble respiratory murmur and vocal resonance may often be detected.

During the reabsorption stage, when the curved line is presenting itself, and when at the apex anteriorly a tympanitic note with increased fremitus and resonance is found, the respiratory murmur there may be peculiarly loud, and what is still more important, may be accompanied by crepitations so distinct that one may feel difficulty in assuring oneself that extensive destructive processes are not at work there. The physical explanations of these crepitations I believe to be a moist and relaxed condition of the air-cell walls. This leads to their being slightly glued together, but separated by air entrance at each inspiration. This occurrence has been referred to by many physicians, most notably perhaps by Trousseau, in his clinical lecture on pleurisy.

About the lower scapular region in pleuritic effusion, œgophony is always looked for, but in my experience is not found so frequently as the books would lead one to believe—at any rate in characteristic manifestation. Lænnec's idea of the production of œgophony was a thin layer of fluid between chest-wall and lung, and some compression of the bronchi, "since this must bring them into a form analogous to the reeds of certain wind instru-

ments, such as the oboe and bassoon, which have something of the bleating sound of œgophony."

Like all physicians, I have met with good examples of œgophony in pleurisy; but I have certainly found it very well marked in certain cases of dense acute pneumonia, with absolutely no fluid at all in the pleura. My view as to its production is that in some way or other the physical conditions of lung, or of lung and fluid required, are such as to block the passage through to the chest-wall of the longer fundamental vibrations, and allow certain of the short-waved overtones to pass. At any rate, in the best marked examples of œgophony which I have met with, I have always noticed on palpation distinct absence of the vocal fremitus.

In connection with the investigation of the physical signs in pleurisy, examinations of the pleuritic effusion and of the blood are important. This subject, however, need not be considered in great detail: it is necessary only to discuss those points which bear on the nature, course, and treatment of the disease.

The pleuritic effusion is ordinarily a somewhat opalescent yellow fluid, varying, however, in tint in different cases. In some pleurisies, notably in those of Bright's disease and heart disease, it may be very clear and watery-looking, resembling then a dropsical transudation rather than an inflammatory exudation; and the latency of the pleuritic symptoms which are associated with it in such cases intensifies this resemblance. In the chronic tubercular cases also, the fluid may be very clear and limpid.

On the other hand, in acute pleurisies, the fluid is usually yellow, like the fluid of a blister, coagulating rapidly on standing with the formation of the usual clot and serum. Ordinarily its specific gravity is about 1020, but it may be higher or lower. With the specific gravity, the amount of solids varies fairly regularly from some 5 or 6 per cent.

upwards and downwards. The amount of ash is fairly constant, about ·7 per cent.

On microscopic examination, cell elements are found in small or large numbers, 500 to 600 per c.mm. and upwards; and with increase in number of these an apparently sero-fibrinous effusion may merge into a sero-purulent one. Clinically we may witness this transition occurring after one or more tappings. It is to be remembered also that with a pleural cavity containing pus, loculi of practically sero-fibrinous fluid may be coexistent. This we often see on draining an empyema.

In most sero-fibrinous pleurisies, some red blood corpuscles can be found. If, for example, the yellow fluid from an acute case is kept for a few days, it will often be noticed that with the formation and contraction of a clot, a slight reddish coloured deposit falls to the bottom of the jar. If this deposit be examined microscopically, it will show red corpuscles with a large number of lymphocytes, these latter apparently not having been, like the polymorphs, seized upon to form the clot, and so falling to the bottom with the red blood corpuscles.

In certain conditions, the number of blood corpuscles becomes so markedly increased, that the fluid becomes a hæmorrhagic one, *i.e.*, from 5000 or 6000 red blood corpuscles per cubic millimetre and upwards. It is usual to consider that such an effusion denotes a tubercular or a malignant pleurisy, but this, though in a way right enough, does not cover all the conditions in which such an effusion is found. It has been said to occur in the pleurisies of Bright's disease; but in my own experience it has occurred specially in traumatic pleurisies, and in the pleurisies of purpura and scorbutus. I have met with it also in what I believe to be rheumatic pleurisies, and in the pleurisies associated with aortic aneurism. The following is a good example of this latter:—

William C., æt. 39, a miner, but formerly a soldier, was

admitted to the Infirmary 8th September 1904, complaining of pain in the right side, and stating that he had been ill for eight weeks. His family history was good, and as regards personal history he reported syphilis and gonorrhœa when about the age of 19, and fever and ague in India a few years after that.

His present illness began about eight weeks before. Without any apparent cause, one morning when he got up he noticed a sharp pain in his right side. He went to work, however, and continued working for a week. At the end of this time the pain was still present, and he noticed that it was worse at night, but did not seem to be aggravated by coughing, or taking a long breath. He stopped work for three weeks, still, however, moving about, and at the end of this time he again attempted work. As the pain continued, he had again to stop it, and about two weeks before admission he woke up one night feeling the pain very much more acute. At this time he seems to have been somewhat feverish, he had a cough, and some yellow expectoration. Finding himself not improving, he applied for admission to the Infirmary.

On admission he was found to be rather thin; weight, 9½ st.; height, 5 ft. 5 in. His pulse varied from 80 to 90; his temperature occasionally reached 99·5°.

Respiratory System.—He had a cough, with greenish-yellow spit, but showing neither tubercle bacilli nor blood. He complained of pain across the right side, which was somewhat aggravated on coughing and breathing, but was always worse at night. During the nights also, with the attacks of pain there was associated a feeling of shortness of breath, so that he had to sit up for an hour or thereabouts, panting. On examination, his right chest was found to contain fluid, reaching up as high as the second interspace in front. On exploration, the fluid was hæmorrhagic, showing, in addition to the blood corpuscles, a large number of polymorphs and lymphocytes.

Circulatory System.—All that need be said here was that his arteries were slightly thickened, that his heart was enlarged and displaced somewhat to the left, that there was a slight systolic murmur at the mitral area, and a distinctly louder and rougher one at the aortic, with pump valve accentuation of the aortic second sound.

His other systems were normal.

After some days' residence in the ward, the right pleura was tapped, and about 30 oz. of blood-stained fluid removed. Improvement in his condition generally, and in the condition of his chest ensued, but with this, distinct evidence of aortic aneurism became manifest.

When, as the result of trauma, blood has been effused in quantity into the pleural cavity (hæmothorax), it is important to note that clotting may not occur at all. In these cases, some pleural inflammation and effusion seems to follow, as the result, it may be, of the trauma, or perhaps even of the presence of the blood itself. In favourable cases, a gradual disappearance of the red corpuscles, and a corresponding decoloration of the pleural contents, is soon observed, and in no long time reabsorption of the whole takes place. Examination of the fluid in such cases usually shows a large percentage of eosinophils.

On the other hand, when, as the result of trauma, the injury to parts has been very severe, more acute pleurisy and purulent effusion follows the hæmothorax. If the lung has been seriously injured, or if foreign bodies have been introduced, a pleurisy associated with pneumo-thorax, and fœtid or gangrenous changes may occur.

Primary fœtid pleural effusions are, I believe, much more common than is generally supposed. The fluid then may be yellow and purulent, but more often it is sero-purulent and greyish in colour. These effusions will be considered in detail later.

Cell Elements in Pleurisy.—Ordinarily the cells in pleuritic effusions are best looked for in freshly drawn fluid, centrifuged before clotting has occurred; and those which may be met with are polynuclears, lymphocytes, endothelial cells, large mono-nuclears, eosinophils, mast cells, carcinoma, and sarcoma cells, and of course blood and pus cells. Only certain of these require further notice.

As might be expected in the less acute clear and watery effusions, the cells are few and mainly small, whilst in the acute cases, with opaque and yellowish fluid, they are more numerous, larger, and show a preponderance of polymorphs. In connection with this, it is interesting to remember that the preponderance of lymphocytes in the pleuritic effusions of the tubercular was the first fact about cyto-diagnosis to which attention was directed. As Grüner[1] says, however, "The enthusiasm which has ascribed to this study an almost pathognomonic importance, has now simmered down into a more sober realisation that lymphocytes may be in excess in other fluids than those excited by tubercle bacilli, while even the latter may be at certain stages associated with excess of polynuclear leucocytes in the fluid."

Probably the main significance of lymphocytosis as compared with polynucleosis, is that it denotes a condition of feebler reaction power on the part of the tissues. In this way, as feeble reaction power is the condition required to give tubercle its virulence, lymphocytosis and tubercular manifestations are usually associated.

On the other hand, increase of the polynuclears indicates stronger reaction, and the greater the reaction power, and the more virulent the infection, the more decided will be the polynucleosis. Conversely, the less will it be, with the opposite degrees either of reaction power, or of infective virulence.

As regards endothelial cells, the only points that need be

[1] *Studies in Puncture Fluids*, London, 1908.

referred to are those two :—First, that the presence of a large number of such cells points to its being of the nature of a hydrothorax rather than of a pleurisy ; and second, that there is often great difficulty in distinguishing endothelial from malignant cells. The presence of eosinophils in blood effusions has been already referred to, and for further information as regards them and other cells, I must refer the reader to the books on cyto-diagnosis. As to malignant and pus cells, reference to these will be made in the chapters on Malignant Pleurisy and on Empyema.

Organisms in Pleurisy.—The presence of organisms in pleuritic effusions, and the relative ease or difficulty of their detection there, varies greatly in different cases. Whilst in the acute yellow fluids and in the less acute pale ones the fresh centrifuged deposit may fail absolutely time after time to show them, in other effusions, notably in the sero-purulent and fœtid ones, they may be found in swarms.

The organisms ordinarily occurring alike in sero-fibrinous and purulent pleurisies are the pneumococcus, the strepto-coccus, and the tubercle bacillus. Others are the staphylo-coccus, Friedlander's bacillus, bacillus coli, typhoid, diphtheria, and influenzal organisms. The streptothrix actinomyces has also been found, and I have had patients in whom purulent pleuritic collections showed a leptothrix.

As already stated, these organisms are to be looked for in the freshly drawn centrifuged fluid, but they have also been seen in the clot. When copious, a simple film of the fluid will do. Their presence may also be ascer-tained by culture or inoculation.

As regards their significance, little that is definite can be said. The frequency of the tubercular element in ordinary sero-fibrinous pleurisies, as shown by inoculation, has already been noted, and need not further be adverted to. In empyemas many organisms can be found, but the pneumo-coccus and the streptococcus are the most common. Even

when an empyema occurs in an individual with confirmed tubercular lung disease, these organisms, rather than the tubercle bacillus, are not unlikely to be present. In an ordinary empyema, a pneumococcal infection is regarded as indicating a more favourable prognosis than a streptococcal. To a great extent, however, this is to be looked upon as the result of the soil rather than of the germ. In my experience of ordinary acute examples of these empyemas, the former —the pneumococcal—occurred largely in the young and robust, whilst the latter — the streptococcal — affected specially the elderly and feeble. Naturally the streptococcal empyemas occurring in serious septic cases are of very grave prognosis. In scarlatina also, a certain proportion of the empyemas are streptococcal.

Blood Examination.—The blood examination in pleurisy reveals changes, varying much with the acuteness of the process. Thus, in the acute cases, with distinct fever and yellow sero-fibrinous effusion, a leucocytosis up to 15,000 may be met with, whilst in the paler and more watery effusions of the less acute cases, a leucocytosis may not be marked at all. As is well known, the absence of leucocytosis, together with the presence of a greater proportion of lymphocytes, is regarded as indicating the tubercular nature of the process.

In empyemas, a leucocytosis, 20,000 or more, is to be expected, except in those cases where the pus is escaping by expectoration or through the chest-wall. In those circumstances, although there may be plenty of pus in the pleura, irritation and resorption is not going on, and so there may be no leucocytosis at all. For obvious reasons, a similar absence of leucocytosis will be found when the purulent fluid is encapsulated. In fœtid empyema, a leucocytosis is to be looked for; but I have seen many such cases in which it was not well marked, even although there had been considerable fever. It is to be remembered that

fœtid empyemas have a special tendency to find their way out through the lung and bronchi, so that for this reason there may be less likelihood in them of leucocytosis; but apart from this, it is to be remembered also that the fœtor and turbidity of a fœtid effusion may not be present at the earlier stages of the malady.

CHAPTER V

PLEURISY ASSOCIATED WITH NEW GROWTHS ON THE PLEURA—MALIGNANT PLEURISY

MALIGNANT pleurisy—that is to say, pleurisy associated with, or dependent on malignant disease affecting the pleura—is not infrequent as secondary to malignant disease elsewhere, as in the mamma, in the lung itself, in the lymphatic glands of the axilla, the bronchial glands, the anterior mediastinum, the gullet, liver, bone, peripheral malignant disease, etc. In such cases the pleurisy may be little more than an easily recognised and relatively unimportant complication of the original disease. But this is not by any means always so, as we shall see.

Apart from these cases, however, a primary malignant pleurisy may occur, and although this is not common, so uncommon indeed that by many its existence has been denied, as a subject it merits close attention from its extreme importance in a clinical, diagnostic, and prognostic sense.

The following case was regarded as an example of primary malignant pleurisy, and although, in the light of subsequent knowledge, the pleuritic condition might possibly be explained as having been the result rather of some chronic inflammatory process, it is of such clinical interest as distinctly to warrant its being recorded :—

F. H., aged 65, a butler, was admitted to the Royal Infirmary on 4th July 1879, suffering from dyspnœa, and

presenting the physical signs of pleurisy with effusion at his left base. He was a fairly well-nourished man, with plenty of subcutaneous fat, and stated that he had been ill with shortness of breath only for a few days. He died suddenly and quite unexpectedly from syncope on the day after his admission, and the sectio revealed the following conditions :—

"*Left Lung.*—Weight 1 lb. 3 oz., collapsed, especially its lower lobe. A number of pearl-like nodules were scattered over the base and towards the root. They were hard, and under the microscope showed the structure of spindle-celled sarcoma. The left pleural cavity contained 76 oz. of sero-fibrinous fluid, with some fibrinous masses in it.

"*Right Lung.*—Weight 1 lb. 5 oz., was congested and emphysematous.

"*Liver.*—Weight 5 lb. 3 oz. The coats of the gall bladder were much thickened, and bound down by a cicatricial band. A cicatricial-like nodule was found at the fundus of the gall bladder, the contents of which was a colourless, glairy-looking fluid. The cystic duct was apparently obliterated from surrounding cicatrisation. Liver substance was somewhat fatty and congested."

The other organs were normal.

The next case is a very instructive example of what was considered a primary malignant pleurisy. There is no doubt about the malignant disease, but there may be room for the usual difference of opinion as to whether or not it was really primary.

S. M., æt. 67, for many years a tinplate worker, was admitted to the Royal Infirmary, 16th January 1892, complaining of great shortness of breath. He had a good family and social history, and stated that he had been quite well up till a month ago, when, as the result of getting wet through, he caught a severe cold, and began

to cough. He however kept at his work, though he did not feel at all well. On 8th January he was worse, and had to stop work. He had pain in the right side, and could not lie on it. On 15th January, the day before his admission, he felt pain also on his left side, so that he could lie on neither side, and this had prevented him from sleeping and made him very weak.

On admission he presented the appearance of a fairly well-nourished man. Height 5 ft. 7 in.; weight 11 st. 9 lb. He was evidently suffering from considerable dyspnœa. His pulse—108 per minute—was of fairly good tension, but showed some irregularity as regards time and strength, varying with inspiration and expiration (pulsus paradoxus); temperature 97° F.

Respiratory System.—Breathing 25 per minute, regular and not painful. Cough slight, bringing up a fairly large quantity of clear viscid spit. Inspection showed the chest well formed, and the respiratory movements fairly equal on both sides. Palpation showed a marked diminution of the vocal fremitus over the lower part of the right chest posteriorly and laterally. Percussion showed dulness over this area, and auscultation showed weak to absent breath sounds and vocal resonance. Over the other side of the chest, respiration was harsh, and accompanied with coarse and sonorous rhonchi.

Circulatory System.—Pulse 108, presenting the characters already referred to. Apex beat in the seventh interspace in the anterior axillary line. The heart was thus enlarged, and displaced somewhat to the left. No murmurs were detected, but the second sound was accentuated at the base. The walls of the radial artery were atheromatous, and a well-marked arcus senilis was noted.

The other systems and organs were found normal, and the urine showed no abnormal constituents nor deposits.

The patient was treated with absolute rest in bed and

with nourishing dry diet. A mixture of potassium iodide and tincture digitalis was administered. He did not improve, however, and as the dyspnœa was increasing, and physical examination showed that the fluid was accumulating, he was tapped on the evening of 19th January. About 50 oz. of a yellow slightly blood-stained fluid were removed. It was noticed that this did not coagulate on standing, and that it threw down rapidly a thick whitish deposit. Microscopically this deposit showed many large cells, some single, many in round or elongated clusters, containing a considerable amount of protoplasm. Nuclei and distinct nucleoli were seen. Some cells showed the impressions of neighbouring ones, others showed branchings, and some were vacuolated. Some of the groups were found to stain deeply with carmine, others only with picric acid.

The fluid had a sp. gr. of 1020, and it contained of albumen 15·75 grs. per oz.

The course of this case was rapidly downward. The chest was tapped again on 26th January and on 1st, 5th, and 8th February, the quantities removed on each occasion varying from 60 oz. to 80 oz., and presenting to the naked eye and microscopically characters similar to the above. Notwithstanding this the dyspnœa became more and more aggravated, and death occurred a few hours after the last tapping on 8th February.

At the post-mortem examination the following were the conditions found :—

"The right pleural cavity contained 10 oz. of blood-stained serum. On removing the right lung the pleural surface of the diaphragm was found to be affected with malignant disease. This occurred in the shape of a considerable number of papular and nodular looking growths deeply pigmented, but showing through them as little yellow masses evidences of malignant growth. A large hard mass about the size of a pear was found at the root of the lung, extending

into the anterior mediastinum; and on examination this was found to be formed of enlarged pigmented lymphatic glands with masses of light coloured malignant growth scattered through it. On examination of the lung a constriction was found in the right bronchus at a small distance from its entrance, and the pleural surface over this showed a cicatricial-like appearance. On cutting into this, thickening was found around and occluding some of the bronchi leading to the lower part of the lung. The pleura over the lower lobe showed also at parts patches of malignant growth. In the upper lobe close to the apex was found a dense cartilage-like nodule about the size of a small bean. The whole lung was deeply pigmented with general increase of its fibrous tissue, and there were signs of bronchitis and œdema.

"The left pleural cavity was obliterated by old adhesions, and the left lung was diffusely pigmented, congested, and œdematous.

"The heart weighed 1 lb. ½ oz. It showed chronic thickening of the mitral and aortic valve cusps, specially the aortic. Both auricles were dilated. The right ventricle was dilated, the left hypertrophied. The tips of the papillæ were fibrous and the chordæ tendineæ thickened. The coronary arteries were atheromatous and tortuous.

"The thyroid gland was soft, and showed at parts patches of malignant growth, also hæmorrhagic patches.

"The pancreas showed as a nodule, about the size of a small bean, a similar patch.

"The liver weighed 3 lb. 7 oz. It was congested, and the lobules appeared slightly fatty at the periphery. Two small multiloculated cysts were found in the left lobe.

"The other organs presented a normal appearance. Microscopic examination of the new growth in the pleura—glands at root of lung, thyroid, and pancreas showed it to present a lymphomatous structure."

The pathological changes in this case I believe to have been:—

1. A long-standing chronic induration and pigmentation of the lung (the man had been for many years a tin-plate worker), with formation of pigmented masses on the pleura, and some enlargement and pigmentation of the glands at the root.
2. A recent rapid endothelial proliferation and degeneration in these masses on the pleura and in the glands at the root, constituting the malignant disease.

Whether this malignant transformation occurred first in the pleural masses or in the glands is not clear; but it was considered at the time that the affections of the thyroid and pancreas were secondary. In the comparative absence of the pleuritic pain and stitch, in the presence of dyspnœa out of proportion to the amount of effusion, in the naked eye and microscopic appearance of the fluid, this case showed, however, whether primary or not, the special characteristics of a malignant pleurisy. It is further interesting to note in connection with this man's occupation, that malignant pleurisy has been said to be specially common in cobalt miners.

Of malignant disease of the pleura, secondary to malignant disease elsewhere, I have notes of some twenty cases. With these, as with examples of primary malignant pleurisy, there is associated a certain amount of dubiety as regards their beginnings, for we are often uncertain during life, and even after post-mortem examination, as to whether the pleura or the underlying lung has been the first seat of the secondary malignant deposit.

Of such secondary malignant pleurisies, the mamma is a common primary focus, and the usual history is like that sketched in the following case:—

Mrs S., æt. 50, noticed a lump in her left breast in the

spring of 1909. It was not interfered with, however, till October 1909, when in the usual complete way it was removed at the Royal Infirmary. In the following November she returned home with the wound healed; but a week after her return, she had what seemed to be a pneumonia of the left lower lobe, with fever, chest pain, cough, spit, etc. In a few days, with persistence of the fever and other symptoms, the physical signs of pneumonia, but to a slighter extent, showed themselves at the right base. On the tenth day of the disease, the acute condition terminated with what seemed to be a crisis, and the patient felt better. This lasted for a few days only, however. The temperature again rose, and soon distinct signs of pleuritic effusions made themselves evident in both pleuræ, more marked, however, on the left. On examination on 10th December the left pleura was found to be half full.

At this time also, the general condition was not satisfactory. The patient was pale, thin, and cachectic looking. Her pulse was rapid and soft, and she had considerable respiratory distress. This latter was aggravated by the fact that she had been suffering for many weeks from pain and weakness in her right arm, so that she could not use it to help her movements in bed. No glandular enlargement could be detected anywhere, and the blood examination showed no distinct leucocytosis.

Exploratory puncture obtained a yellow fluid, like that of an ordinary sero-fibrinous pleurisy. On standing it showed a slight whitish clot, and when centrifuged a reddish white deposit was obtained which, on microscopic examination, showed red blood corpuscles, lymphocytes, and a few polymorphs, with a large number of malignant cells, some large, with single large nuclei, and some very large, with four or five large nuclei.

It will be apparent that in a case like this, the recognition

of the real state of affairs at an early stage is a simple matter. Examples, which for a time are much more obscure, are often met with, and of such the following case is a good illustration :—

Mrs N., æt. 58, a housekeeper, was admitted to Ward 33, 14th December 1903, complaining of breathlessness, and the presence of a small lump on the right side of the chest posteriorly, and stating that she had been ill for twelve weeks.

Her family history and general surroundings were satisfactory enough. As regards previous illnesses, she had only to report an attack of bronchitis a good many years ago, and the fact that three years previously she had had a dislocation of the right shoulder.

Her present illness had begun twelve weeks before, as an attack of pleurisy on the right side. She attributed it to a chill, but as regards onset and course, it seems to have been rather insidious. She told us that she had pain in the side, extending downwards and forwards to the abdomen, but that she was not feverish, and continued to go about her work for three weeks. By the end of the three weeks she was, however, breathless, and this and the pain compelled her to take to her bed. A doctor was called in, who diagnosed fluid in the right pleural cavity. He aspirated, and drew off 3 to 4 pints of what seemed to be ordinary yellow fluid. This relieved the symptoms, so that the patient got up and went about her work again ; but soon the breathlessness returned, and about ten days after a second tapping was performed, which again relieved her somewhat. After this, the breathlessness rapidly returned so severely that tapping was required every few days, and when she was admitted, eight tappings in all had been performed. With each tapping, however, it was noticed that the amount withdrawn became smaller, and at the eighth tapping the fluid was distinctly blood-stained. As time went on, a small lump was noticed

to be forming at the site of the first tapping. Finding herself weak and not improving, she came to the Infirmary, and was admitted as above.

On admission she was found to be a fairly healthy-looking woman, but distinctly thin as compared to what she had been. Her pulse and temperature were normal.

Respiratory System.—She complained of dyspnœa on the slightest exertion, but had no cough nor spit. On examination of the chest there was found over the right side posteriorly absent fremitus and resonance, dulness on percussion, and feeble breath sounds. In the right interscapular region those signs disappeared, and at the right apex posteriorly, with a fairly normal percussion note, rather feeble vesicular breathing was heard. Over the right side anteriorly, percussion note was slightly hyper-resonant, and the respiratory murmur was enfeebled, as compared with the opposite side. The heart was normal as regards position and sounds. Blood examination showed red blood corpuscles 3,600,000, whites 10,940. No enlarged glands could be felt anywhere. At the right chest base, at the site of the first puncture, a swelling about the size of a walnut could be seen and felt. It was not adherent to the skin, but seemed to be adherent to the rib. Its surface was rather irregular, and it had a hard bony consistency. On carefully examining the right mamma, a small tumour could be distinctly felt. She could not state how long it had been there, as she had not known of its existence; it had never caused her pain, and was not adherent to the deep or superficial structures.

She was kept in bed, and endeavour was made to improve the general condition by good diet, syrup of the iodide of iron, etc. She gradually improved in general condition, and the amount of fluid in the pleura seemed rather to diminish. A needle introduced at the right base was felt to pass through very much thickened tissue, but only 13 oz. of fluid could be withdrawn. This was distinctly

hæmorrhagic. Feeling improved as regards general health, she at her own request was discharged on 21st January, and at this time it was noted that the tumour in the right breast was larger than on admission. Slight retraction of the nipple was also noticed, and although no adhesions could be made out between it and the parts around, some small enlarged glands were noted in the right axilla.

She was readmitted 3rd March 1904. Though not markedly emaciated, she was distinctly thinner than formerly. Her breathlessness was greater, and though there was no distinct rise of temperature, her pulse was always above 100, and distinctly feeble.

Respiratory System.—She was very breathless, and often there was orthopnœa. She had a slight cough, but no spit. On examination there was percussion dulness all over the right side posteriorly, most marked in the interscapular region. In front, some resonance was met with over the clavicle, but below the second rib the note was absolutely dull. Over this dull area the respiratory murmur was enfeebled, with somewhat blowing expiration, and crepitation at the end of inspiration. Vocal fremitus was practically absent posteriorly, but was present in front.

The heart seemed fairly normal as regards position and sounds. Examination of the blood showed red blood corpuscles 3,500,000, whites 7500, h.b. 75 per cent. The tumour in the right breast was enlarged to the size of a small orange. It was firm and hard, and adherent to the nipple, but not to the deeper structures. The nipple was retracted. From this tumour up to the axilla a chain of enlarged glands could be felt, some as large as a small marble, and rather painful on pressure. The lump at the right base posteriorly was much the same size as before, *i.e.*, like a walnut. A distinct swelling had made its appearance over the left side of the manubrium, and of the second, third, and fourth costal cartilages adjacent. This swelling was

very hard, and the superficial structures over it were indurated and showed some distended veins. Between it and the left breast there were some small ill-defined masses which were tender on pressure.

On the evening of her admission, an effort was made to relieve the dyspnœa by the withdrawal of fluid. The aspirator was introduced posteriorly, just above the little tumour described previously, and after some time, and with some difficulty, about 16 oz. of hæmorrhagic fluid were withdrawn. To get at the fluid, the needle had to be pushed through what seemed to be very hard tissue. A film of this fluid showed many red blood corpuscles, with very few other cells, mostly lymphocytes, and a few polynuclear malignant cells. Some cells seemed to be undergoing vacuolation.

This aspiration had little effect in relieving the symptoms. Severe attacks of breathlessness occurred from time to time, and in one of such she died on 12th March. No sectio could be obtained, but in this case the sequence probably was (1) malignant right mamma; (2) secondary malignant affection of pleura and lung; (3) rapid extension of malignant disease in the lung, as shown by the small quantity of fluid obtainable by aspiration compared to the physical signs met with; (4) extension of malignant disease to the manubrium and costal cartilages.

It must be noted that although the fluid was yellow at first, and so like ordinary pleuritic fluid, malignant deposit showed itself in the track of the first tapping.

In the following case, the primary source of the malignant disease is doubtful, unless it were the lung itself. As will be seen, however, it commenced as a pleurisy, and therefore merits being reported here.

Mrs M., æt. 53, was admitted to the Royal Infirmary 1st January 1904, complaining of pain in the side and sleeplessness, and stating that she had been ill for three months.

Her family history and her surroundings were good, and she had had no previous illnesses.

Her present illness began, as stated, some three months before, with severe pains in the back, which were looked upon as being due to lumbago. After treatment of this for some weeks by rest in bed, hot applications and stimulating liniments, she improved so much that she got up again and began to do her housework. Shortly, however, she got chilled, which compelled her again to take to bed. This time she had fever, chest pain, and cough, and soon signs of pleural effusion on the left side manifested themselves. Progressive increase in the amount of the effusion occurring, her doctor aspirated,. and removed about 2 pints of yellow fluid. After some three weeks she was aspirated again, reaccumulation having occurred. Then about five weeks ago, as the signs of effusion were still markedly present, aspiration was again attempted, but no fluid could be got. Finding herself becoming weaker and losing flesh, she came to the Royal Infirmary, and was admitted a sstated on 1st January 1904.

On admission she was tired and anxious-looking. Her pulse was about 120 per minute, her temperature varied between 98° and 99°. Her respirations were twenty-four per minute, and she had then no chest pain. She had a cough, very troublesome at night, with some frothy yet tough mucous expectoration. On examination the left chest showed the ordinary signs of effusion, viz., lessened movement with dulness on percussion, and weak to absent breath sounds all over its lower half. Distinct displacement of the heart's impulse to the right was noticed, but in connection with the physical signs at this period, there were some noteworthy points.

The first was that the amount of displacement of the heart to the right was distinctly greater than we usually find it, with the amount of fluid which the physical signs indicated.

Thus a cardiac impulse could be felt in the fifth right interspace, 2 in. to the right of the right border of the sternum, whilst the dulness did not extend behind as high as the scapular spine, and in front as high as the third rib, and the stomach resonance could be traced as high as the sixth rib in the nipple line.

The second point was that at the lower lateral aspect of the affected chest, a slight subcutaneous œdema was showing itself. In this way we were not surprised when the repeated punctures which we made at various parts of the chest in front and behind, found no trace of fluid effusion, but drew off small quantities of blood, which tended so quickly to clot as to block the tube.

As the days went by, this patient rapidly got weaker, and the physical signs of solid lung tumour extended higher up in the chest. The œdema of the left side increased, and at the various points where punctures had been made, small discoloured patches of extravasation showed themselves. All through, her other organs presented nothing abnormal. Repeated blood examinations revealed red blood corpuscles about 4,000,000, h.b. about 90 per cent., and whites about 10,000. Cyanosis showed itself, and she died suddenly on 14th January 1904.

The following case, as will be seen, was really an example of malignant disease of lung and mediastinal glands, but like the last one, it seemed to begin by a pleurisy, so that its being recorded here may be of some use.

Robert F., æt. 46, a blacksmith, was admitted to Ward 31 on 16th January 1906, complaining of disease of the left lung and inability to swallow.

His family history and his home surroundings were very good, and at work he had had the usual muscular strains and exposure of his trade. He had never been addicted to alcohol, and he was a non-smoker. With the

exception of measles and scarlet fever, he had had no previous illness.

His illness seemed to have begun four months before, with an attack of pleurisy. This attack had the peculiarity in that it seemed to be causeless. He stated that he had got up in the morning feeling quite well, but that as the day wore on, the chest pain occurred and became so severe that towards evening he had to go to bed. With this pleurisy he was ill for eight weeks, and he told us that during this time his doctor on several occasions punctured the chest but failed to find fluid. After this eight weeks of illness, he went back to his work, feeling fairly strong, though a little thinner than before.

