faulty or impaired diathesis. This is in marked contrast to the diathetic change produced in the system by vaccinia, or that produced by measles.

Now, vaccinia arises from an implanted virus (by contagion), measles also arises from an implanted virus (by infection). In neither case is the protected diathesis transmitted by inheritance.

These facts would almost entitle us to generalize or attempt to state a law, that *in diseases where there is an active causal virus which requires to be implanted, the diathetic change produced is not transmitted by inheritance.* Should this be true, the converse will also be true.

In this grouping, syphilis ought to be placed in the same category as vaccinia, to which it has many points of resemblance; and there is no reason for believing that the offspring of a parent who has recovered from syphilis inherits any protected diathesis.

As regards the immediate causes of disease, only a passing reference will here be made; but just as the potent causal virus of measles may exist in the atmosphere, so there are many other poisons existing around us in the atmosphere, on the bed clothes, and on the skin itself. These poisons may enter the body either by infection or contagion, and it is the knowledge of this fact which lies at the foundation of all the modern practice of antiseptic surgery.

All future great discoveries in surgery must be in the direction of increasing our knowledge of the causes of disease.

In an address a few years ago upon finality in surgery, Mr Erichsen pointed out that in some departments finality must be looked upon as having been attained. For example, all the arteries have been tied, probably as high as they ever will be tied, amputation has been successfully performed at the hip-joint, and no one is likely to attempt it any higher; such an operation as excision of the elbow-joint has probably been brought to as high a pitch of perfection as it ever can be, but it is altogether different when we come to consider questions regarding surgical diseases and their causation. Here, indeed, there lies before us the great ocean of the unknown, the great field for future discoverers.

II.—AN INQUIRY INTO THE METHOD OF CURE OF EMPYEMA, BEING AN INTRODUCTORY LECTURE TO A COURSE OF SURGERY.

By J. W. B. Hodsdon, M.D., F.R.C.S.E., Assistant-Surgeon, Edinburgh Royal Infirmary.

Gentlemen,—When I determined to deliver a course of lectures upon Systematic Surgery during this winter session, the question

which naturally arose in my mind was upon what subject should I first address you?

For my own part, I am inclined to think that introductory lectures, consisting as they usually do of platitudes, might well be dispensed with, especially as the time placed at our disposal to traverse completely and thoroughly the ground covered by a subject of such magnitude and importance as Surgery is so short. It being the use and wont, however, to deliver an introductory lecture, I had to select a suitable subject. In what follows I shall attempt to indicate the spirit in which you should endeavour to study Surgery, by discussing a subject which affords an admirable illustration of the method I would advocate.

Let me impress upon you at the beginning, that there is no such thing as so-called pure Surgery for him who would be a scientific and successful practitioner of this art, although I would not have you infer from this that there do not exist what are known as pure surgeons. A man may practise Surgery only, and at the same time have an intimate and accurate knowledge of Medicine and other sciences; in fact, it is from among such men that our most eminent and accomplished surgeons are drawn, and the reason of this is not far to seek. They take a broad and scientific view of their art, and do not regard their patients merely as subjects for an operation or for the application of this or that apparatus or splint.

It is the importance of looking upon Surgery from this broad and scientific point of view, and of bringing to bear upon it a knowledge of other sciences, that I would emphasize. Thus, while Anatomy belongs especially to the surgeon, and a thorough knowledge of it makes us accurate in our work and manipulations, and guides us clear of the shoals which lie in our course, we must not overlook the importance of Physiology and Physics. An intimate acquaintance with Medicine is, in the present state of surgical science, almost as indispensable to the surgeon as Surgery itself.

Let me give you a few examples. The antiseptic treatment of wounds was introduced by one who is a physiologist as well as a surgeon; electricity, which is of such value in the treatment of vascular tumours, and which is now being extensively applied to a variety of other surgical affections, could never have been utilized without an accurate knowledge of Physics. Would it have been possible, think you, for the recent advances which have taken place in brain surgery to have been made without a thorough acquaintance, not only with Medicine, but with Physiology also? Undoubtedly they would not; and this question cannot be better answered than by your reading an able address on this subject recently delivered before the British Medical Association in Glasgow.

There is yet another department of medical science in which

great progress has of late years been made, and which serves as a further illustration of what I have been pointing out to you. I refer to the surgical treatment of the lungs and pleural cavity; for while, until recently, abscess of the lung belonged exclusively to the physician, it may be now said to be almost entirely the property of the surgeon.

Having, I trust, sufficiently emphasized the importance of avoiding the pursuit of the study of our subject from a narrow point of view, and having shown you that some of the greatest achievements which have been attained by our art have been in directions where they would have been impossible had a knowledge of Surgery pure and simple been relied on; I shall not detain you further with general remarks, but proceed to discuss the subject of this lecture, namely, "An Inquiry into the Method of Cure in Empyema."