Then about one month later, he noticed his voice somewhat hoarse, and quite suddenly, a few days after this, he found that he could not swallow his breakfast. He told us that he had been quite able to take his supper the previous night, but next morning when he was eating, it seemed as if a ball was rising in his throat, which made his food stick about the level of the first ring of the trachea.

This difficulty in swallowing rapidly increased, till liquids could not be got to go down, except very occasionally, and he began rapidly to emaciate. On 27th December, he came to see me at the Infirmary. On examination, the two sides of the chest were fairly equal in size, but there was marked respiration immobility all over the left side, with absolute dulness on percussion, absent vocal fremitus, and absent breath sounds. With the laryngoscope, immobility of the left vocal chord was recognised, and with the X-rays and screen, I made out dark shading all over the left chest, exactly limited, however, by the middle line of the body. By careful screening, the heart, the shadow of which could not be made out from that of the surrounding lung, was found to be placed more over to the left than usual. That is to say, no part of the heart shadow showed to the right of the

middle line. In this way it was evident that the condition was rather that of solid lung than of pleuritic effusion, and negative exploratory punctures at different parts afforded corroborative evidence of this.

The blood count showed a leucocytosis up to 15,000, the increase being mainly in the small polymorphs. He died on 20th January, and on *sectio* the left lung was found to be practically solid from new growth, and adherent to the pleura from apex to base. The disease had spread to the anterior mediastinum, and also along the glands in the neck.

In the cases of malignant pleurisy just detailed, it will be noted that they all occurred during the latter half of life. It must not be forgotten, however, that malignant pleural and lung disease, secondary to malignant disease elsewhere, may occur in early years. In the list of traumatic pleurisies (pp. 52 and 53), I have referred to two such cases, and I have seen a case also very similar to these, at the age of 9 years.

The symptoms of malignant pleurisy may follow on an exposure and be acute, just like a pneumonia; but as a rule, malignant pleurisies are more causeless in their origin, and latent in their symptoms than are pneumonias or simple pleurisies. Chest pain and cough are less marked, increasing dyspnœa, emaciation, and weakness are what call attention to the condition in many cases.

Aspiration of the pleural fluid may relieve the symptoms, but to a relatively slight extent; and repetition of the aspirations for the constantly returning respiratory distress often finds less and less fluid to be removed, although the physical signs of fluid are as marked as ever. For obvious reasons, this feature is most commonly seen in cases where, with the pleurisy, lung involvement is marked.

The fluid is apt to be hæmorrhagic, and on microscopic examination of the deposit, characteristic malignant cells are to be looked for. Sometimes, however, the fluid is yellow,

like that of an ordinary pleurisy, but it may then yet show the characteristic cells. At other times the fluid may not only present to the naked eye all the appearances of a sero-fibrinous pleurisy, but it may show none of these cells microscopically.

When one meets with a fluid presenting those negative characters for some time, as in Mrs N.'s case (p. 95), one might surmise that the pleurisy had occurred first independently of the malignant disease, and that after a time the morbid changes in the pleura had formed, as it were, a nidus for the malignant process to enter into. But in Mrs N.'s case it will be remembered that the lump on the chest-wall had occurred at the site of the first tapping. Small lumps of this kind I have repeatedly seen formed in this way in pleurisy dependent on malignant disease, and I can say the same of œdema of the side, and of blood extravasations appearing at the site of punctures, as in the case of Mrs M. (p. 98).

CHAPTER VI

EMPYEMA—SPECIAL SYMPTOMS AND SIGNS

THE symptoms and physical signs of empyema are in a way comprised in those of pleurisy with effusion, and thus they have been already referred to in Chapter IV. But what must now be remarked upon is as to the existence of any special features in the symptoms or physical signs of a pleurisy, by which the purulent character of that effusion may be recognised.

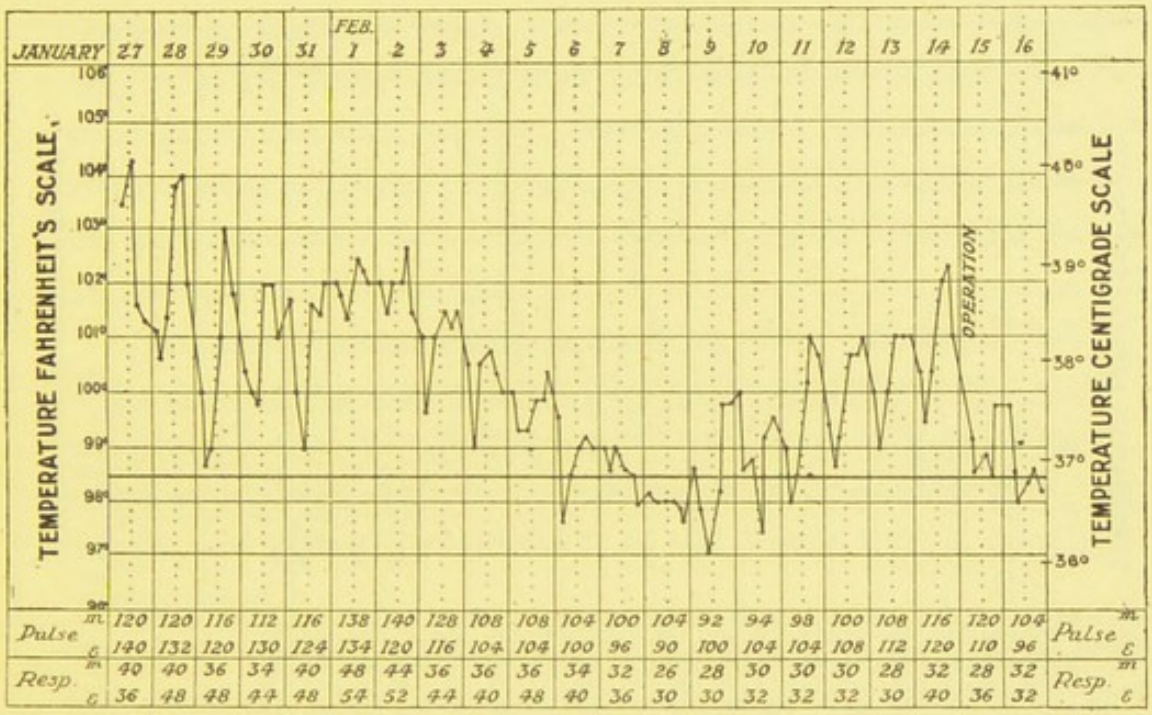
Reference has already been made to the fact that the pain in purulent effusions is specially severe. It has been put down as a rule, that if in a pleurisy or a pleuro-pneumonia the pain is such that hypodermics of morphia or the ice-bag has been called for, the supervention of empyema is almost certain. Moreover, this severe pain may occur very suddenly. In one of my cases, the empyema, which proved to be a fœtid one, began by the man's being seized when walking quietly on the street with a pain in the side, so severe that he practically fainted, and had to be carried home.

In empyema also, the amount of fever as a rule is greater. Whilst in sero-fibrinous pleurisy the temperature in adults is usually under 102, in purulent cases it is usually higher. But a further point is that in addition the oscillations of temperature are greater in purulent cases, and that whilst with the falls in the temperature the sweating is more profuse, with the rises, shiverings or even rigors are not infrequently associated.

Inasmuch, however, as empyemas show themselves differently in different cases, considerable diversities as regards the fever and its associates are to be expected. To illustrate this, I append two charts, A and B. The first chart A is from an empyema coming on, as it so often does, associated with a pleuro-pneumonia. The patient, a boy of 11 years of age, was sent into the Infirmary on the

CHART A.

GEORGE K. AGED 11 YRS. EMPYEMA AFTER PLEURO-PNEUMONIA.



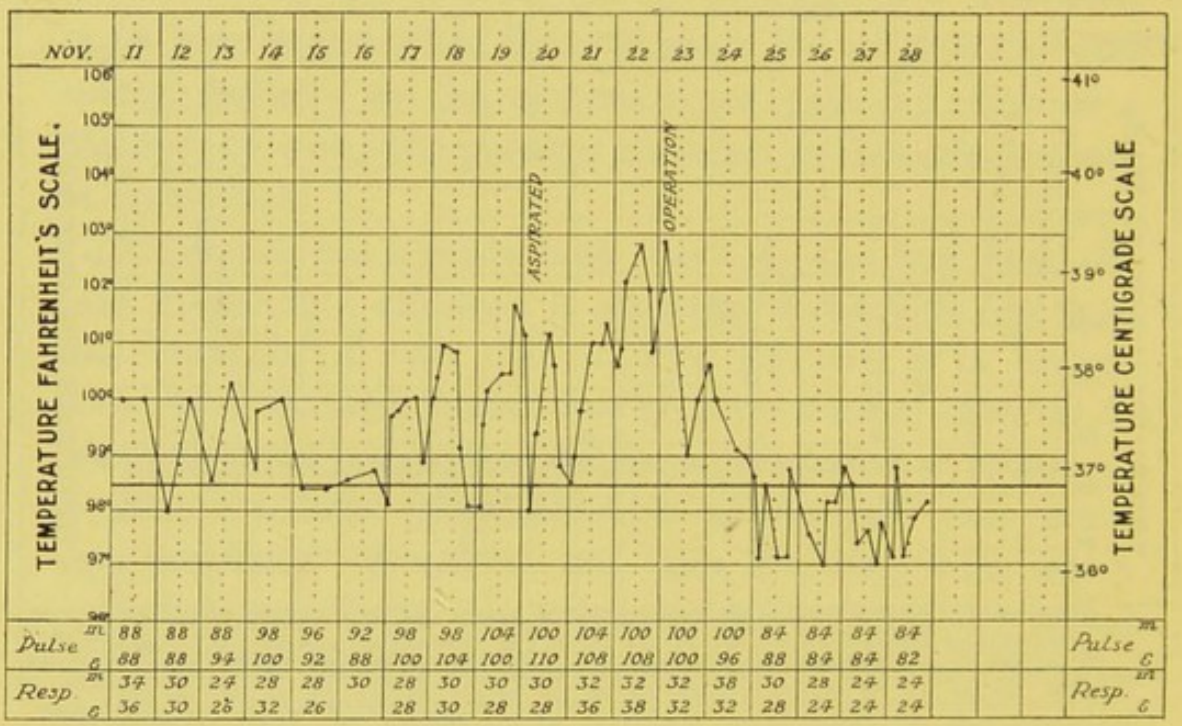
second day of what was regarded as an ordinary pneumonia. The pain, however, was severe, and as the days passed by, the chart was noted to present more marked oscillations than are usual in pneumonia, whilst the crisis fall was a slow one, and was not complete till the twelfth day. This led us to anticipate what was to follow, and we were quite prepared to see the oscillation rises of the temperature begin on the third day after the crisis, and to continue and increase. With the operation on the twenty-first day of his illness, things

rapidly mended, and the patient made a complete recovery. The organism was the pneumococcus.

The second chart B compares appropriately with this. It is that of a lady, æt. 45, and begins on the second day of what appeared to be an ordinary attack of pleurisy with sero-fibrinous effusion following on influenza. It will be observed that the temperature at first was only about 100° F.,

CHART B.

MRS L. AGED 45 YRS. FŒTID EMPYEMA.



and that the pulse all through was comparatively a slow one. The pain, however, was very severe from the start, and there was a good deal of prostration and feeling of weakness. About the sixth day it will be noticed the temperature fell; but the patient was still manifestly ill, so it was determined then to have a four-hourly chart instead of a night and morning one.

As the days passed by, the temperature rose higher, and showed rapid falls associated with sweating. On the eleventh

day, a few ounces of rather cloudy-looking non-fœtid fluid were drawn off by aspiration. This gave practically no relief, and the temperature rises became higher. On the fourteenth day, the exploring needle having revealed the presence of fœtid, sero-purulent fluid, incision, rib resection, and drainage were effected. Improvement rapidly showed itself, and although drainage was a little difficult, the patient eventually made a good recovery. The organism was the influenza bacillus.

In both of these cases, in which it will be noted the purulent character of the effusion was a relatively early feature, the special characteristics, apart from the severe pain, were the height of the fever and the oscillations of temperature. So in a pleurisy which, at first sero-fibrinous, becomes perhaps after several tappings purulent, this purulent change is marked by a higher temperature, more marked oscillations, and often shiverings and sweatings.

But, on the other hand, it must be borne in mind that an empyema may occur, and yet that these symptoms are either so slightly marked themselves or so markedly masked by other symptoms that the empyema may be overlooked. In septic and pyæmic conditions of all kinds this is specially pronounced.

For example, a man, J. G., æt. 49 years, was admitted into my ward on 14th January 1899, complaining of severe pain in the left base and loin, and also over the heart. He had a very alcoholic history, and in the previous December he had been a patient in the Lock ward, being treated there for abscess of the testicle. On his discharge from the Lock ward he had tried to work, but after a few days had had to give it up owing to weakness. Then, without any rigor or shivering, he developed pain in the left loin and side, which rapidly grew worse.

On admission, his temperature was 103°, his pulse 120 per minute, and very soft in character. The pain in the left

loin was so severe that he could not sleep at night. He did not complain of cough, but said that he was afraid to cough for the pain. On examination, with some friction over the left lower lobe laterally, a slight effusion was recognised as existent at his left base. He manifested, however, very great tenderness on pressure in the left hypochondriac and lumbar regions, so that he could hardly allow himself to be moved in bed. His temperature rapidly fell, so that on the second and third days after his admission it became and remained sub-normal. His pulse and respirations, however, kept rapid, and on the fourth day after admission he died somewhat suddenly. On post-mortem examination a left perinephric abscess and a left-sided sero-purulent empyema were discovered.

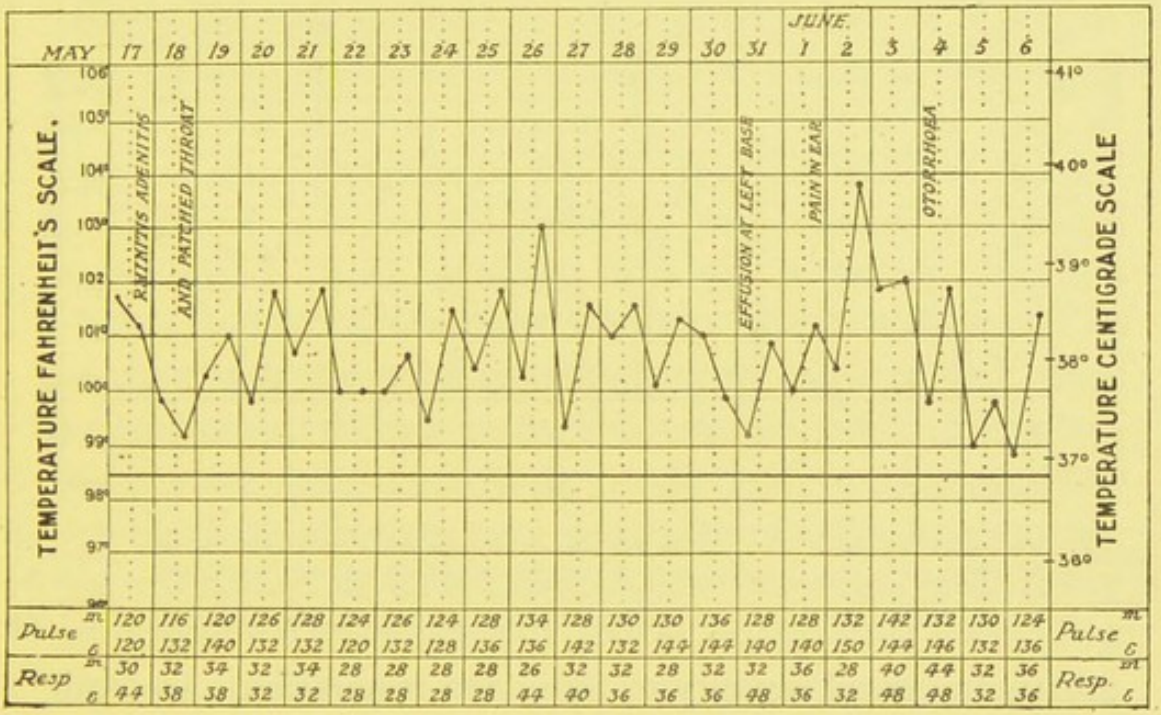
Other empyemas somewhat difficult of recognition are those which occur as a complication of scarlatina. This is in part because in them also the pain and cough may not be much complained of, and also and specially because any temperature rise may be masked by the existence of fever caused by other conditions. In the following two cases, which are depicted by the charts C and D, this is shown very well.

Chart C is that of a girl, Ruth M., æt. 8 years, who was admitted into the City Hospital 19th May 1908. She gave a history of having been taken ill with scarlet fever on 1st May previously, and on her admission desquamation was going on. Her tongue was clean, but the throat was red and congested, with the tonsils ragged and patched, and the uvula dirty. The sub-maxillary glands were enlarged, and there was well-marked rhinitis. As the chart shows, her temperature remained somewhat high, and her pulse and respirations rapid. In the beginning of June, after some pain in the left ear, otorrhœa occurred. But the noteworthy point was, that on the 31st of May, and associated with little or no cough or chest pain, signs of pleuritic effusion were

detected at the left base, and a blood count showed a leucocytosis of 15,000. Next day the affected side was explored for pus at several points. None could be localised then, but after an interval, during which the physical signs of effusion had become more marked, another exploration was successful, and the chest was opened and drained. The organism was the pneumococcus, and the child made a

CHART C.

RUTH M. AGED 8 YRS. EMPYEMA AFTER SCARLATINA.



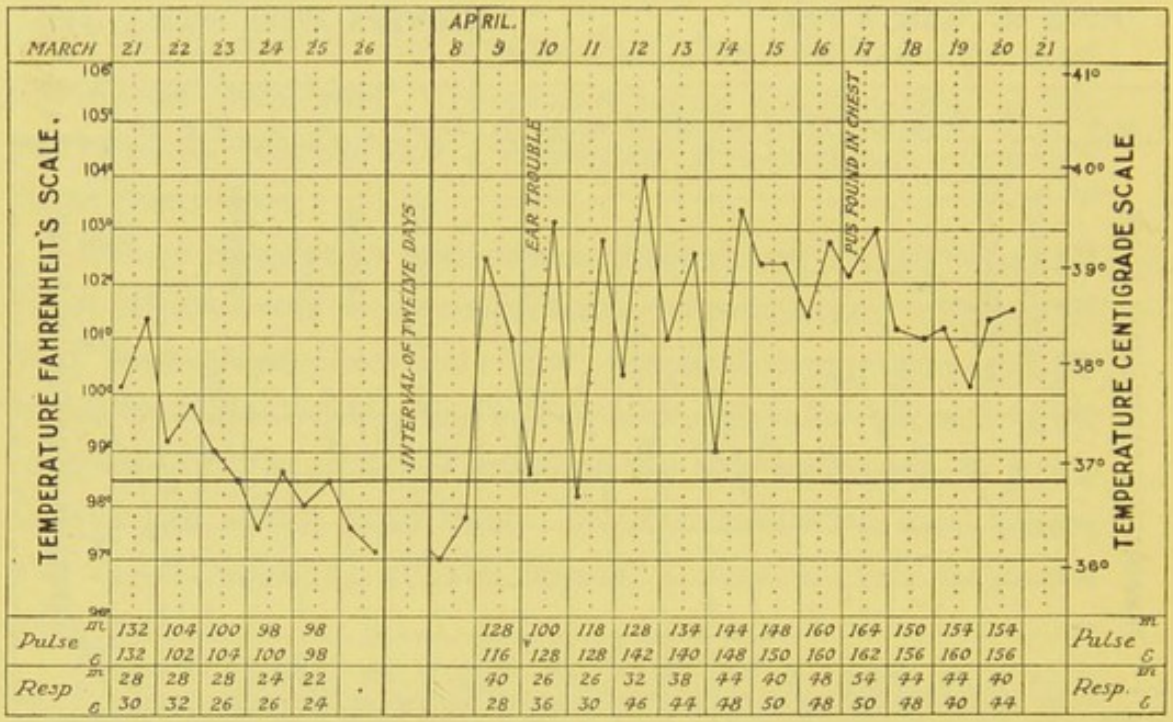
good and rapid recovery. The point, however, is that as a glance at the chart will show, there was very little in it to indicate the existence of an empyema. That is to say, that the more or less continuous pyrexia might readily enough have been ascribed to the throat, the rhinitis, the otitis, and adenitis.

The second chart D is also interesting. It is that of a girl, Maggie W., æt. 18, who was admitted into the City Hospital on 21st March 1909, being then in the fourth day

of an attack of scarlet fever. Rapid recovery followed her admission, and by 25th March all fever had disappeared. For the next twelve days, represented by the space on the chart, she was absolutely apyretic, but on 9th April she suddenly developed fever. This, as the chart shows, presented the oscillating type, and was associated with

CHART D.

MAGGIE W. AGED 18 YRS. EMPYEMA AFTER SCARLATINA.



greatly increased rapidity of pulse and respiration. On 10th April, the entry in the case-book is "Much pain in left ear, no mastoid tenderness nor œdema, but very painful on pressure in front of ear. There is pus in the meatus."

Next, attention was called to the existence of a cough, and to the marked rapidity of breathing, and the complaint of some pain in the left chest. Signs of fluid there, however, were not discovered till 17th April, when a careful exploration with the needle found pus, and a blood examination showed a leucocytosis of 20,000. Next day, 18th April, the chest was opened at the point where pus had been localised, but as

very little pus was found, the tube was left in the wound. In this case, the subsequent course was very prolonged, as proper drainage was not effected, in spite of several further explorations. However, in part by the operation wound, and in part by expectoration, the pus was got rid of, and aided possibly by vaccine treatment the patient recovered, and was discharged on 21st July following, with, however, a maimed lung and considerable chest retraction. In this case diplococci and streptococci were found in the pus.

But now, just as a patient may have an empyema, the existence of which is masked by the fact that the fever present may be ascribed to some other cause, so a patient may have an empyema the existence of which is masked by the fact that there is no fever at all. This is the case in many of those instances in which it may be said that the pus is finding a way out for itself by the bronchi, and being expectorated. Such patients, who give a history of some "inflammation" in the chest weeks or months before, and of purulent spit for correspondingly long periods, are often walking about, though perhaps not fit for work. Examination of them shows a practically normal pulse and temperature, and blood examination may show absolutely no leucocytosis. Yet they have a cough and copious purulent spit. They often describe the cough and spit as being worse when they place themselves in certain positions, *e.g.*, lying on one or the other side, leaning forward, etc. A careful examination and exploration of the chest in such cases frequently finds a considerable empyema cavity.

The physical signs of empyema are those of ordinary pleural effusions, viz., lessened or absent expansion and vocal fremitus, absolute percussion dulness, with feeling of increased resistance, and absent breath sounds and vocal resonance. But purulent effusions are apt to present certain additional peculiarities.

In the first place, inspection reveals obliteration of the

interspaces more frequently in empyemas than in sero-fibrinous effusions. This is brought about by two important factors—(1) the higher tension of purulent fluids, as may be ascertained by the manometer; and (2) the fact that the more severe pleural irritation is apt to cause some inflammatory œdema and paresis of the adjacent intercostal muscles, so that they yield more readily to any pressure. In addition, however, with this muscular œdema, subcutaneous œdema is apt also to occur, and this renders recognition of the interspaces impossible. With this latter, moreover, enlargement of the veins of the chest-wall adjacent is apt to be present, and this is also very suggestive of purulent effusion.

But next, in connection with the physical signs of empyema, it has to be borne in mind that empyemas as compared with sero-fibrinous effusions are often small and circumscribed. In this way, all the ordinary signs of fluid in the pleura may be distinctly indefinite, and the more so is this the case in empyemas which have communicated with a bronchial tube, if there has been any marked escape of the pus. In such cases, what may be found at the part affected may simply be some lessened vocal fremitus, some impairment in the percussion note, somewhat feeble breath sounds, and crepitation—that is to say, physical signs which might suggest only a chronic pneumonic condition. Moreover, empyemas may not only be small and circumscribed, they may be situated at parts of the lung where physical signs cannot reveal their presence—for instance, about the diaphragm, or the mediastinum, or between the lobes of the lung. This will be referred to in the chapter on empyemas in unusual situations.

Here reference must again be made to the fact that marked deformity of the chest (indrawn ribs, lowering of shoulder, lateral curvature of the spine, etc.), much more frequently follows empyemas than it does sero-fibrinous pleurisies. The reason of this is that an empyema, although

it may not by its size alone cause greater collapse of the lung, yet tends to set up irritative changes, and consequent fibrosis in the pulmonary tissue to a much greater degree. More especially is this so when it has penetrated lung tissue, and has communicated more or less freely with a bronchial tube. The physical signs then, in addition to the indrawing and chest retraction, may be very marked. There will be marked immobility on respiration, lessened or absent vocal fremitus and resonance, impaired or dull percussion note, with very feeble breath sounds, accompanied possibly with some crepitation. In cases of long standing, there will also be hypertrophous emphysema of the opposite lung, updrawing of the diaphragm, and especially if the left is the affected side, marked displacement of the heart.

CHAPTER VII

EMPYEMA—NATURAL METHODS OF CURE

If one were asked to state what, from the clinical standpoint, constitutes the most important difference between a purulent and a sero-fibrinous effusion, one would undoubtedly affirm that it lay in the fact that whereas with the latter, Nature's efforts towards healing limit themselves mainly to the promotion of reabsorption by blood-vessels and lymphatics, in the former they manifest themselves rather by giving the pus the power to make a way for itself out of the pleural cavity.

It will be at once evident that any information as to the how, the when, and the where of this natural process of cure in empyema must be of very great interest and use. The how, when, and where must therefore all be considered in detail; but before proceeding to this, it is necessary to remember that this difference between an empyema and a sero-fibrinous pleurisy, though important enough, is not absolute, and that, with a purulent effusion, reabsorption may really occur to a certain extent. More than this, we must remember, there is no doubt that in certain cases reabsorption may indeed occur to the extent of inducing what is practically a cure of the disease. Examples of such are not often seen or recognised in these times, but those of us who have years and experience have occasionally met with them. Thus we have often seen empyemas recovered from after several repeated tappings, and remembering that

a tapping never completely evacuates the fluid, we are driven to the view that when such treatment has been successful, Nature, aided by the tappings, has been able to complete the cure by reabsorption.

But we have still more direct evidence of reabsorption in empyema, and the following is one of the best examples which I have had among my Infirmary cases :—

Joanna N., æt. 20, a housemaid in a hotel, was admitted to the Royal Infirmary 10th November 1894, suffering from pneumonia of the left lower lobe. Her family history was fairly good, and her surroundings at work were favourable enough. Her only previous illness was an attack of acute rheumatism which had occurred three years before, and which had left a certain amount of mitral valve disease. This had not, however, incapacitated her for her work.

Her present illness had begun by a shivering on 7th November, and when admitted, she had all the symptoms and physical signs of pneumonia of the left lower lobe. The disease ran a typical course, the crisis occurring on the eighth day. After the crisis, it was noticed that the temperature chart was not satisfactory. At times it was normal or subnormal, at other times it rose to 100° or 101°. The percussion dulness also, as time wore on, showed no tendency to clear. On 26th November the temperature rose from subnormal to 104°, and percussion at the left base gave a note of such stoney dulness that the presence of fluid was at once suspected. An exploratory puncture was accordingly made, and watery pus was obtained, which showed on staining the pneumococcus. As the patient was very nervous, and as her general condition was fairly good, it was agreed to delay operation. This delay proved fortunate, for gradually the temperature fell to normal, the general condition improved, and on 29th December she was sent, quite recovered both as regards symptoms and physical signs, to the Convalescent Hospital. Careful watching

during her illness led us to conclude that no expectoration of pus had at any time occurred, and that reabsorption had done all that was required. In like examples which have been reported, it is interesting to note that with them absence of organisms in the purulent fluids, and an intact condition of the polynuclear cells present, have been described as their characteristics.[1]

Although I am distinctly opposed to non-interference in empyema, I could give other examples of Nature's workings in this manner, and I feel sure that a good many small unsuspected empyemas end in this way. Moreover, the not infrequent occurrence in post-mortem examinations of localised thickenings of the pleura, containing within them caseous or calcareous masses, indicate also old purulent collections, in which this process of reabsorption has played at least a very important part in their cure.

The following is an example of how Nature's efforts towards cure by reabsorption, though successful in one way, have yet fallen far short of what was required :—

James S., æt. 32, shepherd and ghillie, was admitted into the Royal Infirmary 8th October 1893, complaining of cough and difficulty of breathing, and stating that he had been ill for the last ten years.

His family history was not very good. His father is alive and well, but his mother died, æt. 36, of a tumour, one of his two brothers has chest trouble, one of his three sisters is delicate.

Social History.—Has always had good food, except during the year when his illness commenced, when he was in uncomfortable lodgings. He has always been temperate, and his work, although healthy enough, subjects him to much exposure.

Previous Illnesses.—As a boy, he had whooping-cough.

[1] Dieulafoy, *Text-book of Medicine*, 1910, vol. i., p. 314.

Twelve years ago, that is to say, two years before his present illness began, he had a severe strain from the weight of a shaft of a cart on his shoulder. He states that on coming to a sudden incline the weight of the shaft proved too much for him, and he fell. Next day he spat blood, and this continued for a few days afterwards.

Present Illness.—Ten years ago, when travelling sheep, he got wet through and felt thoroughly chilled, having to walk slowly with the sheep. Next morning he had a cough, and sometime afterwards he had a pain in the right side. At this time he states also that his feet and ankles became swollen. Although feeling ill, he seems yet to have been able to move about to some extent. Seven years ago, a right-sided pleuritic effusion was diagnosed, and at that time, in the Dingwall Infirmary, a large quantity of sero-fibrinous fluid was drawn off. He states that he has never been well since, that the cough, spit, shortness of breath, and loss of flesh have continued, sometimes a little better and sometimes a little worse. He states that he has come to the Infirmary to see what can be done for him.

State on Admission.—Height 5 ft. 7½ in.; weight 8 st. 13 lb.; used to weigh 12 st. 6 lb. Muscularity much impaired. Patient usually lies on his back; if on any side it is the right.

Respiratory System.—Breathing 24 per minute. Cough troublesome, occasionally causing vomiting. Expectoration muco-purulent, containing tubercle bacilli and lung tissue.

Physical Signs.—On inspection the right chest is distinctly smaller than the left, and moves less with respiration. Over the lower part of the right side the vocal fremitus is absent. Upon percussion extensive dulness is met with over the upper part of the left lung; there is slight dulness also at the right apex, and there is complete dulness over the lower part of the right lung, especially posteriorly. Over the left

upper lobe the breath sounds are bronchial or cavernous, with much crepitation. At the right apex there is bronchial breathing, with crepitation. Over the right lung, below, the breath sounds and vocal resonance are more or less absent. The heart and liver are in their normal position, and all the other organs are fairly normal. The temperature varies between normal and 100° F., and the pulse is usually about 88 per minute, and rather soft in character.

In this patient we believed we had to deal with a phthisis of both apices, specially the left, complicated by a pleuritic effusion, and probably a not very extensive one, on the right side. We could not say how long this effusion had existed, but from the absence of reabsorption or hectic fever, except what might be accounted for by the phthisis, we believed it to be a sero-fibrinous one. Remembering the possibly salutary effect of such an effusion on a phthisical lung, we inclined to let the effusion alone. About a week after his admission, however, finding the physical signs of the fluid just as before, we explored his chest with a hypodermic syringe, and drew off, to our surprise, pus, but pus presenting a very peculiar appearance. It was white in colour to the naked eye, and looked exactly like white cream. A few drachms of it were removed and sent to the Physicians' Laboratory for examination. The following report upon it was returned to me by Dr Noël Paton:—

"Specimens were examined unstained, and stained with picro-carmine, methyl blue, iodine green, and osmic acid. The pus cells were all markedly degenerated, the nuclei hardly staining. Many seem to have broken down and set free their oil granules, which gave the peculiar creamy look to the pus. Cholesterin crystals were present. No micro-organisms could be detected. The proportion of solids is very high—14·1 per cent. There is no doubt that this is an old aseptic accumulation."