I selected Empyema as suitable for illustrating what I have just put before you, for several reasons.

In the first place, like so many diseases, it lies on the border line, so to speak, between Medicine and Surgery; and thus, while constantly coming under the care of the surgeon, requires a knowledge of Medicine. An acquaintance with Physiology and Physics is indispensable, in order to understand how a cure is to be scientifically brought about, and failure avoided; and its management, while illustrating many points of general surgical treatment, brings out very forcibly three most important ones—namely, the relief of tension, attention to free drainage, and the use of antiseptics. And I have further selected it, because it is a subject to which I have directed my attention for some time past. I trust to be able in what follows to place before you some observations which, so far as I can ascertain, have not hitherto been published.

By an empyema we mean a collection of pus or, as it is popularly termed, "matter" in the pleural cavity. The causes of this condition I shall not here enter into, because time will not permit of my so doing, nor can I, for a similar reason, dwell upon the diagnosis of this affection.

I shall merely endeavour to show you how in this disease a cure can be brought about; what are our best methods for attaining this end; why in certain cases we fail to obtain a satisfactory result; and the treatment to be adopted under these circumstances.

We may, for the sake of convenience, regard an empyema as a chronic abscess, under special conditions. Like a chronic abscess, its walls are frequently lined with a layer of granulation tissue; and there is, under certain conditions, a tendency to the formation of thick lymphy material. Like a chronic abscess, it can only be cured by its walls coming together after its pus has been evacuated. It is in the walls, *i.e.*, the lungs, chest-wall, diaphragm, and other structures in relation to this chronic abscess, in which these special conditions exist, and to which we have to direct our

attention in determining how the cavity closes when its contents have been let out.

In considering empyema from the point of view which I have indicated, and in order that we may have a clear conception of our aims in its treatment, it will be necessary to take up briefly—(1), The factors which enter into normal breathing, and the state of the pleural cavity (so-called) in health; (2), How the lungs, chest-wall, diaphragm, and other structures are affected by the presence of fluid in the pleural cavity? (3), What happens when, for the purpose of treatment, we let out this fluid and admit air? Having answered these questions, we shall be in a position to inquire—(1), How the cavity closes; (2), What gives rise to failure; and, lastly, I shall point out to you what are our best lines for treatment under the various conditions which may arise.

Before we proceed to consider the state of matters in health, there are two points to which I would direct your attention in order to avoid confusion as to terms we shall frequently employ.

We shall frequently speak of the pleural cavity, but you must bear in mind that there is no such thing in health; for the lung is always in contact with the chest-wall, and that a cavity only exists when air has been admitted or fluid formed.

Again, we shall constantly refer to negative pressure in the pleural cavity. With regard to this, you must clearly understand that this negative pressure is estimated with reference to the atmospheric pressure, which is 760 mm. of mercury; therefore when we speak of — 10, we mean $760 - 10 = 750$.

1. In the first place, then, let us consider *the factors which enter into normal breathing, and the state of the pleural cavity in health.*

The *lungs* are placed in a more or less distended state in the air-tight thorax, and when by the contraction of certain muscles, of which the diaphragm is most important, the thoracic cavity is enlarged, the pressure of the air within the lungs becomes less than that outside the body, and, in consequence of this difference of pressure, air rushes into the lungs through the trachea until an equilibrium is established. This constitutes inspiration. Upon relaxation of the respiratory muscles, the elasticity of the lung and chest-wall cause the chest to return to its original size; the pressure within the lungs is now greater than that outside, air rushes out of the trachea until equilibrium is again established, and this constitutes expiration.

Now, one of the most important factors in expiration is the elasticity of the lung tending to cause it to collapse, and we have to inquire why the lung in a semi-distended condition is pressed against the chest-wall. This elasticity we may talk of as the collapsing power of the lung, which of course is greatest when the lung is distended; thus at the end of a normal inspiration it is

equal to 9 mm., when forced 30 mm. to 72 mm.; at end of ordinary expiration it is equal to 7·5 mm., if forced 62 mm. to 100 mm. (Donders). This force of 9 or 7·5 acts against the intra-pulmonary pressure, which is equal to 760, and we may represent the state of matters thus at the end of inspiration: 760 — 9 = 751. The lung is therefore pressed against the chest-wall by a force equal to 751 mm. The atmospheric pressure equal to 760 acts on the chest-wall, but cannot, on account of the intervention of this structure, press upon the lung. Now at the end of expiration we have 760 — 7·5 = 752·5 as the force with which the lung is pressed against the chest-wall.

From these figures we must conclude that the negative pressure in the pleural cavity at the end of inspiration is 9, and at the end of expiration 7·5. It has been experimentally determined by Homolle[1] to be 10–12 at end of inspiration, and 6·5–4·5 at end of expiration.