In this case Nature had not been able to remove all the pus by absorption, but she had removed part of it, and by forming a thick capsule around, which in puncturing we had felt as a hard, dense layer, she had prevented the empyema doing any harm except in a mechanical way. The pus corpuscles were breaking down, the fluid was becoming inspissated, and any organisms which it had contained at first had long ago been killed, as it were, in the products of their own life.

In this case, the quantity of the fluid originally was probably great, and so Nature's efforts towards spontaneous cure by reabsorption could not be very successful. Yet they had brought about a condition which was so far satisfactory, that at that stage, and associated with bilateral phthisis, we felt we could do no good by interference.

And now as regards the how, when, and where Nature has ordained for an empyema to find its way out of the pleura. Taking the where first, we can at once say that it may be through the lung, through the chest-wall, or through the diaphragm. Of these three, there is no doubt that through the lung is at once the most common and, as we shall see by and by, the most successful method. Of my Infirmary cases, the proportion which burst through the lung, as compared with those which had burst through the chest-wall, was as 16 to 1. Of cases where the pus had penetrated through the diaphragm, I have seen very few—not more than three or four in all my experience both in hospital and in private.

Empyema opening through Lung into Bronchi.—The usual history in such a case is that a child or an adult has some chest pain, cough, and fever, with the physical signs—perhaps somewhat indefinitely marked—of pneumonia or pleuro-pneumonia at one or other base. Improvement occurs about the eighth day—perhaps a crisis shows itself then—but after a few days this improvement is recognised as not

being maintained. After some days or weeks, during which the symptoms and signs of the malady are persisting, the patient has a very bad attack of coughing, perhaps associated with some hæmoptysis and specially severe chest pain. He then is noticed to be spitting up yellow purulent matter in distinct though varying quantity, and associated with this a change for the better in symptoms and physical signs is observed. With gradual diminution in the severity of the cough and in the amount of yellow expectoration, and with gradual improvement in the general condition and in the physical signs of lung mischief, the patient may then go on towards complete recovery.

The following is an instance of a natural cure in this way, and it demonstrates another feature of great importance. This is, that this natural cure is not infrequent in cases in which the symptoms of the original empyema have not been well marked.

Maggie B., æt. 5, was admitted to the Infirmary 27th February 1895. She had always been a healthy child until the previous October. At this time she had evidently been feverish and out of sorts for some days, after which her mother noticed that on walking about, the child always placed her hand over her left side, holding it there, and complaining of a little pain. Shortly after this, she began to cough severely, and whooping-cough being prevalent, it was thought that she was suffering from this. After some seven weeks, the whooping character of the cough disappeared; but the cough continued, and it was noticed that the spit had become much more copious, and distinctly yellow in colour. All this time her general condition was suffering, she was losing in weight, and she was sweating a good deal. On admission, her pulse was found to be a little quickened, but the temperature was fairly normal. The cough was still troublesome, and the spit copious and purulent, the individual spits running together like pus at the bottom of the spittoon. There was no fœtor.

On physical examination of the chest, a distinct area of percussion dulness, rounded above, was found at the left base, with weaker breath sounds and some crepitation. An exploring needle introduced here readily found pus.

Some delay occurred before the operation could be arranged, but on 7th March it was determined to operate. The child was chloroformed, but just before the operation was begun, the usual exploratory puncture was made at the same point as before. This, however, failed to find pus, and although three other explorations were made over this area, no pus could be found. The child was therefore sent back to bed, and rapid improvement followed. The cough became less troublesome, the expectoration gradually ceased. The physical signs at the lung base cleared, and she was discharged, recovered, on 30th March following.

This, then, was an example of a natural cure in empyema by bursting through the lung, and of such I have seen and could report many other cases. In connection with them, I am of opinion that the exploratory puncture, which should never be omitted, often helps in the salutary process. I believe it did so in the case just quoted; and I have always taught that in lung trouble which is in any way of a doubtful nature, exploratory puncture should always be made. Even though it fails to find pus, it can do good by helping Nature to form a way out for the pus through the lung tissue to the bronchi.

Here again, however, I would emphasise the fact, that although this may be one of the methods of natural cure in empyema, although it may be successful when one would hardly have expected it, and although in my opinion it occurs and brings to successful terminations many small empyemas more frequently than most of us realise, it is yet not to be relied on as a means of cure, any more than we can help. That is to say, in all cases in which we find

pus in the pleura, we should endeavour to empty it through the chest-wall, for we can never be sure that Nature's efforts at cure in this way will prove quite successful.

And now let us consider in detail how this natural method of evacuating an empyema may fall short of being successful.

When by absorption along the pleural and lung lymphatics, tracts of suppuration have been formed through the pulmonary tissue between the pleura and the bronchial tubes, and when these tracts are sufficiently open to allow the empyema pus to pass along them, each inspiration by inducing a certain amount of negative pressure in air-cells and bronchi, will tend to aspirate into these structures the pus from the pleural cavity, and each cough will tend by expectoration to get rid of the pus thus aspirated.

The question may be asked here—Why in such a case does not each expiration, and especially why does not each cough drive the pus, and perhaps some of the pent-up air, back into the empyema cavity? We are forced to assume that the communicating tracts are not direct, and that thus valve-like conditions must be existent to prevent this. We know at any rate that an empyema bursting through the lung in this way is practically never followed by a pneumothorax, and when we consider that air is not thus forced back, we can easily conclude that the same may be said of any pus which has found its way into bronchi and air-cells.

In this way it can easily be understood how, with respirations going on continuously, and with coughs occurring whenever a sufficient amount of pus has been collected in the tubes, an empyema may be satisfactorily emptied. With this emptying of the empyema, and aided by expiration and cough, the air-cells will be reopened and the pleuræ will again come into contact and adhere. Then, there being no more pus, the tract of suppuration through the lung tissue will cicatrise, and so permanent and complete heal-

ing will occur. But now it is evident that supposing that the purulent collection should, for any reason, refuse to empty itself completely, and that consequently a very great number of suppuration tracts between pleura and bronchi should form, the course of the malady will not be so favourable. In such instances what will be noticed clinically is that any immediate amelioration which has followed the first escape of the pus is transient. Not for weeks only, but for months the patient will continue to expectorate purulent matter; cough, hectic, and dyspnœa will become aggravated, and loss of flesh and strength become more and more marked. In time the weakness and anæmia will become excessive, œdema, albuminuria or diarrhœa may follow on, and the patient may die after months, or even after years. In such cases, pathologically there occur important changes in the lung and bronchi which, I believe, have hitherto not been sufficiently recognised. These I shall consider in the following chapter.

CHAPTER VIII

EMPYEMA AND BRONCHIECTATIC CONDITIONS

THAT bronchiectatic cavities are frequently the result of too long existent pleuritic effusions every one admits. The lung being bound down by a layer of fibrinous membrane, and the fluid being gradually absorbed, the diaphragm will be drawn up, the heart and opposite lung drawn over, and the chest-wall drawn in. This drawing up, in, and over will not, however, in bad cases be sufficient to fill up the space which the absorbing of the fluid tends to leave, nor will in addition be the emphysematous distension of the affected lung at parts still pervious to air. Hence the bronchial tubes, the most yielding parts, will have their walls drawn out, and bronchiectasis will be produced. But what I wish now to contend for is that an empyema, by its being able to burrow through the lung tissue and open into a bronchial tube, is a specially frequent cause of basal lung disease and bronchiectatic cavities.

To explain this, let us consider some further points as regards the physics of the lungs and chest-wall.

Suppose we have a bronchiectatic basal cavity at (A), the forces which tend to keep this cavity enlarging are,—(1) acts of coughing; (2) pressure of secretion; (3) traction, as the result of cell infiltration, fibrosis, and contraction in the lung tissue around. As regards (1) and (2) nothing more need be said; it is evident that both must always act in distending the cavity. As regards (3) the traction, we have to notice

that it will act partly in distending and partly in contracting the cavity. Thus, as the newly-formed fibrous tissue contracts in every direction, it will, as shown by the straight arrows, by drawing the cavity wall to the parietes, have a distending effect. On the other hand, by its contracting in the opposite direction, as shown by the curved arrows, it will at the same time act in contracting the cavity. Which of these effects of this fibrotic contraction will be the more marked? With a very large cavity—that is to say, a cavity nearly as large as the pleural cavity itself—the enlarging effect would certainly be the greater; but with most smaller

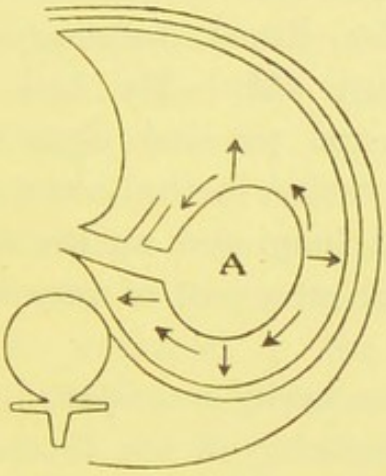


Diagram XII.

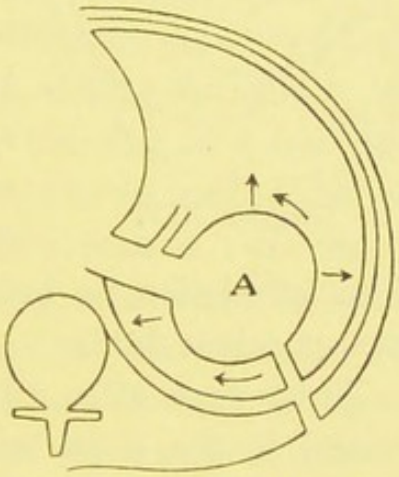


Diagram XIII.

cavities, having in consequence of their smaller size a considerable amount of lung tissue around which can yield to some extent, the contracting effect will be the greater. In ordinary large cavities, however, there will be very little difference in favour of the latter; so that, as against the distending effects of the coughing and of the accumulating secretion, any such contraction of the cavity will have little chance of occurring.

It is quite otherwise, however, when the cavity is opened and drained through the chest-wall, as at XIII. Here the secretion cannot accumulate, and so can have no distending effect, whilst the cough, owing to the drainage opening

through the chest-wall, may as a distending force practically be eliminated. The contracting effects of the fibrosis will now have a chance of manifesting themselves, the walls of the opening through the lung tissue and the adhesions between the pleura and ribs will both be drawn upon, so that with a diminution in the size of the pulmonary cavity more and more space will be yielded by the pleural cavity, as shown in Diagram XIV.

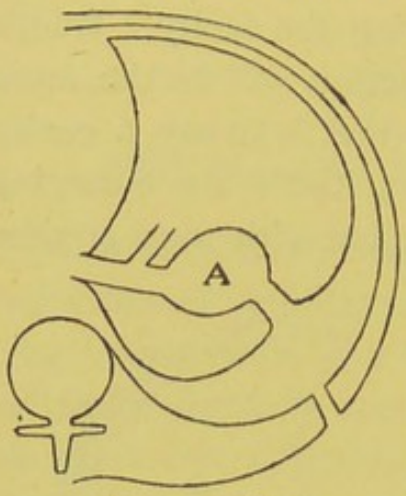


Diagram XIV.

This was precisely what happened in the case of a man, G. W., who was admitted to Dr Muirhead's ward in the Royal Infirmary on 11th August 1887. He had the symptoms and physical signs of a very large cavity in the lower part of his right lung, and on the night of his admission he brought up about a pint of very fœtid muco-purulent fluid.

Three days after admission the cavity was opened, and drained in the usual way. The opening in the chest-wall was made a little outside of the line of the angle of the right scapula, and the huge cavity was easily struck some 2 in. within the pleura, the pleura and lung being firmly adherent all round. The patient was immensely benefited by the operation, but the discharge never entirely ceased, so that drainage had to be continued. Becoming more and more exhausted, he died in April 1888. On post-mortem examination the following were the appearances met with:—

"Right lung weighed 1 lb. 7 oz. This lung was adherent a little way from its anterior border to the chest-wall by a ridge of dense, hard, thick adhesions, which cut almost like cartilage. On cutting through this ridge, and thus separating the lung from the thoracic wall, a large cavity was opened into, which was evidently the pleural cavity. The lung itself

was collapsed toward the spine, and was covered by a dense, tough membrane, the thickened visceral pleura. The lung retained its connection with the parietes across the cavity by means of two thick pillar-like structures about the thickness of a florin; these were evidently limited areas of pleural adhesions, which had been formed before the lung collapsed as a whole. The central part of their pillar-like structures contained pigmented pulmonary tissue, in which the alveoli were clearly visible to the naked eye. These two structures were situated on the upper part of the collapsed lung.

"On the surface of the lung on its posterior aspect and near its inferior border, there were two openings the size of large goose-quills; these were at the bottom of funnel-shaped depressions, and were quite evidently of long standing. One of these opened into a small cavity the size of a hazel nut, which had fibrous walls. This cavity was in connection with a somewhat dilated bronchus. The second one was in connection with an anfractuous cavity, which ran for about 3 in. upwards in the substance of the lung. This cavity had its walls formed of pigmented alveolar tissue, and presented no fibroid thickening, as old cavities do; the largest part of this cavity would hardly hold a pigeon's egg—the greatest part of it was not one-third of that size. In the apex the tissue of the lung was tunnelled with cavities which communicated with one another, and presented the further interesting fact that they were in connection, by long passages, with the pulmonary tissue in the pillar-like structures already referred to. Sections of this lung presented many different stages of destructive action. The more affected portions were deeply pigmented and leathery. The pleura was very much thickened, the bronchi were of a deep purple colour."

In this patient the pulmonary cavity on his admission was so large that complete cicatrisation could not possibly occur. But the point which I want to emphasise is that, as

in Diagrams XII., XIII., and XIV., the diminution in size of the pulmonary cavity meant the formation of the pleural one. Now my contention is that, similarly, a collection of pus in the pleural cavity will, if allowed to drain through the lung, produce a pulmonary or bronchiectatic cavity.

At the risk of being accused of repetition, let me explain this from the physical and mechanical point of view.

In the case of an ordinary circumscribed empyema, the lung tissue adjacent is more or less solid from compression and irritative inflammation, and the acts of inspiration, expiration, and coughing have little effect on the pus. But suppose this pus ulcerates its way through the lung tissue into a bronchial tube? Its escape by the tube is, as previously stated, practically never followed by the entrance of air into the empyema cavity; and so, with this escape there ensues reopening of pulmonary vesicles, re-expansion of lung, approximation of the pulmonary to the costal pleura, and consequent obliteration of the empyema cavity. The escape of the pus will also, as has been noted, be favoured by every inspiration, and the approximation of the pulmonary to the costal pleura will be favoured by expiration and coughing. In time, by this suction and force-pump action of inspiration, expiration, and coughing, the pus is got rid of, the pleural membranes become adherent, and in favourable cases, this, as we have seen, means recovery of the patient.

But suppose that the pus does not all escape, so that through the neighbouring portions of lung tissue suppurating tracts are formed, the result is that all through those parts of lung, irritative and inflammatory changes, and consequent destruction of portions of lung tissue will occur. What then follows is demonstrated in the Diagrams XV., XVI., and XVII. In XV. is represented an empyema of the lower and posterior part of the lung, with suppurative tracts through lung tissue towards the bronchi. In XVI. this empyema has opened through those tracts into a bronchial

tube, and although consequently some re-expansion of lung has occurred, yet a large part of its tissue has had irritative changes set up in it, or has been destroyed. This loss of tissue has to be made up for by fibrosis and contraction, and this must act by drawing bronchial walls to pleura, and so must tend either to separate again the pleural membranes, or to dilate the bronchial tubes. When we reflect that the act of coughing is all in favour of the dilating process, and that the irritation of the bronchial mucous membrane by the pus will have caused increased

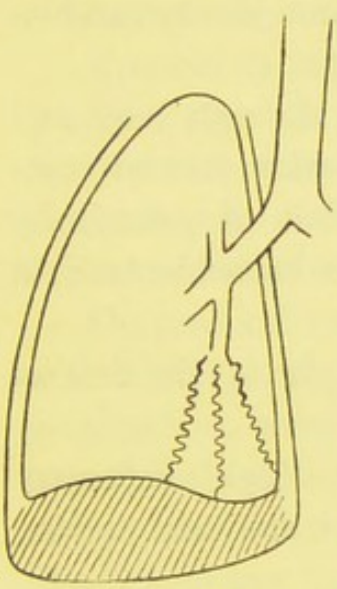


Diagram XV.

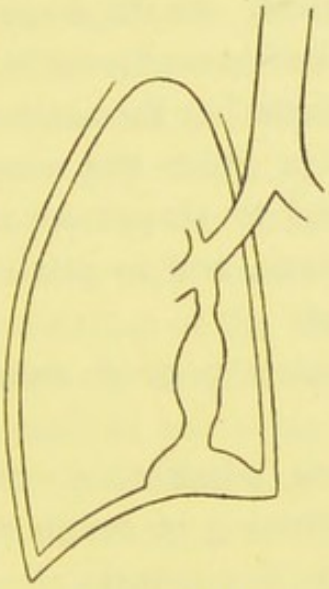


Diagram XVI.

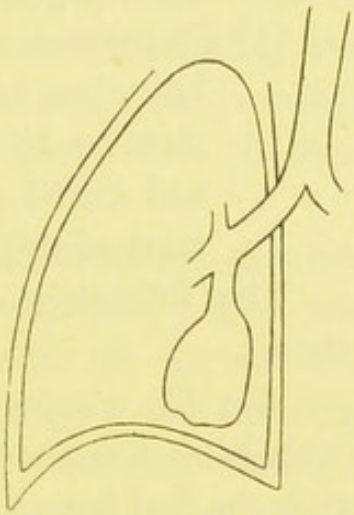


Diagram XVII.

secretion, and in time a similar distending tendency, we can understand how a bronchiectatic cavity, as in Diagram XVII., will then be produced. Here complete reapproximation of the pulmonary and costal pleura has occurred, and the irritative fibrosis around, aided by cough, and perhaps secretion accumulation, has caused the bronchiectatic cavity.

In the case of G. W., whose case of bronchiectasis has just been referred to, careful inquiry indicated that the process had originally started, as described, in an empyema.

I shall now give some clinical examples of empyemas evacuating themselves in this manner, and to make them the more clear, I shall select them as representing the three

stages of this process, represented by the Diagrams XV., XVI., and XVII. That is to say, I shall give examples of:—

(1) Empyemas evacuating themselves through lung and bronchus, but in which this evacuation was so incomplete that reapproximation of pulmonary and costal pleura had not occurred, and resulting bronchiectasis was very slight.

(2) Empyemas evacuating themselves through lung and bronchus, in which the evacuation, though not complete, had yet occurred to such an extent as to leave a relatively small empyema cavity and a distinctly marked bronchiectatic one.

(3) Empyemas evacuating themselves through lung and bronchus, and in which the evacuation was so complete as to lead to reapproximation of pulmonary and costal pleura, and to produce bronchiectasis in marked degree.

The following case is a good example of the first of these:—

Mrs C., æt. 39, was admitted to the Royal Infirmary 29th June 1900, complaining of cough and spit, and stating that she had been ill for five months.

Her family history showed some constitutional delicacy; she had a comfortable home, and she had had practically no previous illnesses.

Two years previously she had had a dead-born child, and had not been so strong since then.

Her present illness began over five months ago. Probably after some exposure, she felt chilly and feverish, and she vomited. After the vomiting, she felt a pain in the right side, with cough, and some frothy spit. Pleuro-pneumonia of the right lower lobe was diagnosed, and three weeks afterwards, as the symptoms and physical signs pointed to the presence of fluid, an exploring needle was used, but no fluid was obtained. The physical signs not being definite,

and as she was feeling fairly well she was allowed up. Shortly afterwards, she was sent to the country for a change. No improvement, however, took place, and on her return she was admitted to my ward in the Royal Infirmary.

State on Admission.—Height 5 ft. 4 in.; weight 6 st. 8¾ lb., used to weigh 9 st. Her temperature varied between 97° F. and 100·4° F.; her pulse, between 80 and 90 per minute, and respirations 28 to 30 per minute. She was distinctly thin and emaciated, and there was slight œdema of the ankles. In bed, she lay on the back or on the left side; if she lay on the right side, she coughed continuously.

Respiratory System.—She had a cough, with copious purulent spit. It was mixed with froth, but looked at in the jar it was seen to be largely made up of pus. In quantity, it was usually about 4 oz. in the twenty-four hours. It contained no tubercle bacilli, only cocci.

On physical examination of the chest, percussion dulness, with feeble, indeterminate respiratory murmur, and a few crepitations, were found at the right base. In the seventh interspace, about the posterior axillary line, the absolute dulness and other physical signs of fluid were most marked. An exploring needle introduced there at once found empyema pus. Next day, the chest was opened, a piece of rib resected, and after evacuating about 5 oz. of pus, a drainage-tube was inserted.

From this moment the cough and spit practically disappeared. She improved rapidly in weight and in general condition, and corresponding improvement in the physical signs at the right base were recognised. She left the hospital, recovered, on the 24th of August subsequently. She has remained well since, yet now and again with any exposure some cough and spit tend to show themselves, the result, I believe, of the fact that although the empyema is healed, some slight localised bronchial dilatation is persisting.

Of the second group of empyemas—that is to say, of empyemas evacuating themselves through lung and bronchus, but in which this evacuation, though not complete, had yet occurred to such an extent as to leave a relatively small empyema cavity and a distinctly marked bronchiectatic one—the following is a good example :—

T. R., æt. 53, a railway guard, was admitted to my ward in the Royal Infirmary 2nd April 1907, complaining of cough and the bringing up of a large quantity of very fœtid spit, and stating that his illness had lasted for eighteen months.

His family history was very good, his surroundings satisfactory, and as regards previous illnesses, the only one of consequence was an attack of pneumonia (?) of the left side five years before. This attack seems to have been a very severe one, and he was off work with it for nine weeks. He informs us that he has never been quite himself since this illness. After it he developed a short, dry, hacking cough, more or less constantly present, and aggravated by exertion. Two years ago he began to spit up a white spit, and he noticed that on some occasions it was streaked with blood. Gradually the cough grew worse, and the spit became more copious, and about eighteen months ago it became distinctly fœtid in character. On inquiry we found that the spit came up at any time of the day; he gave no history of a large quantity being coughed up, and then of a cessation. He noticed that on leaning forward, or on lying on his right, *i.e.*, his unaffected side, the coughing and spitting were specially severe.

State on Admission.—Height 5 ft. 11½ in.; weight 12 st. 5 lb., used to weigh 14 st. He looks distinctly ill, with a well-marked malar flush. His temperature varies from 97° F. to 98·8° F.; pulse usually about 80; respirations 22 per minute.

Respiratory System.—He has shortness of breath, cough

and fœtid spit, in quantity from 5 to 10 oz. daily. His breath is also fœtid. On examination of the chest, the physical signs were found to be nothing like so well marked as one would have expected. At the left base, the percussion note was rather dull, the breathing was somewhat feeble, and a few crepitations were present. With the X-ray screen, some shading was found there, with lessened diaphragmatic movement, but the shading was not at all well marked.

His other systems were normal.

Believing that in this case we had to deal with a relatively small fœtid empyema cavity and a considerable amount of bronchiectasis, and that therefore to strike the pus would require time and care, I got Mr Caird to resect a portion of the eighth rib posteriorly on 12th April. This was done, and as expected the pleura was found thickened and adherent all round. The wound was packed, and the patient was left till 16th April. On this day, the patient sitting up, we carefully explored his lung through the pleural opening, and we found pus about 2 in. from the surface on pushing the needle through fibrosed lung tissue. By probe-pointed forceps, the opening was enlarged, and a drainage-tube was introduced, a large quantity of fœtid yellow pus being evacuated. Some days after, when the cough was better, and when the fœtid spit and fœtid discharge had greatly lessened, the lung was again explored. It was found that a long bent probe, after being passed into the lung, could travel for 7 in. in an upward and slightly inward direction towards the root of the lung, and at the same time that it could also be passed downwards and forwards for about $4\frac{1}{2}$ in., into a narrow pocket on the roof of the diaphragm.

In this case then, I believe, that the long discharging empyema had produced lung fibrosis and bronchiectasis, but that, at the same time, it had not itself become obliterated.

Of the third group—*i.e.*, of empyemas evacuating them-

selves through lung and bronchus, and in which the evacuation has been so complete as to lead to reapproximation of pulmonary and costal pleura, and to produce bronchiectasis in marked degree—I have many examples. I shall at present, however, only quote two, one in which I was successful in striking the cavities, and the other in which I was not.

A. H., æt. 27, belonging to the mercantile marine, was admitted to the Royal Infirmary 19th April 1901, complaining of cough and copious fœtid spit. His family history was very good, as also were his surroundings. He had had the ordinary diseases of childhood, but no other illness, except typhoid fever some years before when in China. He gave a history of rather free indulgence in alcohol.

His present illness began some ten months before his admission. He then had had cough and pain in the left side, and he had been feverish for some weeks. After some weeks he noticed little clots of blood in the spit, and somewhat suddenly the spit became much more copious and fœtid. This cough and fœtid spit continued, sometimes better sometimes worse. Feeling himself getting weaker, he applied for admission to the Infirmary.

State on Admission.—He appeared a fairly well-nourished and developed man; height 5 ft. 5½ in.; weight 11 st. His temperature varied from 97° F. to 99·5° F. His pulse varied from 80 to 90, and his respirations were about 18 per minute. He was able to walk about fairly well. His digestive, circulatory, integumentary, urinary, and nervous systems showed nothing abnormal. His blood showed a slight leucocytosis.

Respiratory System.—He has now no chest pain except after coughing, when he feels a soreness in the throat and left side. His cough is troublesome, and his spit, which is very fœtid and offensive, amounts to about 20 oz. in the

twenty-four hours. The spit shows diplococci and other organisms, but no tubercle bacilli.

On examination of the chest, there was found at the left base an area of dulness, the upper limit of which was curved, reaching its highest just about the line of the scapula, where it was 3 in. above the base of the lung. Over this area the breath sounds were feeble, and crepitations were to be heard. Believing that this was an empyema which had burst into the bronchus, I tried with an exploring needle to strike pus at the lung base. I was unsuccessful, however, at this time, and as the patient declined any more serious operative interference, he was treated in the ordinary way with rest, good diet, open air, antiseptic inhalations, and intertracheal injections of menthol. He improved considerably, and on 24th April the amount of spit had diminished to about 4 oz. in the twenty-four hours. It was still fœtid, though much less so than formerly. Feeling better he requested to be discharged, and of course his request was granted. He returned on 3rd September feeling weaker, and with the cough and fœtid expectoration more pronounced than ever. His temperature now varied from 97° F. to 101° F., and his pulse from 76 to 104. On examination of his chest, we found that the area of percussion dulness, weak breath sounds, and crepitation was much the same as before. Thorough exploration being now advised and agreed to, the patient on 4th October was put under chloroform, and Dr M'Gillivray incised over the dull area, and resected about 3 in. of the eighth rib. The pleura was found thickened and adherent; the wound was packed.

On 11th October I tried to strike pus by means of an exploring needle, but was not successful. Further unsuccessful attempts were made on the 14th, 19th, 23rd, and 27th. The needle, however, on all occasions being felt to pass through firm fibrosed tissue.

On 29th October, on again exploring, pus was struck. It

was found upwards towards the root of the lung, at a distance of about 3 in. from the pleura. The opening was enlarged by probe-pointed forceps, and a drainage-tube, about 7 in. long, with only one side opening near the distal end, was inserted.

11th November.—The purulent collection is draining thoroughly. There is practically no cough nor spit.

22nd November.—Patient left for home, with orders to have the drainage-tube looked to by his doctor. It has been shortened to about 4 in., and some difficulty is experienced in introducing it at times. There is practically no cough nor spit, and he feels quite well.

The next record of him is in the following June (1902), the note being that he had been very well; but that one day he had coughed up some "stringy stuff," and that for three days after he had coughed up some fœtid pus, and passed it through the tube. Since then he had been well.

Lastly, on 14th December 1902, he reported that the cough, spit, and fœtor of breath had all gone, that the tube after having been gradually shortened, had been discontinued, and that the chest wound had healed. Examination at the left base revealed percussion impairment with weak breath sounds, but no crepitation.

In this case there had probably been several bronchiectatic sacculi, only one of which was properly drained. This drainage, however, and the fact that by the rib resection the lung had been allowed to fall in, had practically cured the condition.

The following is an example of a similar case, in which, unfortunately, interference was not successful.

H. P., a little girl of 9 years, was brought to me in the Infirmary in September 1889. The history was that she had been well till about two years previously, when, after exposure to cold, she had had "inflammation" on the right side. The

nature of the inflammation could not clearly be ascertained, but she had had pain in the side, cough, fever, and sweatings. After some two months, during which time she was mostly in bed, she suddenly coughed up about a teacupful of fœtid purulent matter. She seemed to have improved somewhat after this, but the cough and the expectoration of fœtid matter continued, though in smaller quantity.

On admission, she was weak and feverish, and was manifestly ill, and on physical examination of the chest, percussion impairment, cavernous breathing, and resonating crepitation were heard over the lower half of the right lung. Some harsh breathing and crepitation were found at the base of the left.

In this case I resected portions of two ribs, and on some six or eight occasions afterwards explored from the exposed pleura. Although on one or two occasions I seemed to strike pus, I never could establish proper drainage. On three occasions, into an opening made by probe-pointed forceps, a drainage-tube was introduced and left in, in the hope that the pus might find its way into it, but it was of no avail.

Becoming gradually weaker, and with the cough and fœtid spit persisting, the child died some six months afterwards. At the necropsy, the larger bronchi at the base of the right lung were found much dilated, and the surrounding lung tissue in a state of fibrosis. The pleura all over the lower lobe was firmly adherent, except at one spot laterally, where about an ounce of yellow serous fluid was found encapsuled. At the left base there existed, but to a relatively very slight extent, a similar condition of lung and bronchi.

In this case there had been, I believe, a small, possibly fœtid, empyema of the right chest, which, bursting through the lung, had sought to empty itself through a bronchus. In this it had succeeded to the extent of allowing pulmonary and parietal pleuræ to come in contact, but by its irritative and destructive effects on lung tissue and bronchial wall, it had

caused loss of tissue, fibrosis, and dilated tubes, this dilatation being favoured by the harassing cough which the infective purulent bronchitis necessitated. At first, and probably for months, the right side only had been affected, but latterly extension of mischief to the left lung had occurred, as the result probably of some of the fœtid purulent secretion finding its way to the bronchi at the base of the lung on that side, and setting up there similar irritative and destructive changes in connection with the bronchial, peri-bronchial, and pulmonary and pleural tissues.

The small encapsulated pleural cavity containing clear fluid is what is often found in such cases. It is produced by the continued shrinking of the fibrotic lung causing traction on the pleuræ, and so separating again the pulmonary from the costal. As we have seen, with the emptying of the empyema cavity, the pulmonary and costal pleuræ become approximated and adhere; and in many cases this adhesion is permanent. But when the bronchorrhœa persists, and especially must this be the case in fœtid cases, the irritative and destructive processes in bronchial, peri-bronchial, and pulmonary tissues must persist also, and hence the shrinking process must go on. This, while it will tend to induce further bronchial dilatation, may, at the same time, tend to draw again the pulmonary from the costal pleura. Thus loculi may be formed, in which of course fluid will accumulate.

Given an empyema which has burst into the lung, can we form any idea of how long it has taken for this rupture to occur? Bouveret states that in the majority of cases it does not take place till the second month, but there is no doubt that the time must vary practically indefinitely. In fœtid empyemas, it may occur very early. In one of my cases it occurred on the fourth day; but possibly the burst in this case may have been helped by the exploratory puncture,

which was made to reveal the nature of the effusion. In other cases it may not occur for many months.

The following table, which I have constructed from some of my Infirmary cases, may be interesting. It shows in the first column the duration of the patient's illness on admission; in the second column the number of cases admitted; in the third, the number of those which had burst; and in the fourth, the number of those latter which were fœtid.

Duration of illness	Number of cases admitted	Number which had burst into lung	Number of these which were fœtid.
1 month	14	4	3
2 months	9	1	0
3 „	6	3	2
6 „	5	4	3
9 „	2	2	2
12 „	2	1	0
over 12 „	5	4	2

When an empyema bursts into the lung, the amount of pus which may be expectorated varies very much. When the empyema is large, and when the rupture into bronchi is also large and free, the quantity may be so great as to produce the most serious symptoms. An instance of this kind occurred quite recently in my own knowledge. A man had developed a large empyema, and had been tapped, some 70 or 80 oz. of pus having been removed. The fluid collecting again, it was determined to have it opened and drained. On the very morning, however, on which the proposed operation was to be performed, in a paroxysm of cough the rupture occurred. Huge quantities of pus flowed from mouth and nostrils, and the patient died asphyxiated, drowned as it were in his own pus.

When the pus is less abundant, or the opening less free, the symptoms of the rupture are correspondingly much less severe. But even with small amounts, the dyspnœa and distress associated with the first gush may be somewhat alarming.

On the other hand, the symptoms of the rupture may be very slight indeed. An expectoration, slightly more yellow in appearance, or a fœtid odour in spit and breath, may be all the indications.

After a first evacuation, the purulent expectoration repeats itself at varying intervals. Sometimes the patient continues to half fill or to fill his spittoon daily, at other times very little spit comes up for two or three days, and then a large quantity comes all at once. Not unusually its amount is greatest in the mornings, and often the patient will notice that his position, lying on one or other side, or leaning forward, influences the amount brought up. In cases where an empyema which had burst into the lung is being opened and drained, one is often struck with the smallness of the amount of pus in the empyema cavity, as compared with the large amount of purulent matter which the patient has been expectorating. The meaning of this—obvious enough—is that the pus expectorated is not so much pus coming from the empyema cavity, as pus produced by the infective purulent bronchorrhœa which the pus from that cavity is setting up. In fœtid cases, this very copious bronchorrhœa is specially likely to be existent.

Lastly, as regards the part of the lung at which an empyema ruptures through. It will be apparent that as the great majority of empyemas affect the lower lobe, the opening or openings will be somewhere over that part, and will pass thence in the course of the pulmonary lymphatic vessels, inwards and upwards towards the root of the lung and large bronchi situated there. Copeland[1] states that the opening through the pleura is usually about the inferior surface of the upper or middle lobe; but seeing that an empyema may form at any part of the pleural surface, or may be interlobular, the rupture through must vary as regards position.

Empyemas opening through the Chest-wall.—Inasmuch

[1] *Dictionary of Practical Medicine*, 1866, p. 1009.

as an empyema may occur at any part of the pleural cavity, it may open at any part of the chest-wall; but inasmuch as most empyemas affect its lower parts, so there is a part of the chest-wall at which "pointing" occurs with greatest frequence. This is on the anterior aspect of the chest about the third, fourth, or fifth interspaces. The following case illustrates this very aptly, and as in it the empyema had opened as well into a bronchial tube, it illustrates also some other interesting features :—

Robert W., æt. 23, a cooper, was admitted to the Infirmary 17th March 1906, complaining of pain and swelling in the right side, and stating that he had been ill about five months. His family history was very good, his home surroundings were comfortable, but as a cooper by trade he was a good deal exposed to weather.

As regards previous illnesses, he had always been healthy until fifteen months ago, when he had an attack of pneumonia of the right side, which laid him up for nearly three months.

Present Illness.—He tells us that although the pain and the swelling in the side began about five months ago, yet he had never recovered from this attack of pneumonia, and had not been to work at all since. He had had to complain of nothing in particular, but just felt weak and unfit for work. He also felt short of breath on the slightest exertion.

About seven months ago, however, he noticed pain again on the right side below the angle of the scapula, and about six weeks later he noticed a bulging in his chest. This was in front over the fourth and fifth ribs at their costal-cartilage junction.

Shortly after this, his cough became more marked, and he spat up sometimes "thin stuff" and sometimes "thick yellow stuff." He noticed that after coughing up the yellow stuff, the swelling in front used to grow rather smaller. Gradually, however, this swelling increased in size, and

about three months after he had first noticed it, his doctor opened it, and got out about two saucerfuls of matter. It continued, however, to discharge for about two weeks. Next some redness and swelling showed itself about an inch above this opening. The doctor opened this also, and got more pus; but after it had gone on discharging for a week, it closed. A fresh swelling close to the site of the first one next occurred, and this again had been opened about twelve days before his admission.

State on Admission.—Height 5 ft. 9 in.; weight 9 st. 8 lb. He says he has lost about a stone in weight. His temperature for the most part varies from 97° to normal; his pulse about 88 per minute; respirations 24.

Respiratory System.—He has practically no pain in the chest at present. He has a cough, and at times a yellow spit. On physical examination, the right chest appears flatter than the left, and its respiratory movement is distinctly lessened. On percussion there is dulness over the lower part of the right side, extending from the seventh dorsal spine behind to the third interspace in front, and showing the curved line highest in the scapular region. Over this area the breath sounds are very feeble, and some crepitation and possibly friction can be made out at parts. Vocal fremitus is absent here.

Circulatory System.—This is normal, except that the heart is slightly displaced to the left.

Digestive System.—His appetite is only fairly good, and he has some thirst; but under this system, all that need be stated is that his liver is distinctly displaced downwards, in the nipple line, reaching fully an inch below the costal margin.

Hemopoietic System.—Red blood corpuscles 4,000,000, whites 9375. Blood film shows nothing special.

A needle introduced into the seventh space about the posterior axillary line easily found pus, and so on

23rd March a piece of rib was resected there, a drainage-tube introduced, and some 10 oz. of pus evacuated. Three days after the operation a good deal of hæmorrhage occurred, but with this exception the patient's condition rapidly improved. Cough and spit disappeared, and the discharge rapidly diminished. Lung expansion, however, was slow, so slow that on 5th June he was discharged with the tube still in, and in length about 6 in. He kept well, but it was not till about a year after this that the tube, after being gradually shortened, could be dispensed with. He is now well.

The first item which requires explanation is why the pus which, as is usually the case, is mainly in the lower and posterior part of the chest, should make its "point" about the fourth or fifth ribs anteriorly. What I am in the habit of regarding as the explanation of this is, that the suppuration tract from the empyema follows the course of the lymph stream.

The empyema pus, passing probably through the stomata of the interspaces, finds its way first into the intercostal muscle lymphatic vessels. There it, or its infective product, finds itself carried along the interspace in the same direction as the lymph, and this is mainly forward, its progress being probably aided by the respiratory movements. But having been carried forward sinuously along and through the intercostal muscles, it meets the collecting lymphatics, and these, as Sappey's well-known plates show, pass upwards for the most part to the root of the neck, but to some extent also towards the axilla. Hence pus at the lower and posterior part of the chest may indicate its presence by a "point" at the upper and anterior part.

The following case most aptly illustrates this :—

A. P., æt. 40, was admitted to the City Hospital 13th November 1907. He gave a history of a left-sided pleurisy twenty years before, and of cough, spit, loss of strength and

flesh for the last four months. He told us that his illness had begun four months ago by severe pain in the left side, and that three weeks ago a swelling had appeared over his sixth left interspace in the nipple line, which on being opened had discharged fœtid pus.

On admission he was very feverish, and had a distressing cough with fœtid expectoration. His left chest showed signs of great shrinking all over, with posteriorly the physical signs of fluid at the base. Exploration having revealed the presence of fœtid pus there, a drainage-tube was inserted, after which he improved for some weeks. Soon, however, his strength and reaction power began to fail; but the special features of interest in his case were that, as time went on, other suppurative "pointings" through the chest-wall occurred. These were as follows: The first point was, as stated, about the sixth rib in the nipple line, and occurred at the end of November. The second showed itself at the junction of the fifth costal cartilage and sternum, about the end of December. It was also opened, and discharged pus. The third showed itself by swelling in the left axilla about 15th January, and it was followed in a few days by the fourth, which occurred in the second interspace in the parasternal line. This latter quickly increased in size and discharged pus; the other, the fourth, not bursting at all. At the end of January, swelling was noticed above the left clavicle at the root of the neck, and on 5th February this was opened, and began also to discharge.

With these points discharging, some days more, some days less, sometimes one, sometimes the other, and with progressive failure in strength, this man lived on till the 28th March following.

At the *sectio*, the whole left lung was found shrunken and fibroid, except at the apex. It was firmly adherent to the pleura by thick adhesions all round, and its lobes were bound firmly together. The empyema cavity at the lower

posterior axillary region was quite small and empty, but blackened and softened tracts were seen, leading through intercostal muscles and pleuritic adhesions forwards and upwards. The bronchi, specially of the lower lobe, were dilated; but no distinct communications between the empyema cavity and the bronchi could be made out.

The outstanding fact which the consideration of such a case demonstrates is, that pointing in empyema does not in any real way mean emptying of the cavity. We have already seen that when an empyema is said to be finding its way into the lungs and bronchi, the patient may be daily bringing up many ounces of purulent expectoration, but yet that this may be largely bronchorrhœic, and that practically there may be little or no emptying of the empyema at all. With empyemas said to be bursting through the chest-wall, this also pertains; and indeed to a very much greater extent, for the pointing chest-wall abscess discharges pus which is practically all formed there. True, as in W.'s case, p. 141, in which the empyema had opened both into bronchi and through the chest-wall, it was noticed that when more pus was being discharged by spit, less was passing from chest-wall opening, and *vice versâ*. Still, any real emptying of the empyema cavity in either way could hardly be said to be occurring.

Lastly, although what has been said of empyemas pointing through the chest-wall describes what happens in the majority of cases, it does not apply to all. As will be seen later, an empyema may burst by a large direct opening right through the chest-wall, and moreover empyemas may be at unusual places, and may therefore show themselves at correspondingly unusual sites on the chest-wall.

Empyema opening through the Diaphragm.—The extreme infrequency[1] of the transference of suppurative

[1] For the rarity of subphrenic abscess following empyema, see Dr Archibald, *Brit. Med. Journ.*, 1906, vol. i., p. 1148.

processes through the diaphragm downwards from pleural to abdominal cavity need be no more than referred to, but taking into consideration with this its converse, viz., that suppurative and other inflammatory processes can readily pass upwards through the diaphragm from abdomen to thorax, an explanation of this infrequency should be forthcoming. This explanation, I believe, lies in the fact that the direction of the lymph flow in this region is, as was shown by Recklinghausen and others many years ago, from below upwards, that is to say, from abdomen to thorax, and from lower thorax to upper thorax.

True it is that we can conceive of a diaphragmatic empyema firmly walled in all around bursting through the diaphragm, just as, as we shall see by and by, it may burst directly through an intercostal space.

Still, as a rule, it may be asserted that in empyema suppurative spread tends to occur in the direction of the lymph flow, and that the fact that abdominal inflammations excited by thoracic, are just as uncommon as the converse of this is common, is to be explained in this way.

But now it is to be remembered in connection with lymph flow, that whilst the internal lymphatics of the thorax and abdomen, and the external lymphatics of the thorax course mainly upwards, the external lymphatics of the abdomen course mainly downwards to the large trunks in the groin. The importance of this consideration is exemplified in the following case. It occurred in my private practice, and is one of the very few instances of loin abscess following empyema which I have seen.

Mrs S., æt. 50, was first seen 8th January 1910. She had been ill for about ten days with a left pleuro-pneumonia, which after eight days had shown the usual indefinite crisis which foretells an empyema coming on in this way. When seen, her temperature was varying between 99° and 101°, her pulse was about 100. Her tongue was furred, she was

flushed and perspiring, and showed a well-marked herpes labialis. On physical examination of the left chest a slightly tympanitic percussion note was found over the upper lobe anteriorly, whilst posteriorly the signs were those of fluid. The heart was displaced slightly to the right, and there was no stomach resonance over the left lower ribs. The exploring needle readily found pus, which showed on standing a small soft white clot and a purulent deposit, and on staining, mainly streptococci.

Incision in the left posterior axillary line, resection there of a piece of the eighth rib, and drainage were at once carried out, and the patient showed rapid improvement. Drainage, however, became a little difficult to secure, and after seven weeks a large cold abscess showed itself in the left loin posteriorly. By careful probing of the empyema opening, and by the introduction of a longer tube, efficient drainage was re-established. The patient then rapidly improved in condition, and the loin abscess swelling completely disappeared.

In this case there might be some dubiety as to the exact track of the suppurative process which had induced the loin abscess. My own idea, however, was that there had been a small undrained loculus posteriorly at the attachment of the diaphragm to the ribs, that from this there had been a transference downwards and through into retroperitoneal tissue, and thus into the field of the superficial lymphatics of the abdomen.

And now that we have considered in detail Nature's own processes and efforts towards healing in empyema, by giving the pus the power to make a way out for itself through lung and bronchi, through chest-wall or through diaphragm, what is the broad general conclusion which we can draw? Certainly it is that whilst we should fully recognise Nature's efforts towards cure, we should not count upon them. True

it is that empyemas may heal spontaneously, at any rate by bursting through the lung, and equally true that this happens much more frequently than is generally supposed. Still we can say that whenever pus is found in the pleural cavity it should be evacuated, and experience warns us of the risks which are run if it be not so. This is specially the case in fœtid empyemas, as will be shown later.

CHAPTER IX

EMPYEMAS IN UNUSUAL SITUATIONS

THE great frequency of pleuritic processes at the apices in phthisis is well known, but as has been previously referred to, these lead to adhesions and thickenings rather than to fluid exudations, either sero-fibrinous or purulent. Nevertheless, as we shall see, in phthisis purulent effusions may occur there, and in the chapter on Pulsating Empyemas this will be referred to in some detail.

But apart from phthisis, apical or upper lobe empyemas are not so uncommon as they would seem, and as they are very important both from the diagnostic and therapeutic point of view, they warrant special reference.

The following is a good example of an upper lobe empyema :—

A. N., æt 40, a cabman, was admitted into the Royal Infirmary 11th March 1900, with profuse fœtid expectoration and fœtid odour of breath. His family history was excellent, his home surroundings were good, but he had taken a good deal of alcohol.

History of Present Illness.—Until November 1898 he had been perfectly strong and well. At that date, as the result of an exposure he caught a cold, and was ill in bed, with pain on the right side and cough. After two weeks of this, during a severe paroxysm of cough and pain, he suddenly expectorated a considerable quantity of foul-smelling spit. After this, he seems to have improved

considerably, the cough and spit gradually diminished, and in a few weeks he was able to return to work. During the summer of 1899, however, he began to cough again. The spit was not so copious nor bad-smelling, but as time went on, it became worse in this respect. In February 1900 he was so bad that he had to take to bed. His cough was severe, and his expectoration more copious and more fœtid than before, at times showing a little blood. He was also feverish and weak, and was troubled with copious perspirations. It was noticed that on some days there would be very little expectoration, whilst on others it would be very copious. The cough was apt to occur in paroxysms, and to cause great breathlessness and cyanosis. Finding himself getting rapidly weaker he came to the Infirmary.

State on Admission.—Although not extremely emaciated, he appeared very feeble and weak. His face was rather cyanosed, and very slight movement induced severe paroxysms of cough, so that a thorough examination could not fitly be made. His temperature varied from 97° F. to 102°, and showed distinct oscillations. His pulse varied from 90 to 130, and was very feeble. The cough was very troublesome, the slightest change of position bringing on a severe paroxysm, with copious fœtid expectoration. On examination of the chest, the left side was found to be normal to percussion. On the right side impairment of percussion note was met with all over the upper part, and dulness was found over the lower part, especially laterally, where it extended as high as the fifth rib. Auscultation, which was extremely difficult, owing to the feeble condition of the patient and the paroxysms of cough, revealed rhonchi and crepitations over the whole of the right chest, and very much enfeebled breath sounds over the right base posteriorly. Some rhonchi and crepitations were found also at the left side, especially at the base. Except that the urine contained a small quantity of albumin, the other organs appeared fairly healthy.

In this case I believed that I had to deal with a bronchiectatic process at the base of the right lung, the result probably of a fœtid empyema which had burst into a bronchial tube. I felt that the only possible way of doing good in the case was to drain, and I therefore on several occasions made exploratory punctures over the lower lobe posteriorly, to locate the cavity or cavities. Distinctly to my surprise, however, I could never strike pus, nor could I feel that the exploring needle passed through dense tissue. I had therefore to limit treatment to antiseptic inhalations, and to cardiac and general stimulants. The patient got rapidly worse, the cough and fœtid expectoration continued, some days more, some days less. Paresis of the right arm showed itself, and pericarditis supervened, death occurring on 11th April.

Necropsy 12th April. — Body somewhat emaciated. General pallor of surface, slight rigidity in lower limbs only.

Thorax.—Pericardium showed a recent purulent inflammation, its sac containing about half a pint of turbid fluid. The parietal layer was somewhat thickened, and covered with fibrinous exudation.

The left pleural sac contained about 6 oz. of turbid fluid; no adhesions.

The right pleural sac showed a very peculiar condition. Over the upper lobe of the lung anteriorly the pleura showed dense chronic thickening ($\frac{1}{8}$th in.), and was firmly adherent to the thoracic wall. On cutting into this, an empyema cavity was exposed, containing over 15 oz. of fœtid brownish pus. This cavity was confined to the anterior portion of the upper lobe of the lung by dense fibrotic adhesions, but closer investigation showed that the pus had burrowed both into the chest-wall in front, and into the lung below. As regards the chest-wall, it had burrowed through the first and second right intercostal spaces, and had caused gangrenous changes in the

muscular tissues there. As regards the lung, it had burrowed through the fibrous adhesions into the anterior portion of the lower lobe.

On section, this lung showed extensive collapse. The pleura all round was much thickened, and the lobes were all adherent. In the lower lobe anteriorly was found a cavity, of about the size of a walnut, with soft gangrenous walls, and into this cavity there opened three or four bronchi of about a quarter of an inch in diameter, with greatly thickened walls. Owing to the great collapse of the lung, the liver had risen high into the thoracic cavity. The other organs showed nothing specially noteworthy.

This was a case in which there had been a fœtid empyema over the upper lobe anteriorly, which had burst into the lung, and the fact that the purulent expectoration varied much at different times was due to the imperfect communication between the empyema cavity and the underlying gangrenous lung cavity and bronchi. The fact that the empyema cavity there was not diagnosed was probably due to its being comparatively empty at the times of examination ; we had noticed in examining this patient that we had often to wait till, in a paroxysm of cough, he had brought up a large quantity of spit. The dulness and feeble respiration at the base of the right lung posteriorly was accounted for by the great collapse of the lung and high position of the diaphragm.

In the following case the result, as regards diagnosis and treatment, was more satisfactory :—

James M., æt. 28, a surfaceman, was admitted to the Royal Infirmary 9th March 1904, complaining of pain in the left side of the chest, with severe attacks of coughing and foul-smelling spit, and stating that he had been ill for twelve weeks. His occupation and home surroundings were satisfactory, and he did not exceed as regards alcohol or tobacco. He gave no history of previous illness.

Present Illness.—About twelve weeks ago, when patient was at his work, he developed severe pain over the left chest, radiating up over the left shoulder. This continued for about a week, and then severe attacks of coughing began, soon accompanied by foul-smelling expectoration. Feeling then ill and weak, he left off work and called in a doctor. He tells us that the cough had always been worst during the night. He then had severe paroxysms which lasted for hours. The foul-smelling spit was very copious; he usually filled a spittoon during the night. He had not noticed any blood in the spit.

From time to time he had had attacks of shivering and coldness, and during his illness he had lost over a stone in weight.

On admission his temperature varied between 98° F. and 101°, his pulse was about 100 per minute, and his respirations about 26. On examination of the chest, we found on his left side impaired expansion over the upper lobe, with slightly diminished fremitus; also some dulness on percussion, especially over the second interspace, with weakened breath sounds and occasionally gurgling crepitation.

Examination of the spit showed no tubercle bacilli, and blood examination gave the following results:—Red blood corpuscles 4,600,000, h.b. 94 per. cent., white blood corpuscles 10,000. His other organs and systems were normal.

The treatment in this case was begun with rest, good diet, tonics, and intertracheal injections of menthol. Slight improvement showed at first, but as the days passed it was evident that no real improvement was being made. On 26th March, the note is that the cough and expectoration were as bad as ever, and that there had been some slight hæmoptysis. Surgical interference was determined on, and on 30th March he was chloroformed, and Mr Cathcart incised and resected about 2 in. of the second rib anteriorly. An exploring needle introduced here readily found pus, partly under the

pleura and partly in the lung tissue. By probe-pointed forceps, the track was enlarged, and a drainage-tube was introduced, some ½ oz. of very fœtid pus escaping. Gradual improvement followed this operation; the patient was able to get up on 12th April, and he was discharged on the 22nd.

In this case there had been, I believe, a small fœtid empyema over the left upper lobe, in the region of the second interspace. This had burst into the lung, and by the operation we were able to get sufficiently near it to secure efficient drainage.

Cases of this kind are not uncommon, and are often regarded as examples of lung abscess or gangrene. They often recover spontaneously, and the following is one of many examples of this which I have seen :—

David M., a cooper, æt. 42, was admitted to the Infirmary on 30th June 1903, complaining of very severe pain in the right side on coughing, and of bad smelling expectoration. He had always been a healthy man, and his surroundings at home and at work were satisfactory.

Present Illness.—This had begun six weeks before, as a very severe pain in the right chest and shoulder. After about a week of this, a cough came on, and soon he was bringing up a large quantity of fœtid spit. He then took to his bed, feeling very ill. He told us that when the cough began he noticed a little blood in the spit, but this had ceased. Since his illness began he has lost about 15 lb. in weight.

On admission his temperature varied between 97° F. and 101°, his pulse was about 90 per minute, and respirations 24.

Respiratory System.—His cough was very troublesome and painful. It was worse in the morning, and then there was the greatest quantity of spit. The amount of spit was about 10 oz. daily. It was thick, greenish, and purulent, and very fœtid. No tubercle bacilli were found in it, but there were numerous other organisms.

On examination of the chest there was found on the right side slightly diminished expansion, especially at the apex, with an impaired percussion note there, anteriorly and posteriorly. On auscultation over that apex weak but distinctly bronchial breathing was made out, with a few coarse crepitations.

With rest in bed, good diet, and antiseptic inhalations, this man rapidly improved, and on 14th August he was discharged, recovered.

As already stated, such cases have been described as being the result of lung abscess or gangrene. It will be evident, however, that in their mode of onset they resemble a pleurisy rather than a pneumonia; and further that the cough and expectoration as a symptom resembles what one finds in an extra pulmonary rather than in a pulmonary condition. Moreover, in the chapter on Fœtid Empyemas, examples of such are given in which the fœtid empyema appears as the primary occurrence.

But empyemas may form themselves in most inaccessible positions, and one such occurred in my experience lately. It was that of a man, æt. 45, who was admitted to the City Hospital believed to be suffering from some continued fever of the nature of typhoid. In the course of a few days, the occurrence of cough and some muco-purulent spit, with progressively increasing respiration rate and a distinct leucocytosis, attracted attention to his chest. Over the left lung, and especially at its middle part posteriorly, some percussion impairment with feebleness of breath sounds was recognised. An exploring needle was introduced time after time into suspected areas of his chest, but without result. After some ten days' illness he died, and on post-mortem examination a localised collection of about 5 or 6 oz. of non-fœtid sero-purulent fluid was found, between the internal surface of his upper lobe and the mediastinum. It will be evident that in

this situation the fluid was practically out of reach of the needle. Had it burst into the lung, however, exploratory punctures might have struck suppurating tracks, and so drained it.

The next case was, I believe, one of empyema, occurring about the middle part of the lung posteriorly, which had burst into a bronchial tube and also through the chest-wall at its upper and posterior part.

C. K., æt. 53, a traveller, was admitted to the Royal Infirmary 27th August 1903, complaining of pain in the right side, with copious, and at times fœtid spit. His family history was fairly good, and his surroundings at home and at work were quite satisfactory. He acknowledged to have taken at one time a good deal of alcohol. He had had practically no previous illnesses.

His present illness had begun about six months before. He had then caught cold, and was feverish, with pain in the right side, and cough, his doctor telling him he had pleurisy. After some weeks he recovered somewhat, and was moving about; but one day he had a severe fit of coughing, and he noticed a great increase in the amount of spit, and that it had a disagreeable taste and smell. This condition has persisted until now, in spite of all treatment. For some weeks, he tells us, there might be some improvement as regards cough and expectoration, and then for some weeks it would be worse again. At times there was blood in the spit. He has lost 3 st. in weight since the commencement of his illness, and feeling that no treatment was doing him any good, he came to the Infirmary.

On admission, he was found to be of rather poor muscularity; height 5 ft. 3 in.; weight 8 st. 4½ lb. His temperature varied from 97° F. to 99·5°; pulse was usually about 84, and respirations 24 per minute. His digestive, circulatory, urinary, integumentary, and nervous systems were all

normal. Blood examination showed a leucocyte count of 21,000.

As regards his respiratory system, he had a very severe loose cough, with a large quantity of sputum, sometimes amounting to 20 oz. in the twenty-four hours. It was muco-purulent, often brownish red in colour, and, as was expected, showed no tubercle bacilli. The amount of fœtor in the spit varied from time to time.

Examination of the chest showed deficient movement on the right side all over, with an impaired percussion note posteriorly, becoming dull at the base of the lung. In the right interscapular region a curious condition was revealed. Pain was complained of there, and on palpation there and all down the vertebral border of the scapula, bulging with crackling could be felt whenever the patient coughed. On auscultation all over the posterior part of the chest, the breath sounds were very faintly heard, becoming specially faint towards the base. All over the right side, and specially at the lower part, crepitations could be heard.

Believing that this was a case in which an empyema, limited to the posterior border of the lung, had burst into a bronchial tube and through the chest-wall posteriorly, and believing that the rupture into the bronchial tube had set up bronchiectatic and fibroid changes at the lower part of the lung, I thought that my best chance of draining would be by getting at it from below. I accordingly got Mr Caird to remove for me about 2 in. of the seventh rib posteriorly. In doing this, he opened into a sinus in the fascia close to the chest-wall, which led up to a point opposite to the spine of the scapula where the bulging on coughing had been noticed. A long drainage-tube was put into this sinus, and the resection wound was packed.

Some improvement followed from this, as a good deal of discharge occurred from the sinus, but on my return from my holidays in the beginning of October, I determined to

explore more thoroughly. Exploring needles passed into the lung from the resection opening had revealed that the whole lung base was indurated, in fact it felt as if the needles were passing through cartilage. In a straight line inwards, about 3 in. from the pleura, a purulent collection was struck, and this communication having been enlarged, a drainage-tube 5 in. long was introduced.

From this time the patient's condition showed marked improvement. With free drainage along the two tubes the expectoration soon ceased. By the end of October the upper tube had been discontinued, and at the end of November he was discharged, remaining however an out-patient of the hospital, and coming up every few days to get the tube cleaned and replaced.

In this case there had been, I believe, a fœtid empyema, situated about the middle part of the lung posteriorly. It had burst into a bronchial tube, probably about the root of the lung, and also through the fourth interspace in the inter-scapular region.

I have to add that for some two years this patient did very well, but the drainage-tube had to be kept in. At the end of this time he died, I am informed; but as I had lost sight of him, I cannot tell the exact cause.

In the following case, the empyema was located over the front of the chest:—

Matthew N., æt. 42, engaged in the rubber works, was admitted to the Infirmary on 8th February 1904, complaining of pain in the right side of the chest, and of cough with copious offensive expectoration. About two months before his admission, the right half of his tongue had been removed by Mr Caird for malignant disease, and though he convalesced excellently, some three weeks after the operation he developed a cough, some chest pain, and became feverish. He remained in bed for about ten days, and then feeling better, and all

fever having gone, he got up and began moving about. The cough, however, continued, and he then noticed some foul-smelling spit. As this was continuing, and as he felt that he was not gaining strength, he came into my ward.

On admission he was thin—weight 9 st. 7 lb.—and distinctly anæmic-looking. His temperature varied from 99° F. to 102°, his pulse was about 100, and his respirations about 30 per minute. He complained of persistent cough, worse at night, but constantly present. The cough was paroxysmal, a paroxysm lasting sometimes for several minutes. His spit was copious, sometimes as much as 20 oz. in a day. It was very fœtid, containing numerous bacilli, cocci, and streptococci —no tubercle bacilli. He complained also of pain in the right side, the pain being stabbing in character, and being specially severe when he coughed or took a deep inspiration.

On examination of the chest, indications of disease were found only over the lower half of the right lung anteriorly. Here, with deficient movement and vocal fremitus, an impared percussion note was found from the level of the third rib down to the liver dulness. This area was also tender to percussion. All over it the breath sounds were very feeble, and slight friction was audible at its axillary border.

A blood count gave red blood corpuscles 4,300,000, whites 23,000, h.b. 100 per cent.

On 9th February, an exploring needle introduced in the third interspace at the nipple line readily found pus, and next day Mr Caird, under cocaine and adrenaline, incised at this point and introduced a drainage-tube, about an ounce and a half of exceedingly fœtid watery pus escaping. Gradually both expectoration and drainage fluid diminished, though from time to time exacerbations both in spit and discharge were noticed. By the end of March he was distinctly better, and he was discharged on 22nd April with the wound healed, with a very slight cough and non-fœtid expectoration, and weighing about 10 st.

As can easily be understood, empyemas affecting the lower or diaphragmatic surface of the lung must often be looked for. This is, in some degree, because inflammations of the pleura may occur there, just as they occur over the costal or mediastinal portions. But in a still more important degree, it is because abdominal inflammations of various kinds are not infrequent, and their products, or the organisms associated with them, tend to be drawn by the lymphatic stream through the diaphragm into the thoracic cavity, and so to set up inflammations of the diaphragmatic pleura.

Accordingly, in addition to what may be called primary diaphragmatic empyemas, we must be prepared to meet with secondary ones, following on and associated with inflammatory and suppurative inflammations of spleen, liver, or kidney, with gastric ulcer and appendicitis, and with the many forms of pelvic inflammation occurring in puerperal conditions.

But now it must be evident that with most of these diaphragmatic empyemas, the tendency will be for them not long to remain diaphragmatic. That is to say, whether a diaphragmatic empyema has been caused by infection through the diaphragm or not, there will be a tendency for it to spread in time to the costal pleura, and so to behave like an ordinary empyema. Hence, then, as this chapter concerns itself only with empyemas occurring at unusual situations, it might seem that it should deal only with those diaphragmatic empyemas which remain limited to the diaphragm by adhesions. As will be seen, such empyemas do occur, but it can be understood that as such a distinct class cannot well be recognised.