In the normal state we have the *diaphragm* in contact with the lung; and the *heart*, according to Douglas Powell, is suspended between the two lungs. Now, when fluid has formed in the pleural cavity in consequence of inflammation, certain effects are produced upon the lung and other structures, and this leads us

2. To inquire *how the lung and these structures are affected by the presence of fluid.*

As regards the Lung.—We know that the lung tends to collapse, but it is obvious that this cannot take place until the pressure outside the lung is greater than that with which the lung presses against the chest-wall, that is 752·5 or 751.

Some interesting observations have been made upon the tension in pleural effusions by Douglas Powell[2] and Homolle.[3] The former states that positive pressure does not occur until the pleura is two-thirds full, and has found it to vary from 13 to 39 mm. of mercury at the beginning of the tapping. While Homolle found it to be almost always positive, sometimes as low as — 2 or 0, or as high as 20 or 30. But we must constantly bear in mind that anything over — 7·5 will compress the lung.

Let us calculate how much pressure will be exerted upon the lung under these circumstances. We may take the mean negative pressure in the pleural cavity as — 8, and the mean pressure exercised by the lung against the chest-wall as 754.

When the intrapleural pressure is—

− 2	we have	760 − 2 = 758	acting against	754 = 4	as the pressure on lung.
0	„	760 − 0 = 760	„	754 = 6	„ „
+ 20	„	760 + 20 = 780	„	754 = 26	„ „
+ 30	„	760 + 30 = 790	„	754 = 36	„ „

And thus we see that the lung is practically always compressed when there is any quantity of fluid in the chest.

[1] *Revue Mensuelle de Médecin et de Chirurgie*, February 1879.
[2] *Medical Times and Gazette*, vol. ii., 1882, p. 602.
[3] *Loc. cit.*

The amount of compression will, of course, depend upon—(α.) The quantity of fluid and the length of time the effusion has existed—the longer the more compression. (β.) State of the lung. When healthy and elastic, it will be more compressed and more readily expanded than when diseased. (γ.) Condition of the chest-walls. If these be rigid, pressure will be more concentrated upon the pulmonary tissue than when the opposite is the case.

From the state of the lung we now pass on to inquire if the blood-pressure is increased in the pulmonary artery in pleural effusions. I can find no observations on this subject; but one would, *a priori*, suppose that it is especially, should the lungs be elastic and easily compressible and the effusion recent.

The heart, and together with it the mediastinum, as Douglas Powell has pointed out, is displaced from the beginning in pleural effusion, and its displacement bears no proportion to the extent of the effusion. The diaphragm, which is kept arched by the negative pressure in the chest, will be depressed as this diminishes, and if the pressure be sufficient completely bagged down, but this, according to Powell, does not take place until the intrapleural pressure becomes positive. The chest-wall will become bulged out in proportion to its own elasticity and the state of the lungs. Lastly, we know that granulation tissue tends to form adhesions, and, therefore, according to the chronicity and nature of the effusion and the patient's general condition we find the lung more or less bound down.

Having considered the state of matters when there is fluid in the chest, we have now—

3. To take up *what happens when, for the purpose of treatment, we make an opening into the chest-wall.*

By this procedure we substitute air for fluid, for, as the latter flows out a free entrance of the former is afforded; and although the whole of the fluid is not usually at once evacuated, the entrance of air will be sufficient to cause a more or less complete collapse of the lung in proportion to the elasticity of the pulmonary tissue. Any portions of the lung which have not collapsed by the pressure of the effusion will now be compressed by that of the atmosphere, unless they be fixed to the chest-wall by adhesions. For we have $760 + 8$, the collapsing power of the lung, acting against 760.

The blood-pressure in the pulmonary artery will now at least be temporarily increased, the heart further displaced, and the diaphragm forced down.

Having considered the state of the lungs, diaphragm, and other structures when an opening has been made, we are in a position to inquire—(A), *How the cavity is closed?* and (B), *What gives rise to failure in this process?* I have stated, you will remember, that a cure can only be accomplished by the walls coming together. Bearing in mind, then, the special conditions of

this chronic abscess, it is evident that the cavity can only be obliterated by (α), the lung meeting the chest-wall; (β), by the chest-wall going to the lung; or, as actually happens (γ), by a combination of these means.

(*A.*) The question of *how the lung comes up to the chest-wall*, or, in other words, what are the forces which cause its re-expansion, is not yet definitely settled. Many explanations have been offered of this process. Roser[1] and Godlee[2] attribute it to the contraction of adhesions pulling the lung out bit by bit; Weissgerber,[3] principally to the forces of expiration, and Robertson[4] to valvular action at the wound. In what follows I shall endeavour to show you that it does not depend upon one factor alone, but upon many, and to draw your attention to some experiments which I have made bearing upon this subject.

We shall best understand the action and importance of these factors if we consider them in the following order:—(1), Normal breathing and its modifications; (2), Blood-pressure; (3), Movements of the chest-wall during respiration and valvular conditions at the wound; (4), The formation and contraction of adhesions.

1. *What is the part played by normal breathing?* This undoubtedly assists to a certain extent, for it is a physiological fact that the intra-pulmonary positive pressure during expiration is greater than the negative pressure during inspiration; and, according to Weissgerber, a certain quantity of air will be pressed during each expiratory effort from the sound lung through the bronchi and into the aveoli of the collapsed lung, causing expansion, while during inspiration, in consequence of the action of atmospheric pressure through the wound, the lung will be compressed. But the positive pressure of expiration is greater than the negative pressure of inspiration, therefore the lung will on each occasion occupy a greater volume—in other words, undergo an intermittent expansion. While this action of normal breathing in the expansion of the lung is not nearly so powerful as some other forces, it leads us to understand how certain of its modifications act; and of these prolonged expiratory efforts, and coughing especially, are of great importance.

We have seen that expiration in natural breathing tends to expand the collapsed lung, hence it is quite evident that in coughing, for example, when the positive pressure of expiration is still more increased, because more air is forced from the sound into the collapsed lung, benefit will accrue. Dr James[5] has pointed out that patients with an empyema can often be noticed to expire with a closed glottis. A very interesting case bearing upon this

[1] *Berlin. Klin. Woch.*, 18th Nov. 1878.
[2] *Lancet*, 9th Jan. 1886.
[3] *Berlin. Klin. Woch.*, 24th Feb. 1879.
[4] *Medical Chronicle*, March–July, 1888.
[5] *Trans. of Med. Chir. Society, Edin.*, 1886-87.

point was mentioned by Dr Sinclair[1] at a meeting of the Medico-Chirurgical Society in 1887, during a discussion on this subject. A patient of Dr Sinclair's, who happened to be an amateur cornet player, suffered from empyema, which did well, as is so often the case, up to a certain point, when the lung ceased to expand further, and an obstinate fistulous opening remained, which, however, rapidly closed when the man was permitted to resume his favourite amusement. We also know that in small wounds of the chest-wall the lung can be actually prolapsed during expiration; and it has been observed in dogs when the lung is exposed, that a marked expansion takes place on the animal attempting to cry.

I would have you note, then, that expiration, especially when forced, is of great importance in the closing of the cavity. We must bear in mind, however, that it is only when there is an opening that its benefits are so well marked. If there be fluid in the chest the lung can only be expanded by this means through an increase in the cavity of the thorax, for fluid is not compressible; when, however, there is an opening it is different, for air passes out during the expiratory efforts.

From normal breathing and its modifications let us now turn to (2.) *Blood-pressure,* and consider if it can in any way favour expansion. This force has been suggested as an important factor in the process, but, as far as I am aware, no actual observations on the subject have been made. Through the kindness of my friend Dr Noel Paton, I was enabled during this autumn to make a series of experiments on this subject in the physiological laboratory at Surgeons' Hall. Time will not permit of my entering into the details of these experiments, which I hope to publish. Suffice it to say, that by circulating a mixture of equal parts of defibrinated blood and normal saline solution through the removed lungs of rabbits at a pressure of 12 mm. of mercury—the estimated normal pressure in the rabbit's pulmonary artery—I was able to increase the lungs on an average nearly 30 per cent. of their volume. The figures were very constant, and in two cases where the expansion did not reach 30 per cent., it was readily explained by marked ecchymosis of the lung interfering with free circulation.

From these observations we learn that a collapsed lung is capable of considerable increase in its volume even by the normal blood-pressure in the pulmonary artery, and it is not, I take it, unreasonable to suppose that when air is admitted, and the lung collapses, the blood-pressure will be increased. Here, then, we have alone a considerable force for the expansion, even if an increase of the pressure be denied, and when added to other means, such as expiratory efforts, a most important factor.

We still have to deal with—

[1] *Transactions of Med. Chir. Society, Edin.*, 1886-1887.

(3.) *Movements of the chest-wall during respiration and valvular conditions of the wound*, and inquire if they have anything to do with the point at issue. We know that where there is an opening in the chest-wall air passes in with inspiration and out with expiration, and it has been pointed out by Robertson,[1] that in a few days after the opening has been made, while the edges of the wound are separated during expiration, thus readily permitting a free egress of air, they tend to fall together during inspiration and hinder its entrance. He also draws attention to the probability of this valvular action occurring when, as is usually the case, a drainage-tube has been introduced, and assumes that the protective and dressing, becoming soaked with and saturated by discharge, will act as an efficient valve.

Respiratory movements do good, then, in the first place, when a valvular action at the opening exists, for as more air is forced out by expiration then enters during inspiration, there will be, in proportion to this difference, a degree of negative pressure produced in the pleural cavity, which will, of course, be favourable to and assist in expansion.