Of diaphragmatic empyemas caused by liver affections, I can relate several examples, and the following is one which I saw in the Infirmary many years ago :—

A. A., æt. 32, was admitted to the Royal Infirmary 12th November 1880, with right-sided pleurisy and effusion. He had had a rigor on 28th September previously, and com-

plained then of great pain in the back. On 5th October friction was heard beneath the right nipple, and there was some impairment of the pulmonary resonance at the right base. On 14th October his temperature rose, and he expectorated a quantity of pus. His temperature then fell, and he felt better; but on 26th October his temperature again rose, and he expectorated more pus. He then was much better till 10th November, when he was suddenly attacked with great shortness of breath. On his admission to the Infirmary on 12th November, a right pleuritic effusion was now quite apparent, and on puncture 15 oz. of fœtid sero-purulent fluid were drawn off. Only very slight improvement followed this, and soon he became worse. The difficulty of breathing increased, and there was copious expectoration. On 21st November the expectoration had an intensely fœtid odour, and on the evening of that day, he sank, apparently from asphyxia.

Sectio.—On the thorax being opened, a quantity of fœtid gas escaped from the right pleural cavity. The right lung was totally collapsed, and the right pleural cavity contained about 10 oz. of fœtid pus. The whole visceral surface of the pleura was covered with purulent lymph, and an opening about the diameter of a goose quill was observed in it, at the lower part of the lower lobe, and running into the lung substance. On tracing this opening downwards, it was found to run into the upper part of the right lobe of the liver, where it ended in a large cavity, with a somewhat well-defined limiting membrane, and in which was found, quite detached, a rounded cheesy-looking mass, half an inch in diameter. On cutting into this mass, a cavity was found in its centre, containing a little yellowish fluid.

The opening in the pleura could also be traced upwards into the lung substance, where it ended in a sloughing-looking cavity, into which a bronchus opened. Thus a free channel of communication existed between this cavity and

that in the liver. Lying in the right pleural cavity was a large piece of gelatinous-looking membrane, having a few yellowish patches on its surface. Microscopic examination of this showed that it was the sac of a hydatid cyst which had made its way through the above described opening into the pleural cavity.

In this case the pathological sequence was probably as follows :—

(1) A liver hydatid, bulging up the diaphragm, had set up a chronic diaphragmatic pleurisy and caused a large number of adhesions.

(2) Acute inflammatory and suppurative changes had occurred in the hydatid cyst, on or about 28th September, and this had set up an acute pleurisy, with probably sero-fibrinous effusion, about the lower part of the lung, but beyond the area of the old adhesions.

(3) Meanwhile the pus from the hydatid cyst was burrowing through the diaphragmatic adhesions into the lung, and onwards to a bronchial tube. It reached the tube on 4th October, when a quantity of pus was expectorated.

(4) Some relief followed this escape, but the track or tracks got blocked, and the pus was reaccumulating till 26th October, when a fresh escape through a bronchial tube occurred.

(5) Again distinct improvement followed, but as the result, at any rate to some extent, of the increasing pleuritic effusion, causing the lung to collapse, some of the old adhesions were torn through. An escape of the hydatid pus into the pleura then occurred (10th November).

(6) Development of alarming symptoms, due to the further escape of pus and hydatid tissue into the pleura, to greater collapse of the lung, and to progressive decomposition of the now purulent pleural fluid.

It is interesting to notice that a case somewhat similar to this is quoted by Trousseau (Vol. IV., p. 266). It concerned a man who had been under treatment for right-sided pleurisy with effusion, and who after some weeks began to expectorate yellow pus in which bile could be recognised. "Soon his symptoms began to assume an exceedingly serious character, and during the last days of his life his breath and sputa were exceedingly fœtid.

"At the autopsy there was found in the liver an enormous cyst, still containing some acephalocysts; it had opened into the bronchial passages through a gangrenous portion of pulmonary tissue."

Then he adds: "The pleuritic effusion recognised during life still existed, and curious to relate there was no communication between this effusion and the bronchi."

Here it will be evident that the suppurating cyst had been gaining access through the diaphragm and lung to the bronchi, and that there had been no rupture of the previously existent diaphragmatic adhesions, as in the former case.

How salutary the effects of these diaphragmatic adhesions may be can be witnessed also in dysenteric conditions. In such cases also we not infrequently find liver abscesses evacuating themselves through lung and bronchi without having produced any acute pleuritic symptoms at all.

In the following case the empyema was, I believe, of liver origin, and at first diaphragmatic, but it in time found its way through the adhesions and over the lower costal portion of the lung:—

William B., æt. 34, formerly a soldier, was admitted to the Royal Infirmary 8th April 1905, complaining of very severe pain and feeling of distension in his right flank. As regards previous illnesses, he had had typhus fever twenty-five years before. He had lived in India for several years, and been much exposed to dysentery and malaria,

although he had never suffered seriously from either. Recently he had served in South Africa, and had suffered there from rheumatism and a "poisoned" hand. For the last year or two he had been leading a healthy and careful life.

His present illness had begun by a severe shivering fit three weeks previously, for which he had taken quinine and a glass of whisky, and had gone to bed. For the next few days he was rather better, but felt listless and unable to go about. Then a week after his first rigor he took another and more severe one. He then suffered from great pain in his right side and liver, which rapidly grew worse, so that he was unable to rest in any position; he was afraid to cough, and his breathing was extremely difficult. He was treated with opium and quinine, and after a few days when some signs indicative of effusion were showing themselves at his right base, he was punctured there, but only a few drops of clear fluid were obtained. This was followed by no improvement, and in a few days more he was admitted to the Royal Infirmary.

On admission, 8th April, his temperature varied from 98° to 100° F., showing distinct oscillations with sweatings; his pulse was comparatively quiet, 80 to 90 per minute, and respirations about 28 per minute. He reclined constantly on his left side.

On examination, his right chest was found immobile and distended in the region of the liver. Distinct signs of pleural effusion were made out on the right side, extending as high as the sixth dorsal vertebræ posteriorly, and as the fifth rib in the axillary line. Over the ninth and tenth ribs in the scapular line, an area of œdematous swelling was found, which was extremely tender to pressure. Anteriorly, the lower border of the liver could be traced downwards to about 4 in. below the costal margin.

Feeling sure that pus was present either above or below

the diaphragm, repeated explorations were made at various depths and over the entire lower chest, but these discovered only a little blood-coloured serum, or clear fluid, or nothing. At length, on 19th April, pus was discovered by a puncture in the ninth interspace, about the posterior axillary line, and the chest was at once opened and drained, 40 oz. of non-fœtid pus escaping. Rapid improvement followed, the liver returning in a few days to its normal position.

In this case there had been, I believe, a liver suppuration, which, boring through the diaphragm, had set up a diaphragmatic empyema, the extension of which to the costal wall had for long been prevented by the presence of old adhesions.

When now in the causation of diaphragmatic empyemas we pass from inflammations of the liver to those of other abdominal organs, we meet for the most part with empyemas, of which it can be said that they only began as diaphragmatic ones. Thus, spleen abscess, perinephritic abscess, appendicitis, and pelvic inflammations can all act as causes of empyemas, but of empyemas which are only rarely limited to the diaphragm, although they have been caused by absorption through it. In such cases, diaphragmatic perforation, such as was present in A. A.'s case, and probably also in that of W. B., is not to be expected. Indeed, it may be said that simple proximity of the focus of suppuration to the under surface of the diaphragm seems to be of very little consequence in connection with these empyemas. In my own records there is certainly a greater number reported as following pelvic inflammations and appendicitis than as following inflammations of liver, spleen, or kidney.

Further, when we reflect that such empyemas are due to lymphatic absorption through the diaphragm, we can understand that the pleuritic process set up may not always be a

purulent one. Although with a suppurative splenitis, perinephritis, or appendicitis, when a pleurisy does occur, it is likely to be purulent, it may be simply sero-fibrinous, and capable of being reabsorbed spontaneously, and although when the abdominal suppuration is fœtid, as it often is, the pleural is likely to be so also, this does not always hold.

Under the head of empyemas in unusual situations, reference must also be made to empyemas occurring between the lobes of the lungs. Forming as it were a purulent cyst between the lobes, an interlobar empyema seems to have a special tendency to burst through the surrounding lung tissue into a bronchial tube, and all the more frequent is this occurrence likely to be if the empyema is a fœtid one. These empyemas are probably much more common than is generally supposed. Trousseau has specially referred to them, and alludes to the likelihood of their often being mistaken for abscesses of the lung. Clinically their recognition may be very difficult, the special indications being the symptoms of a pleurisy or pleuro-pneumonia, with the existence of an area presenting, perhaps not very definitely, the physical signs of effusion in the region of one or other of the interlobar fissures of the lung.

Lastly, in this chapter, a reference must be made to bilateral or double empyemas. The close association between bilateral sero-fibrinous pleurisies and tubercle might suggest a similar relationship as regards empyema. But a survey of one's own experience and of the literature on the subject does not appear to bear this out. Indeed, double empyema seems to occur with special frequency in the young and vigorous, associated like ordinary empyema with acute pleuro-pneumonic and pleuritic processes. It is, however, a distinctly rare condition.

In the very acute cases, the bilateral existence of the empyema is marked early in the course of the malady, and

as can be readily understood, the general distress and respiratory embarrassment may then be such as to cause great anxiety. In the less acute cases, the second empyema may present itself for treatment after the first has been opened and is being drained. With treatment these cases, however, as a rule progress wonderfully favourably.

It has to be borne in mind that although double empyema is clinically very rare, yet that pathologically it is not so very uncommon. In grave septic and pyæmic conditions associated with pulmonary embolism and suppurative developments, it is not unfrequently found on the post-mortem table.

CHAPTER X

FŒTID, PUTRID, OR GANGRENOUS EFFUSIONS

AS was indicated in the last chapter, fœtid empyemas are not uncommon as secondary to sub-diaphragmatic inflammatory and suppurative processes. It has to be borne in mind, however, that similar fœtid effusions are not at all infrequent as the result of inflammatory processes affecting the lungs and pleura alone. In some of such instances, the lung inflammation will be found to have gone the length of the production of a patch of lung gangrene, and this might be regarded as the determining cause of the nature of the effusion. In other instances, however, a pleuritic effusion will be found presenting all the fœtid or gangrenous characteristics, and yet with no such gangrenous lung change to account for it.

These points are well exemplified in the two following cases, in one of which gangrene was present, in the other not:—

J. B., æt. 53, a domestic servant, was first seen by a doctor 3rd April 1887. She stated that she had been exposed to cold eight days previously, that on the evening of her exposure she had had a shivering, and that ever since she had felt very ill. She had great pain in the left side on taking a long breath and on coughing, and she stated that this pain and cough had been getting so much worse, and that she had been feeling so weak, that she had been compelled to remain in bed. Her doctor had found some impairment of the percussion note about the inferior angle

of the scapula, with weakening of the breath sounds and slight crepitation, and had diagnosed a pleuro-pneumonia, and sent her to the Infirmary.

On admission her temperature was 103·2° F., pulse 120, very soft and compressible, respirations 25 per minute. She had cough, with scanty mucous expectoration, severe pain in the left lower and lateral region of the chest, and the physical signs indicated pleural effusion, the dulness reaching nearly as high as the spine of the scapula posteriorly, and the fifth rib in the axillary line. On the second day after her admission, the pain was somewhat easier, but with the dyspnœa, fever, and sweating, her general condition was manifestly worse. An exploratory puncture was made, and a hypodermic syringeful of greyish-yellow, watery, and very fœtid pus, containing bacteria, was drawn off. On the same day the chest was freely opened, and about 60 oz. of a similar fluid evacuated. Little improvement followed, and the patient rapidly becoming more collapsed, death occurred on the evening of the following day.

On post-mortem examination, the upper part of the left lung was found adherent to the chest-wall. The lower part was collapsed and œdematous, and its surface was covered by a layer of greyish-yellow false membrane. On section there was found on the posterior surface of the lower lobe a patch of gangrene, shaped like a very flat triangle, the base of the triangle corresponding to the pleural membrane, and being from above downwards about 2 in. in extent. The aorta was atheromatous, and the heart distinctly fatty.

In this case, it will be noticed, that there had been no fœtid spit. I am of opinion, however, that had a better nourished heart enabled the patient to live a little longer, the characteristic fœtor of spit and breath would have been present. It will be evident, however, that the lung gangrene can be regarded as the cause of the fœtid effusion.

In the following case, however, there was no lung gangrene:—

J. V., æt. 58, was admitted to Ward 23 on 4th July 1888. His family and personal history were good, and he stated that he had been exposed to cold ten days previously. On the day of his exposure he had had a rigor, and felt very unwell. Next morning he had pain in his left side, which was very severe and caught his breath. He remained in bed, but finding no improvement after some days, he came to the Infirmary.

On admission, his pulse was 120, regular and soft, his temperature varied between 99° and 102° F., his respirations were rapid and shallow. Examination showed dulness over the left chest, beginning in front at the fifth rib, and in the axillary line at the fourth rib. Posteriorly, percussion impairment began about the spine of the scapula, becoming more marked on passing downwards. Over the dull area the respiratory sounds were weak, and over the third, fourth, and fifth interspaces anteriorly friction was detected.

The distress continuing, with shiverings, sweatings, and some wandering at night, and the signs of effusion becoming more marked, the chest was punctured on 20th July. The presence of fœtid pus was demonstrated, but in spite of removal of this by drainage, he died on 26th July. Fœtid spit was never present.

On post-mortem examination the following condition was found:—"On the left side a loculated empyema occupying the posterior half of the lower fourth of the upper, and the upper three-fourths of the lower lobe. The pleuræ over the remaining portion of the lung united by recent fibrinous adhesions. The empyema did not quite reach the posterior border of the lung at its lower extremity, about 3 in. of lung being uninvolved in it. The loculus contained some dark grey intensely fœtid fluid, and the pleura was covered by a greyish-yellow irregularly eroded membrane. On

section, the upper half of the lung was congested, the lower lobe carnified."

In this case there can be no doubt that the condition was one of fœtid empyema alone.

But now a very interesting point crops up for consideration. We know that of all empyemas these fœtid ones are specially prone to find a way out for themselves through lungs and bronchi, and we know also that in the tracks which they make through the lung in doing this, necrotic and gangrenous areas can be recognised. It follows, then, that when on post-mortem examination we meet with such empyemas which have been burrowing through the lung, it may be difficult to determine whether the empyema has or has not been the primary process. It will be evident that in a case in which the primary process has been a superficial patch of lung gangrene, and in which this patch is breaking down, the post-mortem appearances must be very similar. This is shown well in the following two cases:—

Henry C., æt 39, a miller, was admitted to the Royal Infirmary on 11th May 1901, complaining of cough and fœtid spit and weakness, and stating that he had been ill for some months.

His family history showed a distinct tendency to lung trouble. His home surroundings were good, and he had never been ill until the present illness began.

In the previous November he had been exposed to cold, and developed a cough and hoarseness. In February he had what was believed to be influenza. He was laid up in bed for some time, and a cough and spit developed. This had rapidly been getting worse, and his spit had been becoming fœtid. For the last month he had been very ill, he had lost weight, and had noticed that his feet and ankles were often swollen at bed-time.

On admission his temperature varied from 100° to 102·4°,

his pulse was over 100, and his respirations about 30 per minute. He had paroxysms of rapid and painful breathing and cough. The sputum was very copious, the patient filling a spittoon and a half in twenty-four hours. It was liquid, yellowish, frothy, and extremely fœtid, as was also his breath.

On examination of his chest, evidence of the presence of fluid on the left side was recognised, with some friction anteriorly.

This man died suddenly before operative interference could be carried out, and at the post-mortem examination the following was found :—

On opening the thorax the left lung was found to be adherent to the pleura at its upper and anterior parts, the lower lobe was collapsed inwards as the result of the presence of a large amount of pus. This was thick, greenish-yellow in colour, and on pressing the lung *in situ* bubbles of air could be seen to rise up through it.

On examining the collapsed lower lobe, it was found to be covered with a softened purulent false membrane, and on its posterior surface and in its interior, necrotic cavities communicating on one hand with the bronchi, and on the other with the cavity of the pleura, were found.

In this case I think there is no doubt that the fœtid empyema had been the primary process, and that by bursting into the bronchi it had produced those softened and necrotic conditions of lung.

John M., æt 64, a labourer, was admitted to the Royal Infirmary on 4th February 1899, complaining of pain in his left side, of severe cough, and of copious bad-smelling spit. He could give no definite history of his illness, but as far as could be gathered, he had been ill for over a week. He had been living in a lodging-house, and the people becoming alarmed at his illness, sent him to the Infirmary.

On admission, his temperature was 97·6° F., rising at

night to 100·8°. His pulse was slow, about 60, and very feeble, his respirations about 20 per minute. Examination of his chest revealed dulness and feeble breath sounds at the right base. A needle introduced there revealed the presence of fœtid pus. The patient died next day, and the following was found on post-mortem examination :—

"*Right Lung.*—The upper lobe showed a condition of fibroid change, with firm adhesions to the pleura. The lower lobe was somewhat collapsed from the presence posteriorly of about 15 oz. of fœtid pus. The collapsed portion posteriorly was covered with thick fibrinous and purulent exudation. On section there was found about the middle of the lower lobe what appeared to be a small abscess about a quarter of an inch in diameter, and between this and the overlying pleura the lung tissue was gangrenous.

"His aortic valves were incompetent, his heart was enlarged and dilated, and indications of chronic nephritis were also present."

In this case at the time it was thought that the lung gangrene had been the primary change, but it was evident that of this one could not be certain. An example of the difficulty of distinguishing between those two conditions clinically occurred in my experience lately. It was that of a man æt. 55, who was operated on for a fœtid appendicitis, and who, for five or six weeks subsequently, appeared to be progressing most favourably. Some seven or eight weeks afterwards, however, he began to exhibit slight fever, impaired appetite, and loss of flesh, but yet for two weeks after this careful and repeated examinations revealed no indications of trouble in abdomen or chest. After two weeks more, however, it was noticed that though he had no cough nor spit, yet that on occasions marked fœtor of the breath was present for short intervals, and that he preferred not to lie on his left side. A slight leucocytosis was also found. After

another week careful examination showed at his left base in the scapular line a small area of percussion impairment, weakened breath sounds, and an occasional crackle. The exploring needle obtained here a small quantity of bright yellow, extremely fœtid pus, showing microscopically, streptococci, staphylococci, and bacilli like the bacillus coli. Free incision a few hours afterwards revealed hardly any pus, but a small gangrenous lung cavity with adherent pleuræ all round. In spite of drainage and other treatment, death occurred after another week, and although paroxysms of cough developed and "gulps" of fœtid breath showed themselves at intervals, there never was any fœtid spit.

Of cases of fœtid empyema which from the clinical aspect presented evidence of the lung gangrene having been primary, and which terminated favourably, I have had no examples so good as the two following, which I quote:—

(Wagner, *Berlin. Klin. Woch.*, 6th Sept. 1880). A lad, æt. 17, was admitted to Hospital 14th October 1878, suffering from dysentery. For this he was treated with salicylic water injections, and in about two weeks he had quite recovered. On 6th November, however, he became again ill with symptoms which looked like those of an acute pneumonia of the left lower lobe, viz., rigors, pain in the side, cough, and rusty viscid spit; pulse 120, temperature 104° F. On the eighth day of this pneumonia the temperature began to fall gradually, and the lysis was complete on the 21st, the percussion dulness also getting gradually less. After the temperature had been normal for three days he was again seized with pain in the left side, and pleurisy with effusion was diagnosed. On the twenty-fifth day an exploratory puncture revealed the presence of a white thinnish pus of a disagreeable odour, and showing by the microscope bacteria. Paracentesis was performed, and 700 grammes of a disagreeable but not quite putrid character were removed. For three days after this a discharge continued,

but on the third day a piece of gangrenous lung tissue (2¼ in. by nearly 1 in.) was removed through the wound opening. All the symptoms improved after this, but during the healing of the wound it was noticed that on washing out the pleural cavity with carbolic solution, some of the fluid was expectorated, indicating the existence of a communication between this cavity and the bronchus. By 16th February the wound had healed, and a yellow spit was all that remained to tell of the lung trouble. This by 16th June had disappeared, leaving the patient quite well. The explanation of these events Wagner believed to have been that from the dysenteric bowel an infective process of embolic nature set up the pneumonia, and that this was followed by lung gangrene. He also considered that the fact that the fluid in the pleural cavity was at first not very fœtid was due to the gangrenous part of the lung being for a time covered in by the pleura and layer of inflammatory false membranes, so that only a fractional amount of putrid material obtained access to the effusion. The subsequent communication with a bronchial tube and consequent expectoration of carbolic solution was, he believed, due to the corroding action of the fluid on the cicatrising portion of lung surface.

The second is a very similar case reported by Ewart (*British Medical Journal*, vol. i., 1887). A boy, æt. 10, while convalescing during the fourth week of typhoid fever, developed symptoms which looked like those of peritonitis, but which shortly indicated left-sided pleurisy with effusion. After a week he was aspirated, and ¾ pint of thick shreddy pus was removed, with relief to the most urgent symptoms. The following day the chest was opened freely in the anterior axillary line (fifth space) and in the scapular (ninth space), a large quantity of pus escaping. Free discharge continued, but no injection was used. On 15th June perflation was performed under the spray in the manner described in the *Lancet*, 31st July 1886. The air was delivered into

the centre of the chest through the anterior opening, and allowed to escape only at the posterior. The result was the expulsion of fœtid pus, of a piece of necrosed lung, and of heavy false membranes. A smaller piece of false membrane was expelled by perflation the next morning, and a small piece on the third day. From this time the fœtor ceased and the amount of pus decreased rapidly. On the eighth day the discharge was turbid and serous, and it remained serous until the end. Both wounds were closed on the thirtieth day from the date of the incision.

In both these cases it will be observed no mention is made of fœtid spit. It is probable, however, that either it or fœtid breath existed to some extent. Both in lung gangrene and in fœtid empyema bursting into lung, one sometimes meets with instances in which the characteristic odour of spit and of breath shows itself only at intervals of hours, or even days.

In the following case the condition was, I believe, one of fœtid empyema alone; but extensive gangrenous changes were produced by the pus, not on the lung, but on the costal tissue :—

Robert M., æt. 41, shoemaker, was admitted to the Infirmary 10th June 1902, complaining of cough and chest pain, and stating that he had been ill for about two months.

History.—His father and mother are dead, cause unknown. One brother dead from heart disease, one alive and healthy. He has two children, both healthy. His food is good, and he has been temperate as regards alcohol. His home is comfortable, his work is sedentary, and is carried out in a rather confined atmosphere. As regards previous illnesses, he gave a history of acute rheumatism ten years ago, and of mild pneumonia in 1900.

His present illness seems to have begun about two months ago; he felt himself very weak, and he developed a

cough, with a yellow opaque spit, sometimes streaked with blood. His condition seems to have become rapidly worse, until he was unable for his work. Applying then to the Infirmary, he was admitted as stated on 10th June.

State on Admission.—He was a fairly developed man, rather poor as regards muscularity; his temperature was about 99·5° F., pulse 84, respirations about 20 per minute. He had a cough and muco-purulent spit, which, however, showed no tubercle bacilli. On examination of his chest, the physical signs were mainly those of bronchial catarrh. All over his lungs, especially posteriorly, the breath sounds were harsh, with prolonged expiration and some crepitation. At the right apex, percussion gave a note that was slightly impaired, and here the breath sounds were harsher and the crepitations a little more marked than elsewhere. All the other systems were fairly healthy.

We concluded, therefore, that we were dealing with a case of bronchitis, with some catarrhal pneumonia at the right apex. We kept in mind that there might be some tubercle, but as stated, careful examination of the spit revealed no bacilli. We treated him by keeping him out in the open air as much as possible during the day, and by a good diet, and for the cough we gave him a little iodide of potass combined with digitalis, as his pulse was rather soft.

In this way he improved considerably. Four days after his admission his temperature fell to between 97° and 98° F., his pulse varying between 76 and 92, and he described himself as feeling better. On 6th July, however, it was noted that the evening temperature rose to nearly 100° F. and the pulse to 104. Next day his temperature was nearly 101° F. and his pulse 122. On 8th July he suddenly began to complain of severe pain in the left side, aggravated by breathing and by cough. His temperature was now over 101° F., and pleuritic friction was detected over his left side. The pain rapidly became so severe that the side was strapped, and ¼ grain of

morphia was administered hypodermically. Next day the temperature was still higher, 102·8° F., pulse 124, respirations 44. The chest pain and cough were very severe, so that the ice-bag was applied to the side, and morphia again administered. On 10th July distinct signs of pleuritic effusion were found, and this went on increasing in the following days. The temperature chart was noticed to be showing oscillations from 102° downwards, and the patient was sweating a great deal. On 18th July the presence of fœtid pus was revealed by the exploring needle, the pus showing microscopically diplococci in large numbers, along with a bacillus like the bacillus coli. On 20th July Mr Caird incised the chest-wall, and evacuated about 20 oz. of dark green, fœtid, and very liquid pus. At the operation, marked œdema of the chest-wall was observed. A large drainage-tube was introduced, and the wound was dressed. The next day the patient expressed himself as suffering less pain, and the temperature fell at night instead of rising. The subsequent progress may, however, be summarised thus—

The wound, owing to copious fœtid discharge, had to be dressed four or five times in the twenty-four hours. The temperature kept fairly well down below 100° F., pulse usually about 112, respirations between 30 and 40. He remained, however, very restless, especially at night, and was much troubled with cough. On 23rd July it was noted that there was still a great deal of fœtid discharge, and that distinct fluctuation was felt in the skin about 2 in. below the incision wound. On pressure there also, pus could be made to ooze out from the subcutaneous tissue at the incision wound, and it was evident that fœtid pus from this extra pleural collection was finding its way into the pleural cavity at this wound under the dressings. This area was therefore incised, and about 5 oz. of fœtid pus, along with a large circular black slough 2 in. in diameter and ½ in. thick came

away. On 24th July, with the fœtid discharge still continuing in great amount, a boggy swelling of the subcutaneous tissue was felt still lower down in the side. This was also incised and drained, fœtid pus and black sloughs being evacuated.

After this, distinct improvement in his condition was manifest. The side had still to be dressed three and four times a day, pus and sloughs were coming away, but his general condition showed marked improvement. On 6th August the chest was still requiring dressing three times daily, but the fœtor was much less, the sloughs had ceased, the temperature was rather sub-normal, and the pulse had fallen to 80 per minute. On 12th August he was allowed to sit up for a short time in the afternoon, and he was wheeled out into the balcony. On 30th August it was noted that the wounds were healing rapidly, and the pleural tube was shortened. By 15th September the wound had completely healed, and on the 19th he went to the Convalescent Home.

In this case there was at no time fœtid spit, and the sloughs which came away were undoubtedly not from the lung, but from the costal tissues. Considering the very corroding character of the pus, however, it is possible that at some superficial part of his pleural or lung tissue there may have been slight necrotic change.

In the following case the fœtid pus had burst through the lung before the operation of paracentesis was performed:— A. K., æt. 31, a joiner, was admitted to Ward 23 on 3rd April 1891, with symptoms and signs of pleuritic effusion. His family and personal history were good, his surroundings at home and at work were quite equal to the average. He had been a sergeant in the artillery, and had served for ten years, and he had always been fairly temperate. His height was 5 ft. 9½ in., and his ordinary weight was about 12 st.

Present illness dated from about three months before his

admission, when he stated that after having noticed himself somewhat out of sorts for a week, he was, after some exposure to cold at work, seized with pain in the back, which was very severe and continued for days. This gradually mended, and after some three weeks in bed he began to move about. Cough then began to affect him; but in spite of this, and of the weakness and breathlessness which he was experiencing, he continued to go about and do light work. Gradually, however, his symptoms became aggravated, and on 28th March he had again to take to his bed. He was then advised to come to hospital, and he was admitted on 3rd April.

On admission, although his general condition was fairly good, he was evidently pulled down. His weight was 10 st. 6 lb.; respiration 25; temperature varied between 97° and 100·5° F.; pulse 75, regular, rather soft. He had a cough, usually slight and dry; but he had no chest pain unless the cough became troublesome.

Physical examination revealed left-sided pleuritic effusion, and it was noticed that the area of dulness and of absent fremitus, respiratory murmur, and vocal resonance was situated more posteriorly than in ordinary pleuritic effusions. The "curved line" was distinct, but its highest point was about the spine of the scapula, and percussion from above downwards in the mid-axillary line revealed no dulness whatever, *i.e.*, the anterior limit of the dull area was about the posterior axillary line.

The heart was in its normal position, and the tympanitic note of the stomach in front could be detected a little higher than usual. Tongue was clean, the patient enjoyed very good appetite and digestion, and the bowels were regular.

In this case, owing to the general mildness of the symptoms, I diagnosed pleurisy with sero-fibrinous effusion. The facts, however, (1) of the initial pain having been very

severe, and (2) of the position of the dull area being situated more posteriorly than usual in such effusions, were noted; but it was believed that the great pain had been due to some muscular rheumatism or to some associated nephritis, of which there had at first seemed to be some indications, and that the position of the dull area might be explained on the supposition that during the effusion stage he had been lying on his back.

I therefore ordered rest in bed, good diet, and iron and quinine internally, and waited a few days to see if then any indications of absorption would show themselves. On 9th April I examined his chest again, and finding absolutely no change in this respect, I proceeded to aspirate, intending by removing a few ounces of fluid to favour further absorption. To my surprise, however, I found brownish-yellow purulent effusion, with a horribly fœtid odour. The aspiration was at once stopped, and I arranged with Mr Caird to have the operation of free incision and drainage performed next day. Owing mainly to the objection of the patient, the operation was postponed, and on the afternoon of 13th April he suddenly coughed up a large quantity of frothy, purulent expectoration, having a somewhat fœtid odour, and showing under the microscope streptococci, diplococci, and some bacilli. This expectoration continued to come up in large quantity all that day and the following night. Next day the operation was performed, the incision being made in the scapular line, and a small portion of the seventh rib being resected. After a large quantity of extremely fœtid brownish-coloured fluid had welled out, a drainage-tube was inserted, and the wound dressed as usual with salicylic wool. The subsequent course was very satisfactory. The expectoration ceased within a few days of the operation, the discharge gradually diminished and lost its fœtor, and the lung rapidly expanded.

This case is interesting also on account of the general

mildness of its symptoms, but in this respect it resembles a great many others which I have met with.

When fœtid empyema occurs, as it often does, in the case of a lung extensively affected by pre-existing disease, its recognition and determination may not be so easy. The following case may illustrate this point:—

John M., æt. 36, a farm-labourer, was admitted to the Infirmary 17th June 1899, complaining of a pain over the right breast. His family and social history were quite good, but there was evidence that he had been suffering from chronic phthisis for a great many years. He told us that since he had had measles as a child, he had always suffered from a slight cough. Twelve years ago he had had an attack of pleurisy on the right side. He recovered from this fairly well, but a year after he had had a hæmoptysis. Since then his cough and expectoration had been rather more marked, and in his spit tubercle bacilli had occasionally been found. He had, however, always been able to do some work.

His present illness began on 14th June. He began to suffer suddenly from severe pain in the right mammary region, which completely prevented him from working during the day and from sleeping at night. The doctor who was called in blistered him, but as he was not improving, he came into hospital.

On admission, he was somewhat feverish, his temperature rising to 101·5° F, his pulse was 96, and respirations 28 per minute. His right upper lobe showed anteriorly and specially posteriorly evidence of tubercular disease, and at the base there was in this lung distinct evidence of thickened pleura. His other organs showed nothing abnormal.