The second way in which respiratory movements do good is by tending to draw out the lung, where adhesions sufficiently strong to permit of this have formed between the pulmonary pleura and that lining the chest-wall and diaphragm.

Lastly, we know that granulations become converted into fibrous tissue, which tends to contract. (4.) *Adhesions* form, and this leads us to ask what bearing they have upon this process of expansion. We shall see they have a twofold one.

(α.) If they form before the cavity is opened, and when the lung is compressed by the effusion, they will tend to bind down the lung and prevent its expansion, thus contributing the greatest obstacle to a successful termination.

(β.) Their action in bringing about a cure has had much importance attached to it by some writers. Roser,[2] in 1878, stated that expansion took place in consequence of the contraction of adhesions; and Godlee[3] in 1886 writes, that by their action the "lung is drawn out and made to expand."

We know that adhesions of considerable thickness and strength are present in old-standing and septic cases of empyema, but we seldom have an opportunity of ascertaining their presence in recent cases which have been successfully treated antiseptically.

I am indebted to the kindness of Professor Fraser for being permitted to make some observations upon this point in the Materia Medica Laboratory of the University. The experiments were performed in the summer of 1886.

A pleural effusion was artificially produced in dogs, and treated subsequently by antiseptic incision and drainage, the tube removed at intervals varying from two to four weeks, and the

[1] *Loc. cit.* [2] *Loc. cit.* [3] *Loc. cit.*

animals killed when the opening had been closed for the same periods. At the post-mortem examination the lungs were found in all the cases to be completely expanded, and the pleural surfaces united by recent adhesions throughout their entire extent. These adhesions were not of such strength and thickness as to lead one to suppose they had been capable of exercising any degree of traction upon the lung—in other words, of taking an active part in expansion.

From these experiments and from the fact that in some cases of empyema the lung is found to expand to a certain extent almost immediately after the pus has been let out—that is, before there has been sufficient time for adhesions to form between the pleural surfaces, and by their contraction to pull the lung out. I am inclined to differ from those who regard the contraction of recently-formed connective tissue as one of the principal factors in the process of re-expansion of the lung.

That adhesions are beneficial in assisting in the closure of the cavity I have no doubt. In recent cases I think they play a more or less passive part, merely retaining the pleural surfaces in contact when the lung has been expanded by other means; while in the more chronic cases they may possibly, when of sufficient strength, do a certain amount of good by their contraction, and will be of service by favouring expansion during the respiratory movements of the chest-wall and diaphragm if they be in favourable positions.

Note, then, that the formation of adhesions before pus has been evacuated and expansion begun is an important obstacle to the successful treatment; and, further, that if the expansion takes place unequally, and some parts become adherent to the chest-wall, we are liable to have an encysted empyema; while, on the other hand, they may be of advantage in the way just described.

We have now followed the process by which the lung comes up to the chest-wall, and have considered the various factors which take part in the production of re-expansion, and it remains for us to inquire (*B.*) *how the chest-walls, including the diaphragm and mediastinum, meet the lung?*

First, with regard to the *ribs*. When the cavity is opened and its contents evacuated, these structures fall together in proportion to their elasticity, and thus tend to meet the lung and hasten union between the two pleural surfaces; a considerable amount of deformity often remaining in consequence. In children, however, where the parieties of the thorax are notably elastic, this deformity frequently passes completely away, and we can conceive how by this means, with the pleural surfaces more or less adherent, the lung will be further expanded after the cavity closes.

As the positive pressure diminishes in consequence of increase in the volume of the lung the *diaphragm* rises up, and thus assists in the closure of the cavity; while, as we have already seen,

as soon as adhesions form between its pleural surface and that of the lung, it will, by its contraction, further aid in expansion. The *heart* and *mediastinum* in a like manner, and for the same reason, return gradually to their normal position, and also help to obliterate the cavity.

We thus see that in favourable cases a cure is brought about by a combination of the lung advancing towards the chest-wall and the latter receding towards the former.

Having pointed out to you the manner in which an empyema is cured, we have next to take up the causes of failure; but before doing so let me give a brief *résumé* of how the cavity is closed in favourable cases. This result depends, as I have already stated, not upon one factor alone, but upon several, and is, I take it, as follows:—The lung is expanded intermittingly to a slight extent during normal breathing, markedly so by forced expiration and coughing, and these forces are aided by blood-pressure, which is an important factor; air is forced out at the opening during expiration, and is to some extent prevented entering by the falling together of the edges of the wound, or by the valvular action of the dressings, in consequence of which, and also of the possible absorption of a small quantity of air between the times of dressing, if it be not necessary to do so frequently, a degree of negative pressure is produced in the cavity, which is, of course, also favourable to expansion. As the lung is being expanded by these means, the formation of adhesions between the two pleural surfaces serve to maintain it in contact with the chest-wall, which has, in proportion to its elasticity, been falling in to meet the lung, while the diaphragm, by rising up and becoming adherent, and the mediastinum, by returning to its normal position as expansion progresses, also assist in the closure of the cavity.

We are now in a position to seek for an explanation of *the circumstances which give rise to failure* on the part of the walls of this chronic abscess—empyema—to come together, and here, again, we must look to the lung itself and the chest-wall. I may state, however, that by far the greater number, if not the whole, of the cases of failure are met with in adults; for in children, with their more elastic lungs and less rigid chest-walls, it is exceedingly rare for us to fail in obtaining a satisfactory cure.

1. *As to the lung.*

It may have lost its elasticity in consequence of inflammatory changes in its structure, producing fibroid induration and other conditions which render it wholly or partially incapable of expansion. By far the most common cause, however, although the two may be combined, is the fixing of the lung by dense adhesions and the coating of its surface with a thick layer of lymphy material.

If we examine the thorax in such a case we find the pleura enormously thickened, the lung adherent, and partially or totally

incapable of expansion, lying posteriorly in the hollow formed by the angles of the ribs, and constituting part of the wall of an abscess cavity lined with granulations. This state of matters usually arises from delay in opening, but in patients the subject of tubercle, or who have a tubercular tendency, it may occur at a comparatively early period, and is a strong argument in favour of early opening in such individuals. Then, again, septicity and tension, in consequence of bad drainage, favour a continuance of suppuration, which in its turn conduces to thickening of the pleuræ and hindrance to expansion.

2. *With regard to the chest-wall* one need only remark, that the cause of failure on its part is rigidity, which is, of course, marked in proportion to the age of the patient; and that while the ribs do fall in to a certain extent in all cases, this occurs only to a satisfactory extent in young persons. The diaphragm and mediastinum fail to do good, in consequence of want of expansion of the lung producing a sufficient degree of negative pressure in the pleural cavity to enable the former to rise up or the latter to return to its normal position.

We have now seen how a complete cure is brought about, and the circumstances which give rise to a failure on the part of the cavity to close. There is a class of cases between these two, however, which we very frequently meet, and which, perhaps, most commonly comes under the care of the surgeon. I refer to those cases where expansion of the lung, falling in of the chest-wall, and rising up of the diaphragm, take place to a certain extent; but an obstinate fistulous opening remains, and the patient is exposed to all the risks of prolonged suppuration. This condition is most prone to occur where opening has been delayed, septicity is present, and drainage has been imperfect. We find in such cases the lung unequally expanded throughout, adherent to the chest-wall where expansion has taken place; and where this has failed an abscess cavity exists, or there may be several such abscesses communicating with one another, or with the external wound by narrow orifices. This state of matters is due to portions of the lung being incapable of expansion from changes in its structure, and to thickening of the pleura over it, or to both of these conditions.

Having considered the Method of Cure in Empyema and the causes which give rise to failure, it only remains for us to take up the means at our disposal for bringing about this cure, and for remedying unfavourable results when they arise—in other words, the treatment of this affection.

As the *Treatment of Empyema* would of itself form an adequate subject for a lecture, I can only deal with it in a somewhat general way, for time will not permit of my entering into details.

You will remember that I compared an empyema to a chronic

abscess under special conditions, and we have ascertained what these special conditions are. There is, I take it, only one way in which a chronic abscess, of such a size as the one under consideration, can be treated, namely, by evacuation of its pus. The question now before us is how to evacuate? We may do so in two ways—by aspiration, and by incision with drainage—each of which has its advantages. What, then, is the respective value of these methods in the treatment of empyema, and under what circumstances should they be resorted to? By using the aspirator in the treatment of chronic abscess we evacuate its pus more or less completely, according to its consistence, if proper precautions be taken, reduce the risks of septicity to practically *nil*, and, in a small number of cases, bring about a cure; hence it is that the majority of surgeons give aspiration a trial before resorting to incision, and this applies also to empyema. We know, however, that it is the exception rather than the rule to cure a large chronic abscess, such as a psoas or an empyema, by aspiration; and therefore we look upon it as a method which may be successful, but which, on account of its frequent failure, should not be too long persevered in, for the reasons to be presently stated.

In some cases of recent empyema, and particularly in children, aspiration undoubtedly does good, for we produce a degree of negative pressure in the pleural cavity which, if the lung be not bound down, tends to bring about union between the two pleural surfaces by favouring expansion; we further, by the removal of fluid, diminish tension, which is conducive to pus formation. In order to derive benefit from aspiration, it should be employed at intervals of say three or four days, depending upon the rate of reaccumulation, and thus the operation should be repeated before pus has collected to the same extent as it existed at the previous tapping—in other words, we should aim at always maintaining a negative pressure in the pleural cavity, the advantages of which we have seen. We may expect benefit to accrue from this method of treatment when it is only necessary to tap at longer and longer intervals, and when the pus at each tapping is found to be thinner than at the former one, while it will certainly fail if there be a rapid accumulation of thick pus.