On the evening of his admission he had an attack of hæmoptysis. It was not copious, but lasted till next day, when suddenly he coughed up about half a spittoonful of fœtid muco-pus. Physical examination then revealed no

change in the percussion dulness at the upper or lower part of the right lung, but now coarse crepitations and rhonchi were heard all over the right side, and some rhonchi were also detected over the left. As the days passed, the amount of fœtid expectoration gradually lessened, but it continued to come up irregularly—some days little or none, other days a fair quantity. Repeated explorations were made over the upper and lower lobes anteriorly and posteriorly, but although a little pus was occasionally obtained, no distinct loculus was struck. Gradually the condition improved, and it was interesting to find that with the disappearance of the fœtid pus from the spit, tubercle bacilli were again recognisable. He was discharged, recovered, on 4th August.

In connection with this man's case, it is to be remembered that with his maimed lungs and excavation, fœtid spit might be caused by a number of conditions, *e.g.*, decomposition of retained secretion, gangrenous changes in cavity walls, or fœtid abscess. In phthisical cases, one occasionally sees this occurring, and one sometimes notes that with such an occurrence the phthisical process shows some arrest, just as if the fœtid process had some destructive effect on the tubercular. In this patient's case, the condition was, I believe, due to a localised fœtid empyema, possibly interlobular, and although any destructive effect of the fœtid pus on the tubercle bacilli must have been very transitory, the favourable course of the malady in his case was at the time a subject of remark.

So much for a general description of fœtid pleurisies, either simply pleuritic or associated with lung gangrene. Their etiology, pathology, and symptomatology now require some consideration.

Etiology.—Here, it has to be remembered in the first place, that although fœtid effusion dependent on primary

gangrenous changes in the lung is comparatively rare, fœtid effusion of itself is a very common disease. Nearly 30 per cent. of my Infirmary empyemas were fœtid ones, and I have always been of opinion that a large proportion of the cases which are reported in literature as examples of lung abscess or lung gangrene, are in reality fœtid empyemas. This condition may occur in childhood as well as in adult life. When associated with lung gangrene, it is more apt, in my experience, to occur in the elderly, yet I have seen lung gangrene cases in both early and late adult life.

Fœtid empyemas are distinctly more common in males than in females; and although they may affect the apparently robust, they are more frequent in those who are constitutionally weak, or who have been weakened by previous disease. In this connection alcoholism, and amongst diseases, influenza, play important parts. In a large proportion of my cases there had been a history of previous lung or pleural disease, or a history from which the existence of tubercular lung disease might be suspected. As already stated, they are frequent in the pleurisies following on sub-diaphragmatic inflammations, *e.g.*, appendicitis, and for their occurrence also lung embolism can sometimes be blamed.

The *pathology* of fœtid or gangrenous effusions is not well known; but as a clinician the view which I have always entertained as a pathological working hypothesis is that a fœtid effusion, whether associated with lung gangrene or not, is not a pathological entity, but is, like an ordinary pleural effusion, or an ordinary pneumonia, dependent for its existence on the fact that the lung or pleural tissue has had its nutritive power so lowered that it has had to suffer from the activities of organisms or toxines which actually or potentially are always present within it. It will perhaps better illustrate my meaning if I say that in a patient suffering from fœtid or gangrenous empyema, the nutritive power of the lung and pleural tissue has been temporarily in

a condition analogous to that in which the lung and other tissues are apt to be continuously, in the person of a confirmed diabetic.

In the case of an ordinary pneumonia, or an ordinary pleurisy, we can suppose that this contest between soil and germ calls forth a reaction power in the tissues, and probably in the whole organism as well, the results of which are usually to overcome the contesting organisms before they have been able to cause any real loss of tissue. In the case of a fœtid or gangrenous empyema, or lung, on the other hand, the results of this contest on the tissues are much more serious. Not only may the tissues be absolutely unsuccessful in the contest, but if they do succeed, it is only after the parts specially involved have suffered to a greater or less extent a real loss of destruction. Moreover, why one individual should be the subject of a fœtid empyema alone, and another the subject of a fœtid empyema with lung gangrene, seems to be determined by the same constitutional differences which, with two individuals exposed to the same debilitating cause, determine in one an ordinary pneumonia, and in the other an ordinary sero-fibrinous or purulent pleurisy.

I am well aware that the frequent association of fœtid empyema with the fœtid sub-diaphragmatic inflammations, or with embolic processes, must be regarded as pointing directly to special infective agencies. I would, however, with this call to mind that with fœtid sub-diaphragmatic inflammations we frequently get pleurisies which are not only non-fœtid but not even purulent, and from this can be argued the real importance of the nutritive condition of the pleural or lung tissue. Moreover, most physicians must have seen pulmonary embolisms set up pleurisies which, judging from their source, would justifiably be expected to be purulent or fœtid, and yet which proved to be simply sero-fibrinous.

As might be expected, the intensity of the fœtid or gangrenous character varies very greatly in different cases. In some instances it is only slightly marked and very temporary, in others it is extremely strong and persistent. In most instances this intensity tends to lessen with free drainage; but whilst in the milder cases the fœtor and any infecting and corroding influence of the discharge on the wound and surrounding tissues ceases almost at once, in others it is present and persistent for long periods. In the cases of lung gangrene, the fœtor persists till the sloughed portion separates.

Lastly, as might also be expected, the organisms found in fœtid empyemas are varied. Diplococci, streptococci, staphylococci, bacilli like the influenzal bacillus, and the bacillus coli, bacteria, living or dead, and gas generating organisms may be present.

Symptomatology.—Fœtid empyema is usually ushered in by the specially severe symptoms which are associated with a purulent, as compared with a sero-fibrinous pleurisy. The fever is often high, shiverings and rigors are present, and the chest pain is apt to be extremely great. On the other hand, as has been exemplified in some of the cases of fœtid empyema quoted, the initial fever may not be so great, so that unless perhaps for the pain, an ordinary sero-fibrinous pleurisy might be all that was anticipated. As in an ordinary empyema, the cough may be very troublesome. The character of the spit and odour of the breath require special attention.

It is to be remembered that a lung gangrene may possibly pass on to a fatal termination without any fœtor of spit or of breath showing itself at all. We explain this on the view that the sphacelated area of lung tissue is, as it were, shut off from the patent air-cells and bronchial tubes around. Similarly a fœtid empyema may run its course for long without causing fœtid spit or breath. The tendency,

however, in both cases is for these characters of spit and breath to present themselves sooner or later; and in the fœtid empyema cases the tendency to sudden and copious bursts is characteristic, so that the patient, often quite unexpectedly, gets quit by expectoration of a large quantity of the fœtid fluid all at once. In some of my cases before the fœtid collection or the gangrenous condition was discovered, their presence was indicated by the patient every now and then being noticed to "gulp" up a very fœtid breath.

As has been already stated, fœtid empyema, if left alone, shows a special tendency to find a way out for itself through lung and bronchi, and thus to produce more or less permanent lung and bronchial trouble. In this way, very special importance attaches to its early recognition and removal.

It must therefore always be borne in mind, that such empyemas may occur with symptoms so slight that their presence may be unsuspected till a sudden fœtid expectoration occurs. It should also be borne in mind, that following on pneumonic or pleuritic conditions they may be very small, and they may be localised at varying parts of the chest. In this way, their detection by physical signs and even by the exploring needle may be extremely difficult. It must also be evident that if they have been allowed to evacuate themselves through the lungs and bronchi, the difficulty of localising them will be increased enormously. More than once it has happened to me to locate a small fœtid effusion by the exploring needle, which a few hours afterwards found its way out through lung and bronchi, so that when chest incision was performed next day, with the puncture mark as a guide to the spot, the effusion could not be found. This will, however, be referred to again under treatment.

CHAPTER XI

PULSATING EMPYEMAS

Of pulsating empyema, two forms are recognised—(1) pulsating intra-pleural empyema, and (2) pulsating empyema of necessity. In the former, all the fluid is in the pleural cavity; in the latter, a certain amount of it has found its way to the outside of the pleural wall, and forms under the skin a pulsating tumour. In both cases the left has been the side almost invariably affected.

Pulsating Intra-pleural Empyema.—As regards this, it is to be observed, that all the pulsating intra-pleural effusions which have been reported have not been purulent. Several instances of pulsating sero-fibrinous collections have been met with, and interesting examples have been given of such by Douglas Powell and Comby. But the great majority of these cases have either been purulent from the first, or have become so in the course of the disease, so that we have some justification in speaking of them as pulsating empyemas.

Some of the earliest recorded cases are those of Macdonell[1] and Stokes.[2] Stokes' case is specially interesting. He describes how, in a large empyema, which had powerfully pushed the heart to the right, he had observed pulsation of the entire sac. "Thoracocentesis was performed three times, and each time a large quantity of more or less purulent fluid was removed. Before each operation the beats of the heart

[1] *Dublin Journal of Medical Science*, March 1844.

[2] *Diseases of the Heart and Aorta*, Dublin, 1854.

produced a diastolic (?) pulsation of a most strange character, which made itself felt in the left lateral portion of the chest, and through the entire thorax. The bed was shaken by each contraction of the heart, the force of which seemed to be increased, the violence and extent of the pulsations were such that the sleep of the patient was interrupted. The fluid had never any tendency to find its way outside, and it is very remarkable that the heart never left its position to the right of the sternum after any of the emptyings of the chest."

Other more or less similar cases have been reported, a good collection of which is given in Comby's work *L'Empyème Pulsatile*.[1]

Pulsating Empyema of Necessity.—In this form, the fluid has found its way through the chest parietes, producing at some part outside of the thoracic cavity a subcutaneous tumour, pulsating and expansile. Like the former variety, it is almost invariably on the left side of the chest, and the fluid is usually at its lower part. Unlike the former variety, however, the fluid is always purulent. This can easily be understood when we remember that only a purulent fluid can penetrate an intercostal space. Further, the pleuritic process which has given rise to this effusion is usually a chronic one. This we can understand when we remember that time is required for the process of perforation. The position of the pulsating tumour is usually about the precordial region—that is to say, on the left side,[2] somewhere between the mid-sternal and nipple lines. But just as an empyema may perforate the chest-wall and point elsewhere, so the pulsating tumour may be found elsewhere—*e.g.*, in the posterior or lateral part of the chest, or even in the lumbar region.

[1] *L'Empyème Pulsatile*, Paris, 1895.

[2] Dr Colin Macdowell, *Brit. Med. Journ.*, 1906, vol. ii., p. 1035, quotes a case of double empyema with the pulsating one on the right side.

These tumours vary in size; they are usually somewhere about 4 in. in diameter, and the expansile character of the pulsation can easily be recognised. They are readily influenced by the respiratory act—*e.g.*, whilst inspiration is apt to cause a slight diminution, expiration may cause a corresponding increase in their bulk. The acts of coughing and sneezing are specially apt to increase their size, and a change may also be produced by the patient's sitting up or lying down. The tumour may to some extent be reducible, and cases have been reported where partial reduction by pressure and manipulation caused marked increase in the dyspnœa. It is evident that to permit of the occurrence of these variations in the size of the tumour, the opening of communication between it and the intra-pleural collection must be free. In the example of pulsating empyema which I shall shortly describe, this opening readily admitted the finger.

But now, how is the occurrence of pulsating empyema, either with or without a communicating extra-pleural tumour, to be explained? It is evident, in the first place, that the condition or conditions which give rise to this must be rare. Cases of large pleuritic effusion, and cases of *empyema necessitatis* are not uncommon, and yet this pulsating character is very rarely met with. What, then, is it which causes the pulsation?

On this subject a great many theories have been brought forward, to some of which we may refer.

Let me first quote the following from Guéneau du Mussey: "Quand avec l'épanchement se trouve, dans le cavité thoracique, un gaz éminemment compressible ou une portion considérable du poumon encore perméable à l'air, on comprend que la systole ventriculaire puisse refouler le liquid contigu au péricarde, et lui communiquer un ébranlement ondulatoire qu'elle ne peut pas produire quand elle agit sur une masse absolument incompressible."

Féréol seems to have practically adopted this, and bases his explanation of the occurrence of pulsation upon the existence of an associated pneumothorax. But cases have very frequently occurred where there was no pneumothorax.

Traube seems to have believed that the pulsation depended in some way on pericarditis and pericardial effusion, but cases have occurred without them. Walshe[1] says of the pulsation, that it "seems to be merely an excess of the slight fluctuation movement in the fluid which is not very uncommon in ordinary cases." Powell[2] concludes that "probably nothing more is needed than an amount of fluid

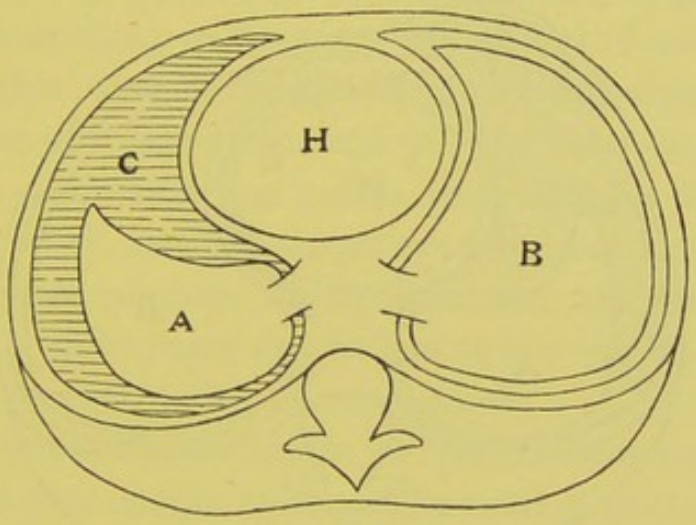


Diagram XVIII.

which shall exercise a certain degree of pressure, neither too much nor too little, upon the beating heart." Many other explanations have been proposed, for details of which I would refer to the works of Bouveret[3] and Comby; but, as it seems to me that none of them are quite satisfactory, and that a study of the physical conditions existing in the thorax may help us in this matter, I shall briefly proceed to this.

Let Diagram XVIII. represent a section through the thorax at about the level of the sixth dorsal vertebra in a

[1] *Diseases of the Lungs*, London, 1871.
[2] *Diseases of the Lungs and Pleuræ*, London, 1886.
[3] *L'Empyème*, Paris, 1888.

patient with left pleuritic effusion; H is the heart, A the collapsed left lung, B the healthy right lung, and C the effusion. It is evident that as the effusion is, like all fluids, practically incompressible, and as when the lung is collapsed it is contained in a cavity, the walls of which are practically rigid and unyielding, the systolic and diastolic movements of the heart will be completely resisted on this side. On the other hand, they will readily be allowed to take place on the right side, the healthy and elastic air-containing right lung readily yielding to the enlargement of the heart in diastole, and as readily following the diminution in its bulk in systole.

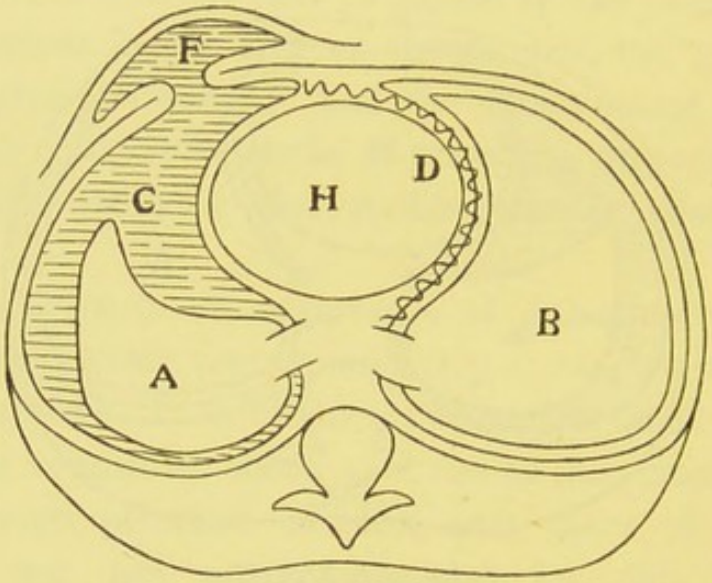


Diagram XIX.

But now see Diagram XIX. Supposing that along the zig-zag line D the heart is firmly fixed by adhesions — sternal, pleuritic, pericardial or mediastinal—and supposing that the fluid C in the left pleural cavity is in free communication with the subcutaneous abscess cavity F, it is evident that the mechanical conditions will be quite reversed.

Increase or diminution in the size of the heart in diastole or systole will readily affect the fluid effusion. This it can because—(1) by the firm resistance on the right half of the pericardium the systolic and diastolic movements of the heart will be the more marked in the left; and (2) the fluid in the

left pleural cavity will readily yield and recoil with the cardiac movements, being now in free communication with the fluid in the subcutaneous cavity, the walls of which are distensile and elastic. Hence we conclude that in pulsating empyema of necessity, the factors which bring about the pulsations are fixation of the heart on the opposite side, and distensibility and elasticity of the walls of the empyema sac.

I shall now describe fully one of the few cases of this condition which I have seen.

Thomas R., æt. 26, a brewer's clerk, was admitted to the Infirmary on 15th January 1895, complaining of pain over the heart, with swelling in the same region, and of cough.

His father died at the age of 32 of heart disease, he had one brother who died at the age of 18 of heart disease, and a sister who is delicate and suffers from cough. His mother, another brother, and two sisters are alive and healthy.

As regards previous illnesses, he had had measles when a child, and eight years ago he had to give up his occupation as a pupil teacher, owing to nerve strain.

His present illness seems to have commenced in July 1894. He then caught cold, and he noticed that on taking a deep breath, or on sneezing, he felt a pain over the region of his heart. He still, however, continued at his work, and he expressly states that he had no cough and no night sweats. About the 5th or 6th January, however, he seems to have again caught cold. A cough commenced, which has continued, and which has been accompanied by severe pain over the heart. A few days after he caught this cold, he noticed a swelling over his heart. This, and the cough and weakness, with shortness of breath on exertion, led him to consult a doctor, who recommended him to come to the Infirmary.

State on Admission.—He is about 5 ft. 6 in. in height, and weighs 110½ lb. He says he has been losing flesh

lately. His temperature varies between 97° and 99°; pulse is usually about 80; respirations about 24 per minute.

On examination, over the præcordia a pulsating swelling is seen. It is somewhat diffuse, and in extent is about 4 in. in diameter, its centre being between the fourth and fifth ribs. On palpation its expansile character is well recognised. It can be somewhat reduced by steady pressure, and when the patient coughs, it can be felt markedly to increase in size. There is pain when its centre, between the fourth and fifth ribs, is pressed upon. Over the swelling, and over the lower half of the sternum, there is some slight œdema.

Percussion gives complete dulness over the whole of this swollen area, and over the entire upper lobe of the left lung in front there is a markedly impaired note. The note is, however, resonant over the lower and lateral portion of this lung. Posteriorly, there is impairment on percussion from the apex down to the angle of the left scapula. Over those parts of the lung which are dull to percussion, auscultation reveals weak bronchial breathing. At the apex on the left side anteriorly and posteriorly, a few crepitations can be heard. Over the lower part of the left lung, and over the whole of the right lung, the breath sounds are vesicular in character. Over the right lung in addition they are exaggerated. Vocal fremitus is absent over the swelling in the præcordia and the neighbouring portions of the left upper lobe. Over the rest of the left lung it is present.

The precise position of the heart cannot be made out exactly by percussion, but it seems to be displaced to some extent to the right. Except that the first sound is slightly reduplicated, its sounds are normal. The other systems and organs are healthy.

In making our diagnosis in this case, several possibilities entered our minds. There was, in the first place, the possibility of aneurism, which indeed we were informed had been suspected prior to his admission. But we remembered

that our patient gave a history of lung trouble, that the pulsating tumour had shown itself suddenly, and had rapidly reached its present stage. We noted further, that the pulsating swelling was not in the position usual in cases of aneurism, that there was no evidence of cardiac or vascular disease, and that there were none of the pressure symptoms of aneurism. We then made an exploratory puncture with a hypodermic syringe, and we readily ascertained the presence of pus. Putting aside the idea of suppuration from a diseased rib, or from mediastinal abscess, we were thus led to the diagnosis of pulsating empyema, and we noted that in some ways symptoms and physical signs noted, and in other ways seemed to contra-indicate this. As regards symptoms, there was practically no fever, hectic, nor sweating, nor was there any marked cough such as one expects to find in empyema. But, on the other hand, we had to remember that in empyema these symptoms are occasionally absent, and that in pulsating empyema all of them are apt to be specially latent. Then as regards physical signs, we had to note that the empyema in this case was limited to the upper part of the lung. But this, although unusual, may yet, as we have seen, occur.

In this way, taking into consideration the history of lung trouble of at least several months' duration, and the fact that there was evidently apical disease as betokened by the crepitation and other physical signs, we came to the conclusions—(1) that our patient had been suffering for months from tubercular disease of the left apex; (2) that this had resulted in pleuritic adhesions over various parts of the left upper lobe; (3) that over a portion of this lobe anteriorly, where adhesions had not formed, a purulent pleurisy had started; (4) that the pus from this, prevented from gravitating to the lower part of the pleural cavity by adhesions, had collected in the upper part and had found its way outward between the fourth and fifth ribs, and formed the pulsating swelling over the præcordia.

On 19th January, the patient having been chloroformed, an incision was made over and into the swelling. The abscess cavity having been opened and emptied, the finger introduced through the incision wound was easily passed through the opening of communication between it and the empyema cavity. This empyema cavity contained some 14 oz. of pus, and as had been diagnosed, was found limited to the upper part of the chest. In order to facilitate drainage, another opening was made into it between the fourth and fifth ribs, in the anterior axillary line, a small portion of rib resected, and an ordinary drainage-tube inserted. After the operation the patient experienced very considerable relief. The left lung began to expand, but it was noticed that the heart showed little or no tendency to return to its normal position. After a few weeks the patient began to show more distinctly, however, the symptoms of phthisis. He developed a cough and expectoration which showed the tubercle bacilli, and his temperature chart indicated fever. He lingered on till 27th March. At the post-mortem examination, with extensive tubercular mischief in the left and also in the right lung, there was found great fibrous thickening along the left border of the sternum, completely fixing the pericardial sac.

In this case it is evident that the factors which brought about the pulsation were the fixation of the heart on its right side, and the distensibility or elasticity of the walls of the conjoined empyema sac and abscess cavity.

But it will now be asked, how is the existence of pulsation in intra-pleural empyemas, or in other intra-pleural collections, to be explained? To this I would answer that the factors are practically the same. The heart must be more or less fixed by the pericardium, whilst the yielding and recoiling of the fluid to its movements may be due to such conditions as the presence of air in the pleural cavity,

to the lung on the affected side being semi-inflated, to a yielding and elastic diaphragm, perhaps to the yielding of the interspaces through paralysis of the intercostal muscles, or perhaps even to the comparatively yielding chest parietes of early life. A good illustration of how pulsation may be brought about by yielding parietes can be obtained from a study of the cranial circulation. Thus, if in the cranium of a living animal a trephine opening be made, through this opening the exposed brain can readily be seen to pulsate. If now this opening be completely closed by a piece of glass, it will be at once seen that the pulsations cease. Hence we may say that whatever permits of the yielding of walls tends to bring about pulsation.

It is evident that the degree of fixation of the heart and the degree of yielding and recoil which the chest parietes or contents permit to the fluid, must vary much in different cases. Thus the strength and extent of the pulsations in pulsating empyemas must vary correspondingly. In Stokes' case, previously referred to, we are told that the strength of the pulsations was so great as to shake the patient's bed. This statement has been regarded by some as incredible, but we have little hesitation in believing it if we remember (1) the hydrostatic law; (2) the fact that the normal contractile power of the heart is somewhere about 4 lb. to the square inch; and (3) the fact that the normal distensile force in diastole, though of course less than this, is still considerable.

We can understand again in other cases how the pulsations may be so slight as to be hardly perceptible, and we cannot doubt that if carefully looked for—*e.g.*, with the manometer, pulsation in pleuritic fluids due to cardiac movements might be demonstrated in many cases in which its existence could not otherwise be recognised.

CHAPTER XII

PHTHISICAL EMPYEMAS

REFERENCE has already been made to the very close association between sero-fibrinous pleurisy and phthisis, and we have seen that such a pleurisy may readily either precede or occur as associated with phthisical lung disease. But reference has also been made to data which indicate that this relationship to phthisis does not altogether hold as regards purulent pleurisies. Whilst, for example, in my hospital cases of sero-fibrinous pleurisy an indication of pre-existing phthisis showed itself in 15 per cent., with the empyemas it showed itself only in 2 per cent. This proportion is strikingly small, possibly a larger number of cases would show it to be understated, still as a fact it is obvious enough. Furthermore, as has also been referred to, if we trace cases of sero-fibrinous pleurisy, and cases of purulent pleurisy for years afterwards, we shall find indications as time goes on that the supervention of lung phthisis is as infrequent in the latter as it is frequent in the former. All this indicates that in a broad general way empyema and sero-fibrinous pleurisy act contrariwise in their relation etiologically to tubercular lung disease.

But now phthisical empyemas do occur, and when they do, whether associated with the pneumococcus, the streptococcus, or the tubercle bacillus, they are apt to present characters somewhat similar to those which we have remarked as presented by sero-fibrinous pleurisies in the

phthisical. Thus they are apt to be rather more latent than ordinary empyemas in their mode of onset and symptoms, the initial pain and distress being frequently less severe. They are also apt to be specially prolonged in their course, re-expansion of the collapsed lung after paracentesis and drainage being difficult to obtain, just as it is apt to be in the phthisical sero-fibrinous pleurisies after tapping. It will be apparent, however, that to some extent this latency of onset and protracted course may be explained as the result of simply mechanical conditions.

For example, as regards latency of onset. It is evident that a purulent pleurisy in a phthisical individual must often mean a purulent pleurisy affecting a lung in which more or less extensive pleuritic adhesions are already present. In this way the free rubbing against each other of pulmonary and costal pleura must be to some extent limited, and consequently pain and liability to extension of the pleuritic process proportionately lessened. Thus, insidiousness and latency of onset may be explained.

Again, as regards lung re-expansion, it will be equally evident that a lung in which a certain amount of tissue has been destroyed by tubercular disease, and in which processes of cicatricial contraction are occurring, must in consequence of this be less capable of expanding with the disappearance of the pleural effusion than will a healthy lung.

But apart from these purely mechanical considerations, it must be obvious that the same lowered nutrition power which gives opportunity to the tubercle organism to assert itself must also handicap lung and pleural tissue in their reaction, and thus lead to such empyemas running a less favourable course, even when there is very little coincident tubercular lung disease.

When a patient therefore with confirmed phthisis develops an empyema, we can quite easily understand that treatment is likely to be followed by little more than temporary relief.

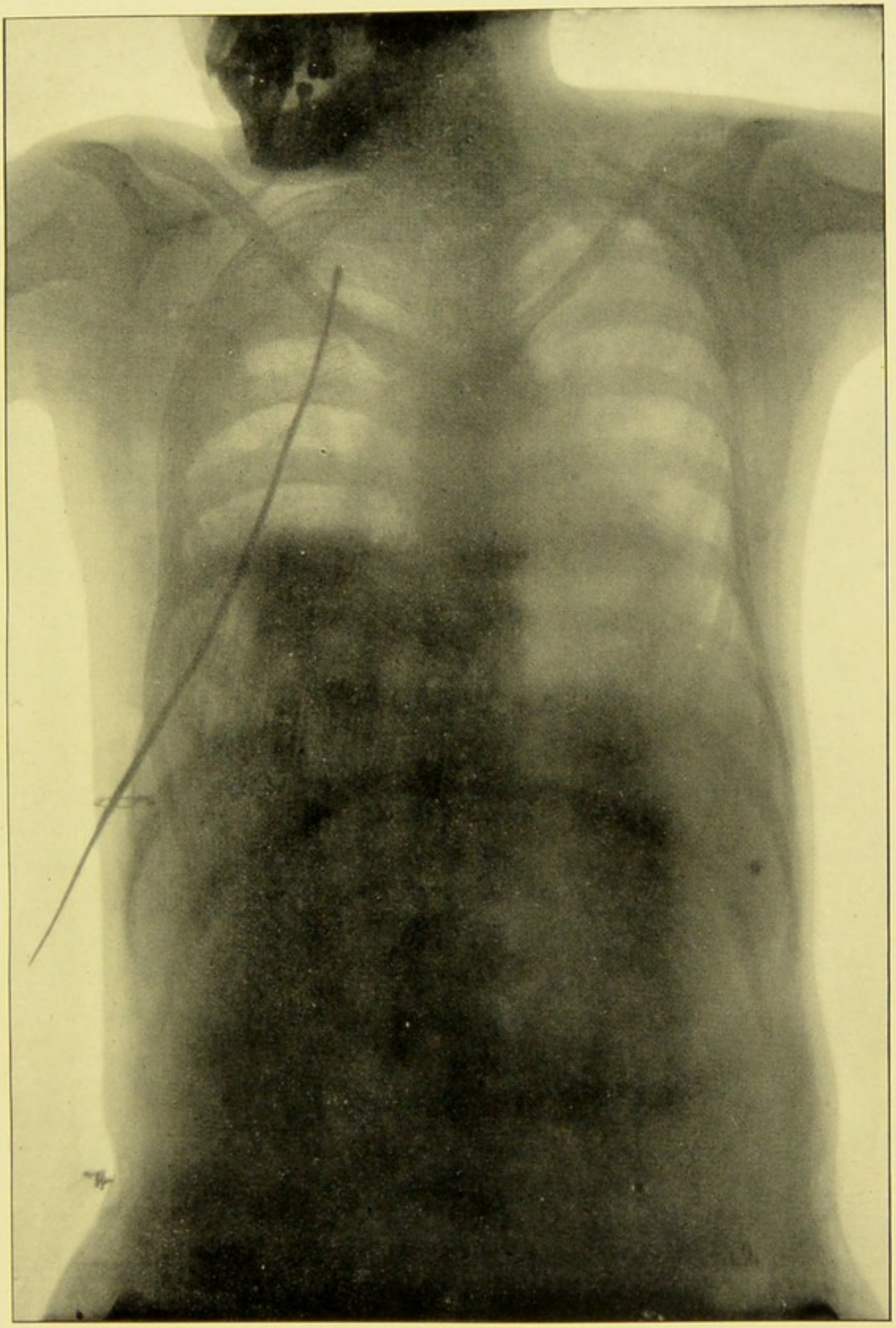
Similarly when a patient with a very delicate family history develops an empyema, even though we find little evidence of tubercular lung disease, we must always approach the treatment of the condition with great anxiety.

Still there is something to be said on the other side. It must be remembered that in an individual with a phthisical lung, the mere occurrence of the empyema may mean increased reaction on the part of the individual, and that this may in itself influence for good the tubercular process. Furthermore, in such an individual, the occurrence of an empyema may mean, mechanically, more rest to the affected lung, and more work to its unaffected neighbour. In this way, a further salutary influence on the tubercular process may be exercised. Of such an influence the following cases are fairly good examples :—

Mary B., æt. 26, a factory worker, was admitted to the Royal Infirmary 18th July 1902, suffering from pleurisy of six weeks' duration. Her family history and surroundings at home were satisfactory, but she informed us that for some months she had been suffering from a cold and cough, with a yellowish-white spit, which we ascertained had shown a few tubercle bacilli.

History of Present Illness.—Six weeks ago she noticed a pain in her side, with aggravation of her cough and gradually increasing breathlessness. Her doctor had diagnosed pleurisy with effusion, and had tapped the chest on three occasions, on the first two of which the fluid had been sero-fibrinous. Her symptoms, however, had not improved, she had been losing flesh, and so she came to the Royal Infirmary.

On admission she was distinctly feverish, her temperature showing undulations between 97° and 102° F. Her pulse was 120, and her respirations 24 per minute. Examination of the chest showed on the right side absent respiratory movement, and absolute dulness all over the back, and dulness in



MARY B., AGED 26. PHTHISIS AND EMPYEMA.

[*Face p.* 200.

front from the fourth interspace downwards. Breath sounds were practically absent, except over the apex in front, where they were feeble, and accompanied by a few crepitations. The liver was displaced downwards, almost as far as the umbilicus, the heart was displaced about an inch and a half to the left.

On the day of her admission, the right pleura was explored and pus was found. It, however, showed no organisms. On the 21st Mr Cathcart operated, resecting a piece of rib and inserting a large drainage-tube, the patient standing the operation well.

Her improvement was slow after the operation, but wonderfully complete. Her temperature for long continued to show marked oscillations, up to 100° or 101°. The pulse also continued rather rapid, 90 to 100. Some cough, with expectoration, showing however no bacilli, continued, and the wound continued to discharge, the amount of this gradually lessening. With this, improvement in appetite, digestion, and increase in weight were associated, so that on 30th October following, although there was still such discharge from the wound that daily dressing was required, she was sent to the Convalescent Hospital. She remained there for six weeks, and then went home, daily dressing of the wound being still required. Fortunately, her home surroundings enabled this to be done well, and she gradually improved in condition. On 17th July 1903 she was again admitted for examination, and for whatever further treatment might be deemed necessary. At that time, with slight impairment in the percussion note all over the affected side, faint breath sounds could now be heard; whilst a few crepitations, with somewhat bronchial breathing, were audible over the upper lobe in front. The wound was still open, and through the opening a long gun-shot probe could be passed upwards for 10 in. The X-ray photograph was taken with the probe in this position.

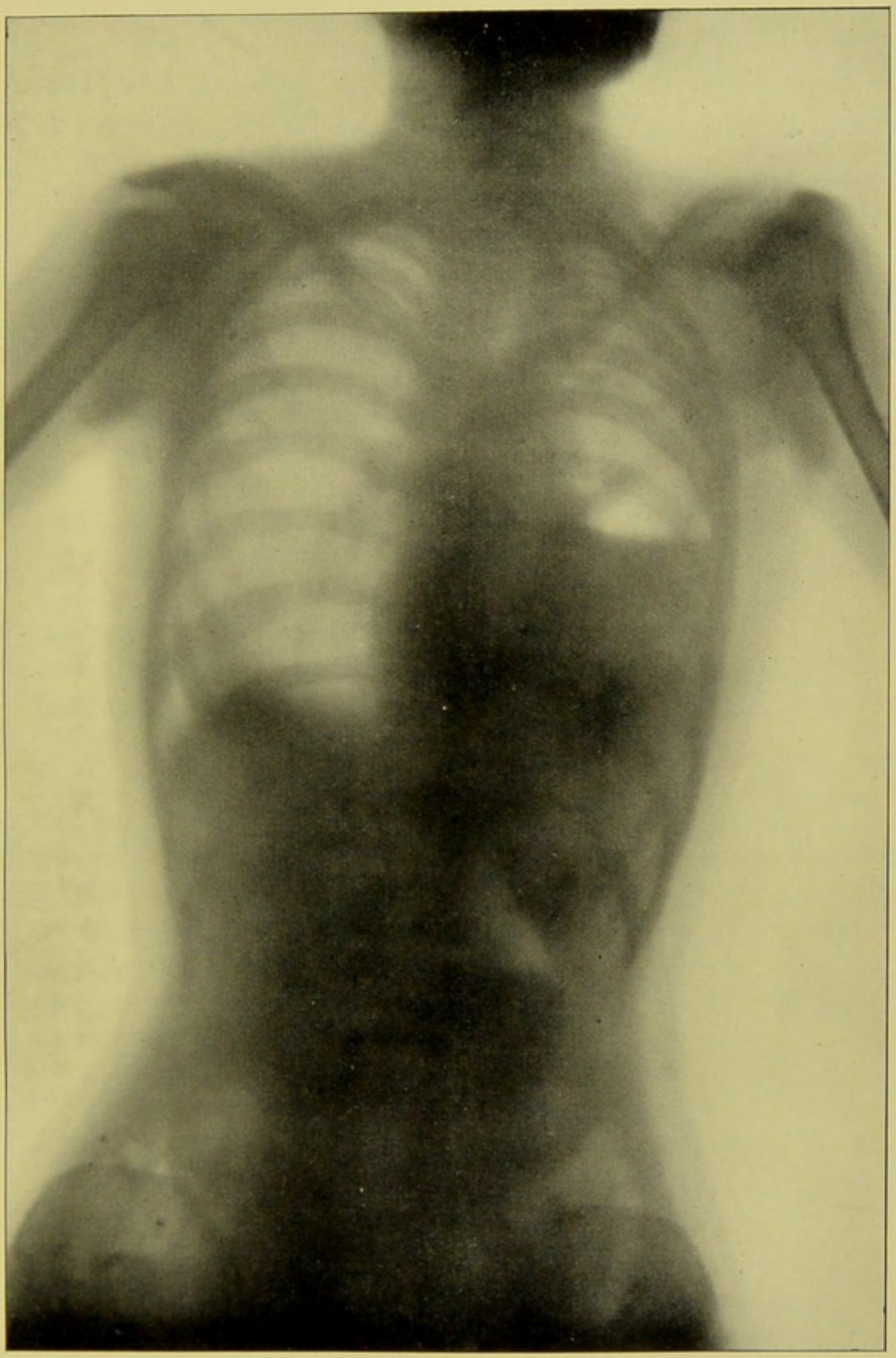
The sinus opening was washed out daily with peroxide of

hydrogen, and after some weeks' residence in the ward she was sent home, with directions to come up to the ward at short intervals for examination and dressing. Gradually the long sinous-like wound closed from above, the tube being correspondingly shortened, and by the summer of 1904 the wound had completely healed. Her subsequent progress has been very satisfactory: she has grown fat and healthy-looking, and though distinct dulness at the right apex has persisted, she has little or no cough, and she feels practically well.

In this case we believe that the amount of apical mischief was relatively slight. It had been great enough to cause shrinking of the whole upper lobe, when the lower part of the lung was collapsed by the empyema, but yet not great enough to prevent a re-expansion of the lung sufficient to permit approximation of pulmonary to costal pleura, without further chest resection.

A consideration of these phthisical empyemas indicates to us that they precisely are the varieties of empyema for which extensive resection operations are most emphatically appropriate, if only the patient's strength will permit. This is because in them efforts to drag the lung outwards are not only very likely to prove futile, but may readily be productive of serious harm. This will be referred to in discussing the treatment of empyema. Meanwhile I quote another case of phthisical empyema, in which the result was also wonderfully satisfactory, although the patient was left with a pyo-pneumothorax:—

Caroline G., æt. 29, a shop-girl, was admitted into the Edinburgh City Hospital, 20th March 1908, suffering from phthisis of the left upper lobe, of some two years' duration. Her disease had shown itself after an exposure, and it was running a chronic course with very little fever, slight cough and spit, and with at times very slight streaky hæmoptysis. She kept fairly well till the beginning of June 1908, when for



CAROLINE G., AGED 29. PHTHISIS AND PYO-PNEUMOTHORAX.

[*Face p.* 202.

about a week she suffered from an attack of left-sided pleurisy. The attack was a mild one, the temperature rising at night to about 100°, and it passed off, leaving no effusion. She then continued fairly well till the following August and September, when she had two fresh attacks of left-sided pleurisy. These were distinctly more severe than the former, the temperature in the September attack rising one evening to 104·5°. They left her with her chest about half full of fluid, but the temperature fell, and by October the fever had settled down. She then kept fairly well till January, when some fever and chest pain recurred, and it was evident that the fluid had begun to increase. Dyspnœa became manifest, and physical examination revealed a large quantity of fluid on the left side.

On 14th January 1909 it was noted that the left chest showed great flattening at the apex, with almost complete immobility on respiration. It was dull from apex to base, and except at the very apex, the breath sounds were absent. The heart was displaced to the right. An exploratory puncture showed that the fluid was distinctly purulent, and the blood examination showed a leucocytosis of 15,000. Drainage rather than aspiration was of course considered advisable, and this was done on 19th January 1909. A large trocar and canula were used, and an india-rubber drainage-tube was introduced through the canula and left in, 2 pints of sero-purulent fluid being thus removed. In about a week all drainage had ceased, the temperature had fallen to normal, all pain had gone, and she was feeling quite relieved as regards breathing. The wound was allowed to close, and the patient rapidly gained strength; but physical examination revealed the existence of a pyo-pneumothorax. This has also been demonstrated by an X-ray photograph.

Although still troubled by some cough and dyspnœa on exertion, she eats well and keeps well, so that I consider further interference inadvisable.

CHAPTER XIII

STREPTOTHRIX AND LEPTOTHRIX EMPYEMAS

PERSONALLY I have had very little experience of disease associated with actinomyces, and the notes of my hospital cases of pleurisy and empyema show none in which these organisms were found. It must always be remembered, however, that they do occur, and that a considerable number of instances of pulmonary actinomycosis appear in literature.

From the clinical point of view, the examples of such published many years ago by Sir Douglas Powell,[1] and by Dr Gamgee[2] are certainly worthy of reference, and all the more so since, in Powell's case, the condition, although it simulated empyema, could hardly be said to be so.

In Powell's case, the disease occurred in a boy aged 9 years, who was the son of a dairyman, and in whose family there was a distinct tubercular history. He was brought to the Brompton Hospital on 8th October 1888, having then been distinctly ill for four months. He presented fever, hectic, emaciation, and cough, and there were over his right chest a number of small swellings, one of which, by its appearance, suggested the early stage of a pointing empyema. On physical examination, his chest presented all the signs of effusion over its lower two-thirds. Empyema was suspected

[1] *Med. Chir. Trans.*, London, vol. lxxii., p. 175.
[2] *Brit. Med. Journ.*, 25th May 1899.

but chest exploration by the needle was negative. Later on, a more complete exploration of one of the swellings and of the pleural cavity was made, and soft pultaceous masses, showing the actinomyces, were found in both. The boy died on 26th February 1889, and the following was found on post-mortem examination :—

"*Thorax.*—There was a large mass of soft material occupying the pleural cavity of the right side from about the level of the second rib downwards. It was homogeneous, appearing to consist of the same material in different stages of degeneration. Very soft, pultaceous, and canary coloured, much resembling coarse, badly made mortar. There was also a large amount of this material between the base of the lung and the liver, the diaphragm appearing to have been incorporated in it. There were some recent adhesions in the upper part of the pleura.

"*Lungs.*—Right: the lower anterior part of the lower lobe and the middle lobe converted into tough fibrous material, which has an irregular interlobular distribution, the bands enclosing small pinkish areas like altered lung tissue. Here and there in the midst of the fibrous growth there are irregular, canary-coloured masses just like that described above. The upper lobe is plentifully studded with grey nodules the size of a hemp seed, some slightly pigmented, and exactly resembling tubercular nodules. Left: aerated throughout, but contains scattered nodules of the size of a pea to a small Spanish nut, which, on section, have the characteristic canary-coloured appearance.

"Between the liver and the diaphragm there was a collection of pultaceous matter similar to that described above, but the liver itself appeared healthy. On section, however, there was seen an abscess about 1 in. below the upper surface, of the size of a small orange. Its wall was about a quarter of an inch thick, and consisted of fibrous

tissue. The contents consisted of tenacious yellow pus, which had a trabeculated appearance.

"The right lateral sides of the dorsal vertebræ were covered over by the growth and superficially eroded.

"Some of the ribs also between which the growth had passed to get to the outside of the chest were eroded.

"Nothing worthy of note was found in any other of the organs."

Dr Gamgee's case was that of a man, æt. 65, who had been suffering from an abscess of his abdominal wall—the nature of which had been obscure—for many years. When admitted to hospital, he had developed symptoms and signs of pleurisy with effusion in his left chest.

After his admission he developed irritative head symptoms, with convulsions, and died suddenly.

On post-mortem examination, with thickening of the pleuræ in both chests, 3 pints of thin purulent fluid were found in the left pleura. A large actinomycotic abscess was found in the liver, with similar slighter developments in the brain and kidney.

Of purulent effusions, associated with the presence of leptothrix filaments, I have seen very few examples. One of these occurred in a woman of 45, who had an empyema of the right side. In her case, the disease had begun very acutely, but the effusion at first had been sero-fibrinous. After a single tapping it had become purulent, and soon thereafter it burst into the bronchi, so that a copious purulent spit developed. As a rule, this was not fœtid, but now and again distinct fœtor of it and of breath were noticed. The chest was afterwards opened and drained, but the interesting point is that on several occasions, along with diplococci, leptothrix filaments were seen on examination of the pus.

The following is, however, the most interesting example of this condition which I have seen. It occurred in my ward

in the Royal Infirmary, and was admirably worked up and published by my then resident, Dr Paul Matthews,[1] now of Shrewsbury.

I give practically verbatim his account of it, and I take this opportunity of thanking him for the most valuable assistance which he rendered me in connection with it.

The case was that of E. M., a youth of 19, who was admitted to the Edinburgh Royal Infirmary on 6th January 1904, complaining of pain in the left side of three weeks' duration. His previous illnesses included diphtheria eight years ago, and measles during childhood. He had had at the same time broncho-pneumonia, and his chest had been weak since. He had had a cough, on and off, for nearly ten years.

His family history revealed nothing of note, except the occurrence of tubercular caries of the spine in a brother. He was a surveyor's apprentice, and his social conditions were satisfactory. His present illness had commenced three weeks before, when he complained of sharp pain in the left side of the chest, a little below the nipple. This got worse, and he had to stop work, and lie up with it for a day or two. He improved a little, and resumed work for about a week, but the pain returned and became suddenly worse. He had been suffering from his usual cough, which had been no worse than it was previously, but had been aggravating the pain considerably. He had a delicate appearance, and his musculature was rather poor. He had a marked flush, but no cyanosis; temperature 101·4°.

Respiratory System.—He complained of sharp pain on the left side, most marked just below the nipple, but also felt posteriorly over the base of the lung. It was increased on coughing and on deep inspiration; respirations 28 per minute. His cough was somewhat paroxysmal, and

[1] A case of localised empyema associated with the presence of a leptothrix. *Practitioner*, Feb. 1905.

accompanied by small quantities of muco-purulent spit, negative as regards tubercle bacilli.

Examination of chest showed it to be rather flat but fairly well formed. Expansion equal on the two sides. Vocal fremitus well marked everywhere, except over the left base posteriorly, where it was slightly lessened.

Percussion showed slight impairment of note over the left base posteriorly, and also a rather high-pitched note over the left apex posteriorly.

Palpation revealed the presence of an area of tenderness about 3 in. in diameter over the fifth to seventh interspaces below the left nipple. Percussion caused considerable pain there, and it was also the seat of pain when he made any sudden movement. The breath sounds were vesicular — rather harsh in character in front, a trifle weakened over the left base posteriorly. Some slight very soft friction could be detected over the painful area.

For the ensuing three or four days there was little change in patient's signs and symptoms. On 9th January he had an attack of pharyngitis which subsided under treatment, and his temperature, which had risen to 101·8° on 8th January, gradually came down to 99° on the 11th, after which it commenced to rise again.

On the 12th a slight diffuse swelling was observed over the tender area. There was no inflammatory redness, and fluctuation was absent. Examination of his blood showed a leucocytosis of 12,500. His temperature commenced to swing slightly, but was very irregular. Fluctuation was observable in the swelling on the 16th.

His temperature rose on the 18th to 102·2°, and on 19th January, under ethyl chloride as a local anæsthetic, an incision was made into the swelling. About 4 to 5 oz. of fœtid pus escaped. The cavity was explored. It appeared to be shut off from the general pleural cavity, and no

communication could be found with any diseased rib. A drainage-tube was put in.

On the 20th the temperature again rose in the evening, and about 1 oz. of fœtid pus was again evacuated. Thereafter the discharge lessened rapidly, and the tube was taken out on 24th January. The pus obtained was examined bacteriologically and found to contain staphylococci, together with numerous long filaments of leptothrix.

Patient improved rapidly, and he was allowed to sit up in a chair on the 25th. The same night, however, he complained of pain over the left base posteriorly, and examination showed the presence of an area of very acute tenderness about 3 in. in vertical, and 2 in. in horizontal diameter, over the angles of the ninth to eleventh ribs. There was no œdema or redness. On percussion (which caused great pain), there was dulness extending out towards the axilla slightly beyond the area of tenderness. Vocal fremitus was absent, and the breath sounds were distant and faint. An exploring needle was put into the tender area, and at a depth of about 1½ in. some very foul-smelling, thick, greenish-yellow pus was found. This was examined and found to contain numerous filaments of leptothrix, together with several large colonies of the same.

On the following day a needle was put in 2 in. further out, but no pus was found.

On the 29th Mr MacGillivray made an incision over the tenth interspace close to the edge of the erector spinæ. Pus was not found until an exploring needle had been put in several times. A small pocket of pus was then found about 2 in. from the surface and somewhat nearer the middle line. The pocket was opened up and a drainage-tube put in. The pus was in very small amount, and was foul-smelling and somewhat slimy. Subsequent examination showed the presence of leptothrix filaments in it.

Except for a slight rise of temperature on 1st February,

patient made uninterrupted progress, the discharge was very slight, and the tube was left out altogether after about a fortnight, the wound subsequently healing rapidly. Patient gained 9¼ lb. in weight, and was sent to the Convalescent House on 23rd February. He remained there three weeks, during which time he increased a further 12 lb. in weight. The wound was quite healed, and patient's general health was excellent.

The pus and the organism merit further description. The pus obtained was thick and foul-smelling, of a somewhat yellowish-green colour, that obtained from the second or posterior abscess being distinctly slimy. The pus from both collections showed the presence of numerous small yellowish granules of rather firm consistence. These were about the size of a small pin's head, and were most numerous in the posterior collection of pus. Examined microscopically, they were found to consist almost entirely of felted masses of leptothrix filaments, together with entangled pus cells and broken down red blood corpuscles.

The pus from the first or anterior collection showed the presence of numerous fine free filaments of leptothrix and also staphylococci. The pus from the second or posterior collection showed leptothrix, but no staphylococci at all.

The leptothrix organism occurred in both collections in the form of free filaments and in colonies. The filaments were of varying length. These filaments stained readily with the ordinary stains, the stain being retained rather irregularly. By Gram's method the stain was decolorised in most of the free filaments, but there was a tendency to retain it irregularly in some of the colonies. The filaments were not stained by the Ziehl Neelsen method. The filaments, in addition to showing some irregularity of staining, showed some irregular thickening along their length. The ends of the filaments were frequently slightly swollen, and

where this was the case the ends stained more deeply and had a greater tendency to retain Gram's stain. No branching could be seen.

Attempts were made to cultivate the organism on the following media:—Agar, bouillon, gelatin, glucose-agar, glucose-bouillon, and fresh slices of kidney. No growth of the leptothrix was obtained in any case. From the pus of the anterior abscess staphylococci were cultivated, but not from the posterior one.

A guinea-pig was inoculated with the pus of the posterior collection, with negative results.

The interest of the above case chiefly rests with the organism alluded to as a leptothrix. Without being definitely identified with the L. Racemosa of Vicenteni, it presented many points of similarity to that organism.

L. Racemosa is stated by Goadby[1] to be present in the mouths of most people. Williams[2] says it is present in practically all mouths. Though it appears to be associated with caries of the teeth, it cannot be said to have been proved to be pathogenic. It is of interest to note, however, that the above patient had eight or nine teeth extracted for caries (at the root of one of which a small alveolar abscess was found) three weeks before the onset of the pulmonary symptoms. It does not appear improbable that he may have inhaled some of the carious débris during the extraction, and that the leptothrix may have thus gained entrance.

[1] Goadby, in Smale and Colyer's *Diseases and Injuries of Teeth.*
[2] Williams, *Dental Cosmos*, 1899.

CHAPTER XIV

TREATMENT OF PLEURISY AND EMPYEMA

WHEN now one passes to the consideration of the treatment of pleurisy, whether acute or chronic, whether dry or associated with sero-fibrinous or purulent effusion, whether in its early or in its late stages, one meets with as good illustrations as can well be met with of some of the big general truths of Nature.

One sees, in the first place, that Nature is always working for good, and is always and in her own way indicating to us her methods towards this end. Thus in the early stages of an acute pleurisy, she indicates by the stabbing pain and cough, the necessity for respiratory rest, in order by lessening the to-and-fro movements of the inflamed pulmonary and costal pleuræ, to diminish the acuteness and limit the spread of the pleuritic process. In cases where, in spite of this, inflammation is still persisting, she brings about effusion, which separates the mutually irritating surfaces of pleura, collapses the lung, and induces an air scarcity, which tends to make the demand for the required respiratory rest all the more irresistible.

When in time the inflammatory process has terminated, Nature then, by bringing about subsidence of the pain and fever, and by inducing the return of appetite, digestive power, and strength, leads the patient to crave for bodily movements. These, by calling forth respiratory activity, tend in their turn to favour the reabsorption of the effusion, and the re-expansion of the lung.

But one sees also in pleurisy another important truth. This is that whilst Nature is always working for good, and indicating her own methods to us in her own way, she is inexorable as regards these methods. The result is that whether successful or the reverse from the individual point of view, these methods will always be employed.

For example, in a purulent effusion, Nature's method towards cure is to cause the effusion in time to burst, either into the lung or through some other part of the chest parietes. In a certain proportion of the cases in which the burst is into the lung, Nature is, as has been seen, successful. But in a very large proportion of cases she is not, and as we have also already learned, her chances of success are infinitesimally small when the burst has been elsewhere through the chest parietes. Still more unfortunate than this, however, may be the result of her efforts, for they may indeed directly prevent us doing the good which, but for them, we could easily have done. Let me give an illustration of this.

A small empyema, especially a small fœtid one, is detected and localised, so that chest opening and drainage would undoubtedly be a simple and successful matter. But before this can be arranged for, the pus bursts into the lung, so that though carefully looked for, it cannot again be found. Still present, however, this pus goes on inducing its destructive changes on the lung and its toxic influences on the system generally, the result of which is that in the course of days or weeks or months the patient may die. In such an instance there can be no doubt that but for the results of Nature's own efforts towards cure, the patient might have been put in the way of rapid and complete recovery.

All this reminds us that what we have to keep steadfastly before us in the treatment of all diseases is perhaps specially important in the case of pleurisy. This is, in each individual case carefully to strive to interpret what Nature is doing towards betterment, to judge carefully as to what her

measure of success is likely to be, to take full advantage of whatever success her efforts may obtain, and to interfere in her favour whenever these efforts are likely to be unequal to their task.

Keeping all this in mind, we realise that in a considerable proportion of sero-fibrinous pleurisies, and in a small proportion of purulent ones, Nature's efforts towards cure are such that treatment is to do little more than simply stand by. But we also realise that in the vast proportion of purulent pleurisies, and in a certain proportion of the sero-fibrinous, Nature's efforts may be so futile and possibly conflicting, that direct interference time after time is an absolute necessity if recovery is to be hoped for at all.

In the milder pleurisies, either dry or associated with sero-fibrinous effusion in small amounts, which run their favourable course in a few days or a week, it will be evident that rest more or less absolute, a flannel chest bandage to restrain respiratory movements, a restricted diet, and attention to the state of the bowels, skin, and kidney, is really all the treatment that may be required. But practically, certain of the symptoms of the disease are at one time or other apt to call for some special care, either because they manifest themselves too markedly, or because they persist too long. In this way to any means of combating these some attention must now be directed.

Chest Pain.—If this is distinctly severe, a good remedy is a fly blister in addition to the bandage. It need not be a large one—2 in. by 2 in. is the size I generally advise—and it is best placed over the part of the chest where the pain is most severe. One sometimes hears it said that the fly blister should be reserved for the later periods of pleurisy, when efforts to favour reabsorption are required. I agree, however, with the older physicians, in the view that blistering in the early stage of a pleurisy tends to lessen the risk of future trouble from effusion.

Instead of the blister, fomentations with or without turpentine, poultices, or the application of four or five leeches have all been recommended, and I have seen benefit from the use of anti-phlogistine to the side. When the chest pain is extremely severe, as in the pleurisies which are going to be purulent, the ice-bag, or morphia hypodermically into the side can often give great relief. When the pain is low down, as in diaphragmatic pleurisies, a morphia suppository may be used.

Cough.—Inasmuch as the cough in pleurisy is caused mainly by the rubbing together of the inflamed pleural surfaces, whatever, like the bandage, lessens this movement, will correspondingly relieve the cough. When in spite of this it causes distress, or when, as in a pleuro-pneumonia or in a phthisical pleurisy, it arises from the associated lung disease, special treatment for it is demanded. In the ordinary acute pleurisies a mixture of potass iodide with tinct. camph. co., and in the phthisical pleurisies the Begbie or the Brompton mixture will often relieve.

Fever.—In an ordinary sero-fibrinous pleurisy, when the temperature does not rise above 101° or 102°, no special treatment is required beyond the rest in bed and the general means already described. The fact that pleurisies are not infrequently associated with the rheumatic element, may however suggest possible benefit to be obtained from the use of such drugs as salicin, quinine, phenacetin, etc., and of course the potass. iodide. For the higher temperatures which are met with in cases of pleuro-pneumonia and purulent effusion, these drugs may also be used, and it is to be remembered that the ice-bag on the painful chest-wall, and that tepid sponging have also a like beneficial cooling effect. In such cases, the heart's action may cause some disquiet, and then digitalis or strophanthus may be useful alike for this and for the fever.

When, with a fever which may be below or above 102° F.

a tubercular element in the disease is manifest, the advisability of open window treatment along with red meat and red wine has to be considered. In my experience, however, it is rather in the later and effusion stages that the special treatment of tubercular pleurisies is most important.

Effusion.—Whilst a sero-fibrinous effusion is collecting, beyond carrying out the treatment already mentioned, we as a rule have only to stand by. We know that the fluid is salutary, in that it separates the mutually irritating pleural surfaces, and in that it brings about lung rest. We know further that at any moment if it should become so great in quantity as to entail real respiratory embarrassment, we can at once relieve by aspiration. All that we have to do therefore is to be sure that the fluid is sero-fibrinous, and to be sure of this the exploring needle is really the only trustworthy guide. This point will, however, be more appropriately emphasised when the treatment of empyema is being discussed.

Reabsorption of the Effusion.—When the fluid has ceased to accumulate, reabsorption is to be looked for, and for the promotion of this, various plans may be followed. In the first place, remembering that Nature is able in many cases to accomplish this by herself, we watch carefully to see what she is doing. In favourable cases we note a lessening of the fever, and improvement in the general condition as associated with it. When the effusion has completely filled the affected pleura, the earliest sign of reabsorption is a rise in the diaphragm, and this is quickly followed by a return of resonance at the apex of the affected lung. Thereafter, as the days follow one another, we look for the "curved line" forming, and becoming gradually lower and flatter. When the reabsorption process has gone on to the extent of leaving the chest about a third or a quarter full, and more especially when along with this the patient's general condition is so much better that he is beginning to crave for movement, we

follow Nature, and let the patient get out of bed and move about gently. Here let me make a digression to emphasise the importance of following Nature in this matter. It has been my lot on many occasions to be asked to see cases of sero-fibrinous pleurisy which had been running a quite favourable course, except that the reabsorption process, having progressed so far, seemed to be hanging fire, in spite of counter-irritation and general medication of all kinds. In such circumstances I have often found that the doctor has been over-anxious and over-cautious in the way of keeping the patient in bed, and that if leave to get up is granted and acted upon, little else is required to cause reabsorption to be completed. What I consider the wisest course at this stage is to discover if the patient has any desire to get up, and if he has, to let him do so, carefully watching the temperature chart for any return of fever, and carefully consulting the patient for any return of chest discomfort or pain—conditions which would naturally render a return to bed necessary.

To still further promote reabsorption some form of gentle exercise—stair drill, *i.e.*, ascending so many steps once or twice daily; dumb-bell or light club exercises; blowing a wind instrument—the chanter is a good one for this purpose —may all be tried, cautiously at first, and gradually increasing in vigour.

Counter-irritants are useful in certain cases: painting with iodine, small fly blisters, 2 in. by 2 in., repeated at various parts of the chest-wall—perhaps better, a large one, 8 or 9 in. by 4 in., laid along the lower part of the chest— and the rubbing in of the blue or red mercurial ointments, have all their uses. By the older physicians, the large fly blister was very favourably regarded. I once saw one 9 in. by 9 in. employed with good effect, and although I have seen smaller sizes than this produce kidney disturbance, no permanent harm resulted. Further to promote reabsorption, the patient may be advised to drink as little fluid as possible,

and with this the administration of cardiac tonics, diuretics, and laxatives may be followed by favourable results.

Aspiration.—Aspiration of the effusion is indicated whenever respiration is embarrassed, and whenever reabsorption is being delayed. Alike as regards indication for and methods of aspiration, some important considerations must now be dealt with.

In the first place, as regards respiratory embarrassment, we frequently notice very great differences in different individuals. As has previously been pointed out, we may meet with one man walking about and making little complaint of breathlessness, although his chest is full from apex to base, and we may meet with another who, even resting quietly in bed, may be calling out for the relief of his dyspnœa, although his chest is only one-third full. One explanation of this difference may be, that in the first class of patients the effusion has been very slow in collecting, and in the second it has been very rapid. But probably another factor among the latter class is a constitutional nervousness or sensitiveness which makes them feel anything like air-hunger very keenly. The plan which I am in the habit of following as regards this latter class is to explain to them that the effusion of fluid is one of Nature's steps towards the curing of their ailment, and that if the presence of this fluid can be "tholed" to some extent, it will be better for them in the end. If with this counsel, and with the administration of stimulants, anti-spasmodics, or sedatives, the breathlessness is still complained of, I then aspirate. Inasmuch, however, as the aspiration then is merely for the purpose of relieving the breathing, I do not endeavour to remove any more fluid than is necessary at the time for this purpose, and I have seen relatively small quantities—10 to 20 oz.—prove quite sufficient. On the other hand, in elderly people, in whom the heart is rather feeble, it must not be forgotten that small effusions may cause serious embarrassment, and that then

the relief which aspiration can give is most valuable. In such patients, however, one has often very great anxiety, for it is in the pleurisies of the elderly that sudden and unexpected heart failure is apt to occur, either before aspiration has been employed or during its performance.

When the quantity of fluid is large, the onset of respiratory distress is of course the signal for aspiration, and any quantity, from 60 to 120 oz., may then be drawn off with advantage.

And now as regards the methods of drawing off the fluid.

The seventh or eighth interspace in the posterior axillary line is the point usually chosen, but when there is plenty of fluid, a site further forward, *e.g.*, the mid-axillary line, may be preferred. The advantage of this latter site is that the aspiration may be carried out with the patient lying quietly on his back during the whole time of the operation, the head and shoulders only being raised. If the site more posterior be chosen, the patient must be sitting up in bed, or propped up in a semi-recumbent position.

If the trocar is not a large one, if it is sharp, and if it is introduced exactly in the middle of the interspace, so as to completely avoid injuring the rib, the pain of the introduction is not so great as to demand anæsthesia local or general. A little whisky or brandy before, during, or after the operation is often useful in case of sudden pain or faintness.

I as a rule prefer the ordinary aspirator to the syphon arrangement. I always take care, however, not to exhaust the air too much, and so in this way make the withdrawal process a slow and gradual one. If as the result of tearing adhesions, severe chest pain is complained of, or blood appears in the fluid, I regard this as an indication to stop the flow for some minutes, and then recommence. And whilst with large effusions one always wishes to withdraw a proportionately large quantity of fluid, I believe it is at

times better to stop the aspiration at an incomplete stage, and repeat it another day, than to continue it. It is to be remembered that a rapid and complete lung re-expansion may mean renewal of the to-and-fro movement of the pulmonary and costal pleura, and possible recrudescence of the pleuritic irritation. Moreover, with sero-fibrinous pleural effusions, it is not necessary to remove every ounce of the fluid, and this is specially so if striving to do this entails pain and distress to the patient.