Aspiration should be employed, then, only in the manner indicated, and we should never wait until the chest is full before repeating it; further, unless we find it doing good, we should not have recourse to more than two, or at the most three tappings. In tubercular patients especially, where inflammation tends to be of a low type and we are prone to have thick layers of lymph and granulation tissue rapidly formed, we should guard against trusting too long to this method of treatment, for the longer we delay opening the greater the risk we run of allowing the lung to become bound down by adhesions and incapable of expansion.

I have told you that aspiration will succeed only in a small

number of cases, notably in children, and I am inclined to agree with those who place little or no reliance in this method of treatment for adults. Now, undoubtedly, one of the principal reasons why the use of the aspirator fails, is because we do not obtain complete removal of pus; and as this is most important in order to effect a cure, we accordingly have to adopt the other method of evacuation to attain this end, namely, incision and drainage.

With regard to incision, one may state it must always, at first at all events, be accompanied by the use of a drainage-tube, and that the employment of strict antiseptics is indispensable. Much discussion has arisen over the question of the best site for the opening. It should not be too low, for the rising up of the diaphragm will interfere with drainage; nor too far behind, in order to avoid delay in the expansion of the lung and adhesion of the pleural surfaces from the back.

The fifth intercostal space, about the mid-axillary line, is a situation which I have found a good one, and, as a rule, one opening only is necessary. Having fixed upon the site for incision, how are we to open? Bearing in mind that free drainage is most important, it is advisable to remove a small portion of rib at the time of opening, in order to prevent the tube being pressed upon when the ribs fall together. This should be done by making an incision along the rib itself, stripping off its periosteum, and cutting about an inch out; we then make a small cut through the thoracic wall and dilate the opening with the forefinger, on the withdrawal of which a large-sized drainage-tube, with a shield or a safety-pin passed through it, should be introduced, and the pus allowed to escape under a piece of lint or cotton wool soaked previously in carbolic or corrosive sublimate solution. When the pus begins to flow out slowly, we should dress with protective and several layers of corrosive sublimate wool. It will be found necessary to renew the dressing frequently during the first few days, and probably more than once during the first twenty-four hours.

Such is the method of opening and draining an empyema; and now as regards the after-treatment. Unless the pus be septic before opening, which sometimes occurs on the left side, as pointed out by Dr Russell,[1] or where there is a communication with the lung, we should not wash out the cavity, lest we should destroy recently-formed adhesions. We should only dress when the discharge has soaked through the wool, and when doing so we should make the patient hold his breath, in order to prevent air entering the cavity during inspiration; this is quite practicable, if everything be in readiness before the dressing is removed. The tube should be shortened as the discharge diminishes. The time at which it should be removed is important, for by keeping it in too long much harm may be done in consequence of its irritation causing continuance of suppura-

[1] *Glasgow Medical Journal*, Sept. 1883.

tion. One cannot lay down any definite rules for its removal, but each case will have to be judged according to the special conditions present; and our best guides are the intervals at which dressing is necessary, and the extent to which the lung has expanded. The patient should be directed to lie on the affected side, in order to favour drainage, and further, half-way round on his face, in order that the lung may tend to fall down by gravity toward the ribs; we should instruct him to have frequent recourse to forcible expiration and coughing, to be followed by cautious inspiration; and abdominal breathing will be of use in favouring expansion when the lung has become adherent to the diaphragm.

This method of treating empyema by incision and drainage will in a large number of cases lead to a satisfactory result. We may expect a favourable termination in all cases in children, in all cases in adults which we get before the lung has been rendered incapable of expansion, and which are not allowed to become septic. There can be no doubt that a serious objection to making an incision into the thoracic cavity, and introducing a drainage-tube, is that, by bringing about a further collapse of the lung, we interfere with its expansion, and accordingly various modifications of the method we have just considered have been adopted from time to time. Thus some surgeons have suggested the use of a drainage-tube with valves. These are, however, liable to become blocked with discharge, often necessitating their removal in order to clean them, and hence cannot be considered more efficacious than an ordinary dressing carefully applied in the way I have mentioned, and maintained accurately in position. The method of continuous drainage by means of the syphon action, however, undoubtedly presents many advantages. By this means we produce a constant negative pressure in the pleural cavity, and thus, by causing a continuous suction power on the lung, which can be regulated at will, favour expansion, we further prevent the admission of air, and, in the avoidance of frequent dressing, greatly diminish the risk of septicity.