The other indication for aspiration is when reabsorption is being too long delayed, and of these delayed resorption cases, two main classes may be recognised. The first comprises cases in which, with continuation of the fever, the process of reabsorption does not appear to be beginning after two or three weeks of illness. The second class comprises those cases in which, after an illness of a week or thereabout, reabsorption begins, but after another week or thereabout, it ceases. In this way, as the days go by, the patient, although better as far as fever is concerned, has yet a large quantity of fluid in his pleura, which shows no signs of disappearing.

In both of these classes the pleurisies are of the kind which are apt to prove tubercular, inasmuch as they occur in individuals in whom the trophic power is so low that the ubiquitous tubercle bacillus has had little difficulty in asserting itself, either at the start of the pleurisy, or after it has gone on for weeks or months.

Fortunately, however, whether the tubercle bacillus has asserted itself or not, a fair proportion of these cases run a favourable course. In those of the first class, in which as stated the fever keeps persisting for three weeks or thereabout, and in which the large effusion shows no tendency to disappear, a single large aspiration may be at once followed by subsidence of the fever, and in time complete re-expansion of the lung. In those of the second class also, in which

the fever has largely disappeared, and in which the process of reabsorption has progressed to some extent, a single aspiration may similarly be sufficient to allow Nature to bring about complete recovery.

But in both cases what one sees happen too often is that the first aspiration has after two or three weeks been followed by a second, this second by a third, and so on. In such instances, with imperfect lung re-expansion, and with some thickening of the pleura and falling in of the chest-wall, the patient eventually either recovers with permanently enfeebled health and a maimed lung, or else dies, as the result of tubercular or other lung development and spread.

For patients in which such developments seem likely to impend, the treatment which I have always advocated is that of repeated aspirations at short intervals. This procedure is equally applicable to cases in which a large first aspiration is found after a few days to be succeeded either by a reaccumulation, or by indications that further reabsorption is not taking place, and to cases in which the first aspiration has caused severe pain, cough, or bleeding, and so has had to be stopped after a small quantity only of the fluid has been removed.

In such cases the drawing off every four to seven days of whatever quantities of the fluid will easily come away, using a small aspirating needle, so as to cause a minimum of pain, and inserting it at whatever parts of the affected side yield fluid most easily, will often be found most efficient. By this plan the advantage to the lung gained by one aspiration is not lost by reaccumulation of the fluid. Each succeeding aspiration thus gains for the lung a little more expansion than the preceding one, adhesions have time to form between the re-expanded portions of lung and the costal pleura, air-cells are kept expanded and expansile, and the blood-vessels and lymphatics of the lung are preserved in their integrity.

The case of J. B., quoted at p. 68, may serve as an

example of this treatment, and I could relate others more marked than this, in which it was successful.

But treatment by repeated tappings is also specially applicable in cases of phthisical pleurisy. It must be obvious that when pleurisy and effusion supervenes in a patient whose lung is already the seat of phthisical softenings and excavations, the collapse of the lung thus induced must to a certain extent mean the obliteration and healing of these. I say to a certain extent only, because the cavities, etc., being mainly in the upper lobe, and the collapse caused by the effusion being mainly of the lower, anything like their complete obliteration can hardly be expected. But an important point to be remembered is that in a phthisical lung there is always a tendency to shrink, and that the lung collapse caused by a pleuritic effusion must materially favour this tendency. Inasmuch further, as this shrinkage is one of Nature's methods of healing tubercular lung destruction, it will be evident that when pleuritic effusion occurs with a phthisical lung, a certain salutary influence must be recognised in it. It must be evident also that if in such a case aspiration is performed, and any large quantity of fluid is suddenly removed, the consequent re-expansion of lung may, by the forcible opening up of its diseased portions, bring about serious harm. I have seen a huge hæmoptysis follow on aspiration in a phthisical lung, and the late Dr Brakenridge informed me that he had seen this operation in similar circumstances followed by a fatal pneumothorax. Still, as it is very important to get quit of the effusion, this, as stated, is best done by this plan of small aspirations, repeated every few days, or as long as fluid can be obtained. A small-sized needle should be used, and in a case for example in which the pleura is about one-half full, we may begin with quantities of 10 to 20 oz. It is specially important in such instances to stop aspirating if severe pain be complained of, or if blood appear in the fluid, and so to avoid too forcible tearing through

of any existing adhesions. In such cases, the constitutional treatment for phthisis—red meat, red wine, and open air—should also be carefully observed. It is to be remembered, too, that for them treatment with the tuberculins is sometimes employed.

In practice, however, every now and again we meet with cases which give trouble as the result of the extremely rapid accumulation of the fluid. I have seen such in the pleurisies of chronic Bright's disease, in which the effusion seemed to be pathologically a hybrid between pleuritic exudation and serous transudation, and in these, frequent copious tappings are just as necessary as they are in hydrothorax. But now and again, and especially in tubercular cases, we meet with instances in which repeated tappings prove inefficient. Pleurisies, for example, in which quantities of 60 to 100 oz. have to be drawn off every week or thereabout for months, and in which the amount removed has been as great at the last tapping as it was at the first. What, then, is to be done for such?

Always premising that the local and general treatment already described has been fully tried, and specially that repeated tappings have been given a chance of proving their efficiency, further methods to which we may have recourse are—

1. The drawing off of a certain amount of the fluid, and allowing sterilised air to pass in in its place.
2. The introduction into the pleural cavity of such substances as adrenalin or formalin solutions.
3. Free drainage, either by incision, with or without resection of a rib, or by a canula and tubing.

I shall consider the third method first, as this will enable us to understand the *modus operandi* of all three.

As just stated, it may be accomplished by free incision and insertion of a drainage-tube, with or without rib resection ; or it may be carried out by means of a large-sized

trocar and canula and a piece of tubing. The size of canula which I have often used is one which will allow No. 8 drainage tubing to pass through, and the procedure is first to make an incision through the skin, and then having pushed the trocar and canula through the interspace, to withdraw the trocar and pass the tubing, preferably with side openings, into the pleural cavity. The canula is then withdrawn, leaving the tubing, which can be fixed in position by a safety pin and strapping. Alike with this plan, and with the free incision, antiseptics must be carefully observed, and during the operation the greatest caution must be taken to prevent too rapid egress of the fluid. It must be remembered that contrary to what one meets with in purulent effusions, the lung is usually freely collapsible and expansile.

There is no doubt that carried out in either of those ways, this method of free drainage may prove successful where repeated tappings have failed.[1] How it succeeds is in that it seems to exercise such an influence on the pleural membrane as in time to stop its secretion. Usually the operation is followed by watery discharge in huge amount, soaking through dressings, and it may be, requiring their complete change several times daily. After some days, however, a change is noted. Possibly associated with some slight feverishness, the secretion suddenly ceases, and then withdrawal of the tube and closure of the wound is rapidly following by reabsorption of the retained air and re-expansion of the lung. In other instances, after some days, and also associated with some fever, the discharge becomes purulent. This again may be not unfavourable, for with gradual diminution in the amount of discharge, the lung gradually re-expands, and the patient recovers.

In these cases where free drainage proves successful, one cannot help calling to mind the similarly excellent results

[1] For examples of such, see West, *Brit. Med. Journ.*, 27th April 1895, and Rutherford Morison, *Brit. Med. Journ.*, 13th July 1895.

which may follow simple exploration and letting in air into the abdominal cavity, in tubercular and other morbid affections of the peritoneum. The general conclusion which we can draw is that in some way or other the access of air has been able to influence beneficially the abnormally secreting serous membrane.

But unfortunately it has to be borne in mind that the results of this treatment are not always thus favourable. The copious discharge may continue copious for weeks, the patient rapidly becoming feebler, and dying from some rapid tubercular spread, or with purulent transformation of the secretion, and more or less marked fever, a fatal result may ensue from exhaustion.

But now, the fact that in those cases of long continuing effusion free drainage and the access of air may beneficially influence the abnormally acting pleural membrane, explains to us how the other methods mentioned can exercise a similar influence. Thus, the introduction into the pleura of sterilised air in the manner proposed by Ewart, Barr, and others may be believed to act in exactly the same way.

Similarly, it is by influencing this over-secreting pleural membrane that the intra-pleural injection of adrenalin or formalin solutions will exercise their effects. Personally, I have had no experience of formalin injections,[1] but with adrenalin the plan which I have followed in cases where repeated tappings seemed insufficient was the one recommended by Ewart. This is, having drawn into the barrel of the exploring needle 10 drops of the 1 per 1000 solution of adrenalin, to introduce the needle into the chest, and draw into the barrel a like quantity of the pleural fluid, then to drive the mixture of fluid and adrenalin into the pleura. In this way, the very diluted adrenalin is brought into contact

[1] Chapin, *Boston Medical and Surgical Journal*, April 1898, reports a case in which 1 oz. of glycerine containing 10 drops of formalin were injected successfully.

with the pleural membrane, and thus the excessive secretion may be checked. Where necessary these injections of adrenalin may be repeated, and I have certainly seen instances where the combination of adrenalin injections and tappings proved most efficient.

The treatment of *purulent effusion* is to incise the chest and drain off the pus, and in an ordinary acute frank empyema, this treatment is usually as simple as it is successful. In such a case we can with every confidence predict that if the chest is at once opened and drained, the patient will be able to be out of bed in two or three weeks, and the tube will be out of his chest in four or five. I need not in this work enter into all the details of the operation for empyema. In most instances at the present time, a piece of rib is resected; but I have seen many acute cases quite successfully opened and drained without resection. As already stated, in those acute cases, purulent secretion will have stopped, and lung expansion will have become complete in four or five weeks, so that if ribs and interspaces are normal, I am one of those who believe that rib resection is unnecessary. But what I always plead for is that whether resection is done or not, the opening through the chest-wall should be kept large, and the drainage-tube should not be dispensed with or shortened too quickly. The large opening enables discharge, and more especially shreds and flakes to be coughed out by the patient during the earlier dressings, and if the empyema is at all a large one, a rather long tube lessens the risk of retention and loculi formation.

During the operation, and when the patient is under chloroform, the best position is one which will allow of him lying as much as possible on his back, and as little as possible on his sound side. This makes manipulation inconvenient and awkward for the operator, especially if rib resection has to be done, but in the patient's own interest this must just be tolerated. When the amount of pus is

very large, the same care which is observed in sero-fibrinous effusions should be taken to prevent it escaping too rapidly; and for the same reason, for although the lung is fixed in empyema, as it is not in sero-fibrinous effusions, a too sudden alteration of the physical conditions in the chest may have a disastrous effect on the heart. Further, as early as possible after the operation, the dressings, cleansing of tube, etc., should be done, with the patient sitting up in bed. Until convalescence has begun, the four-hourly chart should be kept up, and any rise of temperature should be an indication to guard against retention of pus or of shreds and loculi formation. The patient should be encouraged to cough during the dressings, and whatever length of tube has been used, it should, as already stated, not be shortened too early. If a loculus does form, the pus increasing in volume will then be able to find its way more readily into it.

In *double empyema* the required operative measures are the same, but their carrying out naturally causes rather more concern. Fortunately, however, experience shows their treatment as a rule to be efficient and successful.

In cases in which the empyemas are small, both may be opened at the same operation without risk of harm from pulmonary collapse. As, however, it is well that the procedure should be carried through as rapidly as possible, simple incisions and the insertions of drainage-tubes are distinctly preferable to rib resections. Such a simple operation may be carried out with a minimum of chloroform anæsthesia, or under cocaine locally.

In cases in which they are large, one, preferably the larger, may be opened and drained, and the other left for a few days; or if the patient at the moment is suffering from respiratory embarrassment, the other may at the time be simply aspirated, and after a few days be in its turn opened and drained.

In *fœtid empyema* washing out the cavity is, in the

majority of cases, inadvisable and unnecessary from beginning to end, as for the most part the fœtor will disappear in a few days with proper drainage. When it does not do so, or if the fœtid fluid is associated with gangrenous processes in lung or chest-wall, washing out of the pleural cavity may have to be employed. This must be done very carefully. The plan which I have followed is to let fall some warm boracic, or some very dilute carbolic solution, drop by drop, into the pleural cavity, and after one or two ounces have collected, to make the patient cough it out. This can be repeated several times at each dressing.

But although, as has just been stated, the treatment in an ordinary acute frank empyema is as simple as it is successful, one is not long at work before one meets with cases presenting difficulties. When, after years of experience, one contemplates these difficulties with the view of being forewarned and forearmed, one may, I believe, appropriately range them in four classes.

1. Difficulties arising from the fact that the empyema, though existent for weeks or months, has not been suspected, and so not looked for.
2. Difficulties arising from the fact that the empyema has been suspected, looked for, but not found on exploration.
3. Difficulties arising from the fact that the empyema, although suspected, looked for, and found on exploration, is not found on operation.
4. Difficulties arising from the fact that although the empyema has been diagnosed, opened and drained, yet that owing to some constitutional or physical cause the purulent secretion persists, the collapsed lung, enveloped by thick false membrane, does not expand, and the chest-wall, more or less rigid and immobile from narrowing of the interspaces and approximation of the ribs, does not further fall in.

In this way there is left between the lung and chest-wall a cavity which varies much in size in different cases, and from the walls of which purulent secretion oozes forth in varying degrees.

1. Difficulties arising from the fact that the empyema, though existent for weeks or months, has not been suspected, and so not looked for.

I have always taught that in practice the large empyemas seldom give any serious trouble. Either they are diagnosed as such, explored or drained, or else a diagnosis being made of pleuritic effusion, and the performance of an aspiration revealing its purulent character, the proper treatment by incision and draining is at once carried out. The difficulties which arise as the result of unsuspected empyemas are mainly with the small ones. These, usually situated at the lower part of the lung posteriorly, may yet be present at any part of the costal or diaphragmatic surface, or may be interlobular, and the symptoms and signs may differ little, if at all, from those which one associates with a slowly resolving pneumonic or catarrhal pneumonic condition. Fortunately, it may be, Nature herself often comes to the rescue in these cases, and the sudden and unexpected expectoration, after weeks or months of illness, of a quantity of non-fœtid or fœtid pus, demonstrates alike the true nature of the malady and the *vix medicatrix naturæ*. But as we have seen, Nature is not always able in this way to obtain complete success. Indeed her efforts may have the unfortunate effect of making the work of the attendant the more difficult, for the purulent collection being still present, and hardly less capable than before of causing disaster, is now so much smaller in size by the escape of the pus, that it is all the more difficult to find with the exploring needle. All this shows the importance of exploration in every suspicious case; and although the finding of the pus may be a difficult matter, it is often wonderfully easy. I have

not infrequently found pus just inside the costal wall, in cases where the empyema had been existent and opening into the bronchial tube for over a year. Oftener it is more difficult to find, but this is better considered under the next class of difficulties.

2. Difficulties arising from the fact that the empyema, though suspected and looked for with the exploring needle, is not found.

In such instances, whether pus has been expectorated or not, we must just carefully examine the lung all over, and wherever we find an area, the physical signs at which are suspicious, we must thoroughly explore it at different depths. An X-ray screening may help us, and we can usually tell by the feel of the exploring needle whether it is going through solid or fibroid lung and thickened pleura or not. Further, although the lower parts posteriorly are those most usually affected, we must never lose sight of the fact that, as shown in Chapter IX., purulent collections may be found at any part of the lung.

To the patient and to the patient's friends, the pushing in of the exploring needle every few days, and especially so when the effort to strike pus proves a failure time after time, is both discouraging and painful. We must just, however, make clear to them the real necessity for such repeated efforts, and by using as small a needle as will be efficient, and by introducing it each time without injuring the rib, we must reduce the pain of each exploration to a minimum. We can tell our patient also, and with truth, that though we may eventually even fail to localise the pus collection, yet that these punctures often do good of themselves. By forming small channels through the thickened portions of lung tissue, they often aid the escape through to the bronchi of small quantities of pus, which, if retained, would be capable of doing great harm.

It has been pointed out at p. 140, that after an

empyema has opened into a bronchial tube, the amount of pus being expectorated daily is often very great, although the amount of empyema pus is often very small. It was explained there that this is due to the fact that in many cases the pus expectorated is rather the result of a purulent bronchorrhœa, set up by the pus, than of the emptying of the empyema cavity. Still, this purulent expectoration in a certain proportion of cases comes direct from the empyema cavity, and it will be evident that as the cough with the copious purulent expectoration is often paroxysmal, there will be times when the empyema cavity will be relatively full, and times when it will be relatively empty. It will be further evident that it will be more easily struck by the exploring needle at the former times than at the latter. Inasmuch as paroxysms of cough with purulent expectoration are apt to occur just after waking in the morning, a good time to explore is the early morning, before such paroxysm has occurred. I have frequently seen exploration in the early morning prove successful, when at other times it had failed. For such cases, Rutherford Morison[1] suggested the administration over night of a dose of morphia, so as to diminish cough and expectoration, and as it were "imprison" the pus and make it more easily found.

But in cases where we feel sure that purulent collections are present, although repeated explorations have failed, and especially in long-standing cases in which the discharge of pus through the bronchus has led to lung fibrosis and dilatation of the bronchi, there is a plan which I have always advocated as a very good one. This is to have a pleural window made, and to explore through it. The patient being chloroformed, an incision is made over the suspected area and about an inch and a half of contiguous rib resected. In this way a considerable area of pleura is exposed, and if the right site has been chosen, the pleura

[1] *Brit. Med. Journ.*, 1909, vol. ii., p. 1676.

will be found thickened and adherent to the underlying fibrosed lung. The wound is then packed, and the patient sent back to bed. The advantage of this procedure is that on subsequent days, not only can we explore a very much larger area of pleura and lung, but that this can be done with comparatively little pain to the patient, as the pleura and lung are relatively insensitive. In making these subsequent explorations it will be found best, I believe, for the patient to be sitting up in bed. Instead of the ordinary sharp exploring needle, one of like size but made with a probe-like point and a side opening will often be found advantageous. It can easily be pushed through the lung tissue in all directions, and whilst it can readily reveal loculated empyemas, or bronchiectatic tubes, there is less likelihood of its causing injury to large blood-vessels at the root of the lung or to diaphragm at the base, than there would be when manipulating in these neighbourhoods with a sharp needle. When the pus is struck, a larger communication with it is made by a probe-pointed forceps, and through this the drainage-tube is then easily enough introduced.

In cases where the empyema has opened, not into a bronchial tube, but through the chest-wall, the finding of the pus is usually not very difficult. As has been explained at p. 143, whilst the "point" is usually about the fourth or fifth spaces anteriorly, the empyema cavity is usually about the lower lobe posteriorly. There or thereabouts, therefore, we must look for it, and drain it when found. From what has already been stated as regards empyemas opening through the chest-wall, it must be evident that except in very rare instances, any attempts to drain them through their own openings would be hopeless.

3. Difficulties arising from the fact that the empyema, although suspected, looked for, and found on exploration, yet cannot be found on operation.

Cases in which pus has been readily enough found by the

exploring needle, and yet in which—perhaps next day, and when the patient is on the operating table — another exploration performed for the satisfaction and guidance of the operator fails to find pus, occur every now and again.

By far the most frequent cause of this in my experience has been the fact that the empyema to begin with has been a small one, and that in the interval between the exploration and the operation, it has opened and largely emptied itself through a bronchus. If now the operation be proceeded with, and the pulmonary pleura exposed, one is often surprised at its smooth glistening appearance, and at the marked absence of signs of inflammation which it presents at the part where pus has assuredly been present. The finger, however, cautiously introduced, will certainly feel adhesions in one or other direction around. If cautious probing of these, in the direction in which physical signs indicate the presence of fluid, does not at once reach pus, what I generally advise is that the wound be packed, as in long-standing cases, and a pleural window left for exploration and draining on subsequent days. In this way then, by a bent probe or by the sharp or blunt pointed exploring needle, surrounding areas can easily be explored.

Inasmuch as fœtid empyemas have a specially great tendency to burst through lung tissue into bronchi, they are apt to give trouble in this way much more than non-fœtid ones. I believe, too, that I have seen cases in which the exploratory puncture has directly hastened this escape, by the point of the needle accidentally entering the lung, and in this way forming a channel for the pus to find its way into a bronchial tube. Anyhow, I am strongly of opinion that with fœtid fluids the shorter the interval allowed to elapse between their detection and their drainage the better. These empyemas are frequently quite small in size, and once having opened into the lung, they become smaller still, and

very difficult to find. Yet they are there, and their more or less constant discharge of fœtid corroding pus through lung tissue and bronchus will often go on till they are detected and drained.

But again, the formation of loculi of pus which prove very difficult to get at, is common also in cases where purulent collections have been removed by simple aspiration. Such loculi may be diaphragmatic, costal, or interlobular. As time goes on, their increasing size by pus accumulation may be an advantage, for it may lead to their more easy detection by the exploring needle. But they may burst into a bronchial tube, and then prove specially difficult to be discovered, so that in such cases the making of a pleural window may be again necessary.

In this connection, a matter which merits reference is the suggestion that in very large empyemas, a preliminary aspiration should be made, in order that, by the removal of a quantity of the pus, the subsequent incision, resection, and drainage under an anæsthetic may be performed with less risk. My own inclinations are, however, distinctly against this procedure, and mainly on account of its increasing the risk of loculi formation. Such large empyemas are generally acute, and for them rib resection is not required, so that if the patient be kept as much as possible on his back, and if care be taken to allow the pus, especially at first, to escape slowly, the incision and drainage operation can be performed under chloroform without risk.

For cases of purulent loculi which cannot be drained, vaccine treatment[1] naturally suggests itself. My experience of this method is, however, small, as in the great bulk of my unsuccessful cases of this kind, the want of success was the inability to strike the loculus and obtain the pus. It will be at once evident that in this way the making of an autogenous

[1] Lyon, *Lancet*, 1905, vol. i., p. 1718, quotes a case of vaccine treatment.

vaccine was rendered as impossible as was the drainage of the purulent collection.

In a few cases, however, where one of several loculi or one of several bronchial dilatations had been struck, and was being drained, I have employed it; but these have been so few, that I feel unable to offer any trustworthy opinion as to its efficiency.

4. Difficulties arising from the fact that although the empyema has been diagnosed, found on exploration, opened and drained, yet that owing to some constitutional or physical cause the purulent secretion persists, the collapsed lung, enveloped by thick false membrane, does not expand, and the chest-wall, more or less rigid and immobile from narrowing of the interspaces and approximation of the ribs, does not further fall in. In this way there is left, between the lung and chest-wall, a cavity which varies much in size in different cases, and from the walls of which purulent secretion oozes forth in varying degrees.

Premising that all the ordinary plans for bringing about lung re-expansion have been tried in vain—exercises, forced expirations, blowing wind instruments, etc.—and premising that all efforts to improve the patient's general condition by fresh air, sunlight, red meat, and red wine have also proved inefficient, it will be evident that the mechanical approximation of pulmonary to costal pleura is in such cases the *sine qua non*. It will be equally evident that until this is done, purulent discharge will go on more or less from the pleural surface, and will entail, more or less, a corresponding drain on the patient's vital powers.

To bring about this approximation, certain well-recognised modes of procedure, to promote, in the first place, more complete drawing out of the lung, and in the second, more complete falling in of the chest-wall, are in vogue.

To promote more complete drawing out of the lung, plans of inducing a greater degree of negative intra-pleural tension

than is induced by the valve-like action of the dressings have been followed, and in Edinburgh, Duncan's syphon and Hutton's valve are well known. Another method, by the use of a pump, has been proposed by Grünbaum.[1] With any of these methods, however, very great care must be taken. A too great negative pressure may easily be produced, and cause severe pain and bleeding from the over-congested pleural vessels. The onset of such symptoms is to be sedulously guarded against, and in a general way it may be said that what we should aim at is the induction of a slight negative pressure which would act continuously, rather than that of a great negative pressure acting for shorter periods of the day.

When the binding down of the lung is so marked that all these methods are inefficient, decortication or the removal of the thickened false membrane of the visceral pleura, by dissection or curetting, has been advocated. The little I have seen of this operation leads me, however, to believe that the part played by this false membrane itself has been overestimated. We know that in empyema the pleural inflammation is of a much more intense and deeply spreading nature than in sero-fibrinous cases, and that in empyema, inflammatory changes affecting injuriously the integrity of the underlying air-cells must exist to a very considerable extent. It is obvious that these must act powerfully as a factor in this non-expansion. Still, decortication merits consideration. Inasmuch, however, as for it, access to the parts by an opening through the chest-wall is entailed, decortication as an operation is practically associated with the resections and other operative procedures, to permit more complete falling in of the chest-wall. These must now be referred to.

Estlander's well-known plan was to expose the ribs and interspaces anteriorly by a large curved incision, and then to resect portions of the ribs, the widest portions from the

[1] *Brit. Med. Journ.*, 1905, vol. i., p. 812.

centrally situated ones, and gradually diminishing the length of each piece upwards and downwards. It must be obvious, however, that the number, precise position, and extent of each rib resected must vary in different cases according to where and to what extent the falling in of the chest-wall is most required.

A plan which Hogarth Pringle[1] has advised is to resect small portions, alike from the anterior and posterior ends of the ribs, leaving their central portions for the most part intact. In this way it is claimed that approximation of parietal to visceral pleura can be obtained without the falling down of the shoulder and associated scoliosis, which are brought about by an ordinary Estlander.

By Thoracoplasty[2] is meant the removal, not only of ribs, but of periosteum, intercostal tissues or what remains of them, and of thickened and villous costal false membrane over the empyema cavity, leaving only the flap of skin and subcutaneous tissue to fall in and obliterate it. Theoretically, thoracoplasty presents the advantage, in the first place, in that all the parts left being soft parts, they can very easily fall in, and in the second, that the periosteum being removed, there is no chance of rigid bony tissue being again formed to prevent this. It is, however, an operation associated with serious risks, and if the empyema is at all an extensive one it should be done in stages. It will be evident that with any of those operative procedures, decortication may or may not be combined.

Such, then, are the plans suggested. But I am one of those who hold that except in certain tubercular cases, the need for them should never occur. If an ordinary empyema is opened, and if in the after-treatment drainage is efficient, and care is exercised to pick out and properly drain any loculi which may form, it is wonderful how well re-expansion

[1] *Brit. Med. Journ.*, 1905, vol. i., p. 811.
[2] See Ballance, *Brit. Med. Journ.*, 10th Dec. 1904.

will occur, even after many months have elapsed, and I have seen such do well even after a year. Of these ordinary empyemas which were slow in closing, the ones which gave me most concern were the very few which had followed trauma, and in which a hæmothorax had preceded the pyothorax. But each case must be judged individually, and when such operations are contemplated, not only must the position, size, and conformation of the empyema cavity be carefully ascertained, but the condition of the lung itself, its potentialities for expansion, and the nature, duration, and efficiency of the means already employed to bring this about, must be critically reviewed and judged upon.

A good plan in all cases in which it seems likely that re-expansion may give trouble, is to make, at a fairly early date, a chart of the chest and of the affected pleural cavity. This we can compare with similar charts made at subsequent periods, and in this way form an idea of what in the way of re-expansion the lung can do. The method which I have followed is, by the cyrtometer, to get a tracing of the chest at the level of the wound opening, and then by introducing a long probe through the opening in various directions in the horizontal plane, to mark out on this tracing the points at which it comes in contact with the inner surface of the pleural cavity. At the same time, valuable information as to the size and shape of the parts above at the apex, and below at the diaphragm, can also be obtained.

In the chart (Diagram XX.), the heavy line represents the condition of the right pleural cavity at the level of the sixth interspace, in a man some five weeks after a phthisical empyema had been opened and drained, and the dotted line represents the condition of the parts some five months later. This man had been suffering from right-sided phthisis and attacks of pleurisy for two years, and on his admission he was found to have a large empyema which had opened into

his bronchi. At the operation, 120 oz. of pus had been removed.

During the interval between the two records which the chart shows, an Estlander had been performed, and from 1½ in. to 2½ in. of the third, fourth, fifth, sixth, seventh, and eighth ribs had been removed. This had resulted in great improvement at the upper half of the chest, and there pulmonary and costal pleura had become quite approximated, but at the level represented by the chart it will be seen that the lung had hardly expanded at all. His respiratory capacity, as ascertained by the spirometer, had however

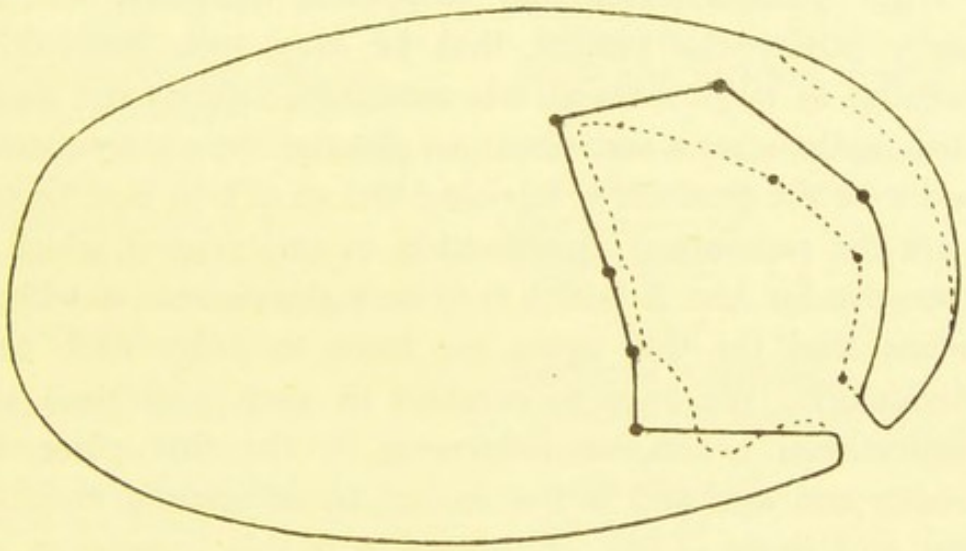


Diagram XX.

risen from 54 to 90 cub. in., and he had improved so very considerably in general condition, that although his case was one in which further interferences were distinctly indicated, he could not be persuaded to consent to them. It will be evident further that in such a case, with pronounced tubercular lung disease, resections or thoracoplasties would be just as appropriate, as would strenuous efforts to bring about lung re-expansion by valvular mechanisms or by decortication be hazardous.

Finally, as regards the treatment of pleuritic conditions during the convalescent stage and afterwards, it will be

evident, from what has been from time to time adverted to, that a difference of an important kind must be recognised between the sero-fibrinous and purulent varieties. This difference lies in the fact that whilst an acute empyema is for the most part like an acute pneumonia, or an acute specific fever, in that it can leave the patient in due course practically as well and as physically fit as he was before, an acute sero-fibrinous pleurisy is in large part more like a phthisical manifestation or a general breakdown, indicating that the conditions of the patient's life and work have been too much for his constitutional strength.

After recovery from an empyema, therefore, we can usually advise the patient that he may look forward to resuming in large measure his accustomed duties and mode of life, whilst after a sero-fibrinous pleurisy we are frequently faced with the problem of advising him as to how best he can secure the permanent amelioration in this respect, which is imperative for him if health is to be maintained. It will be evident that for this again we have to judge each case individually. We have to consider in each individual the constitutional health, as influenced, in the first place, by heredity and age, and in the second, by occupation, environment, and mode of life. Equipped with this knowledge, we have then to discuss with the patient and his friends how and in what ways the required ameliorations for his future can best be obtained.

INDEX

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