Dr Robertson, Surgeon to the Oldham Infirmary, has recently published an exhaustive account of what he terms a Method of Subcutaneous Drainage and Irrigation for the Treatment of Empyema, the principle of which is based on that of the syphon. He employs two tubes, which are introduced into the pleural cavity at different levels, one of which is used for drainage, and the other for irrigation. Time will not permit of my entering into the details of his method, and I would refer those of you who may be interested in this subject to his paper, which appeared in the *Medical Chronicle* from March to July of this year. His statistics of thirteen cases, all children, are not more favourable than we are accustomed to look for in these patients when simple incision and drainage are employed; and, before expressing an opinion as to the value of his method, I should like to know something of the results obtained by its application to adults.

Mr Duncan[1] has also employed a method of syphon exhaustion. The apparatus consists of a large flat piece of rubber, a long rubber tube, and an air-tight, pliable bag. The cavity is filled with warm boric lotion, and the tube, also filled, is inserted into the cavity through a hole in the rubber, which is rendered air-tight at this spot; an elastic bandage round the body keeps the rubber in position. The air-tight bag is filled, and by rolling it upon itself rendered half-empty again, and while the bag is brimming over, the tube, passed through its screw stopper, is inserted into it and screwed home before the pressure is released. The advantage of this method is that the patient can walk about with the bag in his pocket. In the case in which it was tried it undoubtedly did good. In one month the cavity diminished from a capacity of 30 ounces to 7 ounces when the bag was raised, 5 ounces when held on a level with the wound, and 2½ when the bag was lowered, and ultimately to ½ ounce. I saw the man in September, and a small cavity still remained. The case was, however, already chronic when this method of treatment was begun.

Theoretically, the syphon action would appear to be our best means for treatment, but there are many difficulties in the way of its application. I doubt the feasibility of applying it to the extent of causing any marked degree of suction in recent cases, in consequence of the pain and discomfort it would produce; and in a chronic case in which I tried it recently it had to be abandoned on that account.

I am afraid we must return once more to the chronic abscess before I conclude. We know that in this condition persistent suppuration sometimes occurs, and a cure cannot be brought about by simple drainage and incision, and we have to resort to other means; the removal of the cause where that is possible. There are cases of empyema in which, for the reasons already given, the lung cannot come up to the chest-wall, or the latter fall in sufficiently to cause obliteration of the cavity. We have an abscess cavity throwing off large quantities of pus, most probably septic. What are we to do under these circumstances? There are two courses open to us—namely, to leave the patient alone, and exposed to all the risks of prolonged suppuration, such as pyæmia and amyloid degeneration of his organs; or to endeavour to close the cavity.

When the question is thus put to us, there can be no doubt that the latter is our proper line of treatment; in other words, the lung having failed to expand, and being incapable of coming up to the chest-wall, and the cavity in consequence remaining unobliterated, we must make the chest-wall fall in to meet the lung. This can only be produced by the excision of ribs known as Estlander's operation; and I would impress upon you that, before we adopt this line of treatment, we must be convinced that a cure can take place

[1] *Edin. Med. Journal*, July 1888.

in no other way; for it implies that all other methods have been given a fair trial and failed; and such being the case, our operation must be thoroughly done, and not less than six, and probably eight ribs, according to the size of the cavity, should be resected.

Gentlemen, I have now finished. There are many points upon which I would fain have dwelt at greater length, and others which it has been necessary to pass over completely, in consquence of the time at our disposal being so short. If what I have told you concerning the "*method of cure in empyema*" has impressed upon you the object I had in view when I began this lecture, namely, the importance of pursuing the study of Surgery from the broad and scientific point of view to which I have already referred, I shall feel that this hour has not been misspent.

I may have appeared to have underestimated the importance of the practical part of our work, but my remarks apply with equal force to this also. Let me give one out of many illustrations bearing upon this point. You must not regard this or that splint as *the* one to be applied to this or that fracture, but, given a broken bone, you should consider what is the best means for treating such an injury, the principles (and here a knowledge of Anatomy and Physics comes in) which guide you in the selection of a particular form of retentive apparatus, and the results you desire to obtain by its adoption. We are here to study Systematic and not Clinical Surgery, but I would have you bear in mind that, when you come in contact with cases of surgical disease and injury, you are not to expect them to fit in accurately as regards their symptoms, diagnosis, or treatment with the description given in your text-books. It will be then, if you have adopted the method of work I would venture to recommend, that you will understand the value of having studied Surgery from a wide and general point of view, for your diagnosis will be scientific, not empirical, and your treatment rational, not routine. Besides, from an examination point of view, which now-a-days pervades the whole work of the student's career, you will find that by cultivating your reasoning and thinking powers, by not taking for granted statements which occur in your text-books; but, by bringing an all-round knowledge to bear upon them, by working them out for yourselves and forming your own estimation of their value—in short, by avoiding cramming, you will not only make the best possible preparation for those necessary evils, examinations, but lay a secure and permanent foundation for your life's work.