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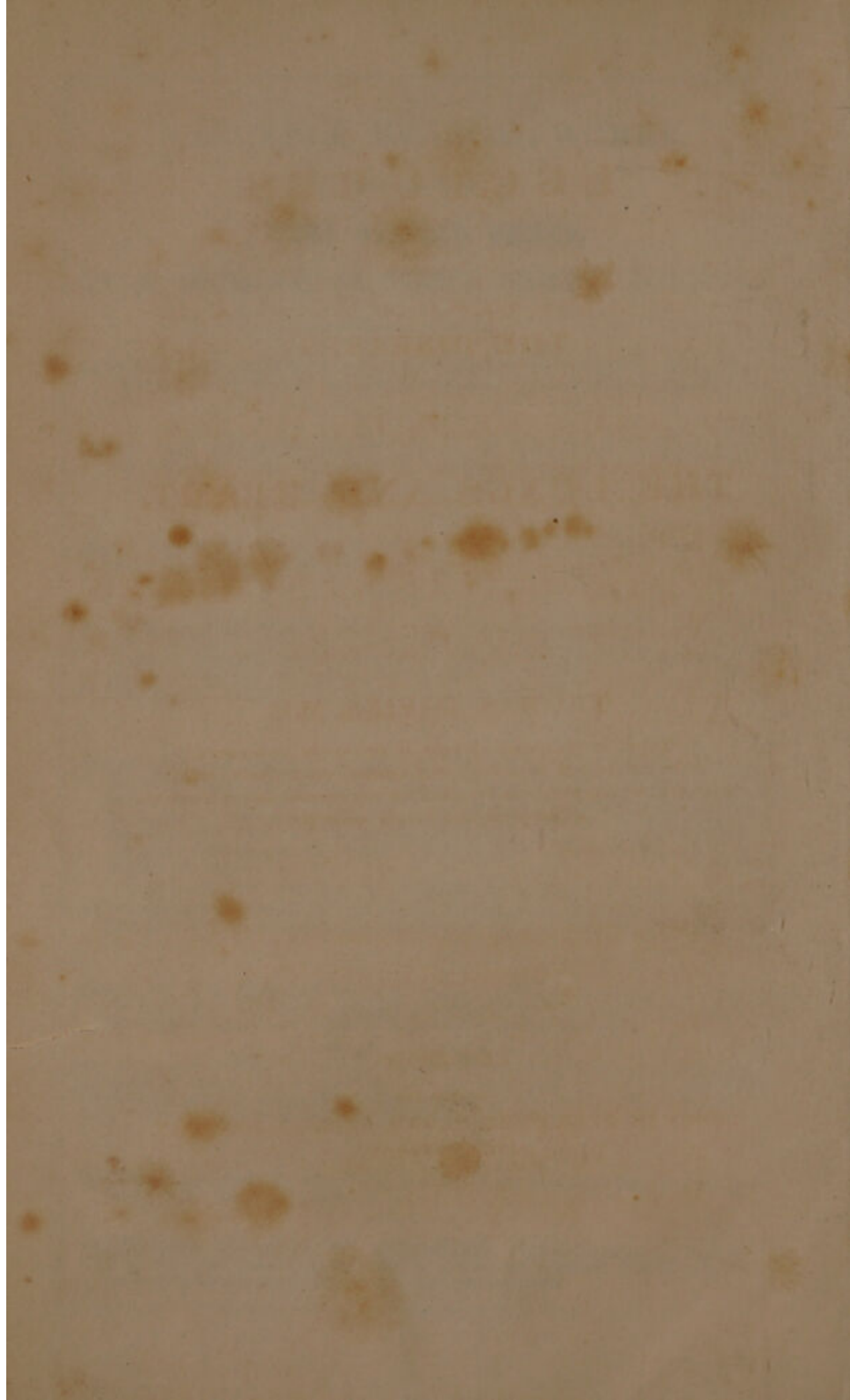
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THE LUNGS AND HEART

THOMAS DOUGLAS, M.D.

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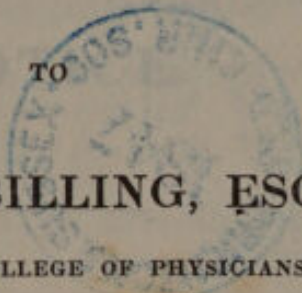
LECTURES

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THREE VOLUMES

AND INDEXES

A TESTIMONIAL OF THE GREAT RESPECT

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THE following Lectures were originally delivered to a few medical practitioners, and they subsequently became a part of a general course on the Theory and Practice of Medicine, given at the London Hospital: they then were published in the *Medical Gazette*, and are now offered to the Public in the present form.

The Author feels that he may be accused of presumption in supposing a work on this subject necessary, after the publication of Dr. Forbes's admirable translation of the *Traité de l'Auscultation Médiate*, and of Dr. Hope's classical monograph upon the Diseases of the Heart. He believes, however,

that the subject is not yet exhausted, and will be perfectly satisfied if his professional brethren think that he has added his contribution, however slight, towards the extension of the knowledge of such important subjects as Percussion and Auscultation, as applied to the investigation of the diseases of the Thoracic Organs.

30. *New Broad Street, June, 1835.*

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ERRATA.

- Page 45. line 11. from bottom, for "cases" read "causes."
135. l. 17. for "seised" read "seized."
144. l. 5. and 145. l. 20. for "Dipthlerite" read "Diphterite."
353. l. 5. for "rapture" read "rupture."
410. l. 13. for "engagement" read "engorgement."
413. l. 17. for "ventrical" read "ventricle."
470. l. 8. from bottom, for "affect" read "affects."
474. l. 8. for "membrane" read "aneurism."

REVIEWS

1. The book is a very good one, and it is well written.
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LECTURES
ON
DISEASES OF THE CHEST.

LECTURE I.

DISEASES OF THE CHEST.

Importance of Subject—Frequency of Pulmonary Diseases—Complication—Percussion and Auscultation—General Causes of Pulmonary and Cardiac Diseases—Historical Lecture—Arrangement of Subject.

Diseases of the Lungs—Theory of Signs—Functional, local, and general Signs—Examples.

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THERE are few subjects connected with the study of medicine, of greater importance than the diseases of the chest; they derive that importance from a variety of circumstances, some of which I shall detail.

In the first place, there are certainly no diseases

of such frequency ; for I may truly say, that there scarcely exists the individual in this climate who has not, at one period or other of his life, been subject to some form of pulmonary disorder : nay, I may even say still more ; there perhaps scarcely exists the individual in these latitudes who is not even *annually*, more or less, affected in this way.

Neither is there any class of affections which produces so great a positive mortality. I say *positive*, not *comparative* ; because the diseases of the organs contained in the chest are not more fatal than those of the brain, or the abdominal viscera, but it results from their extreme frequency. It is true that the data which we possess, by which we judge of this, are insufficient, as medical statistics have been, unfortunately, too little cultivated in this country ; but those which we do possess, err, I am quite satisfied, on the side of omission rather than of exaggeration. From the London bills of mortality, we see that more than one third of the annual mortality of this metropolis depends upon pulmonary disorder : and if we take into consideration the number of cases of dropsy, and the many cases of apoplexy, depending upon diseases of the heart, I am quite sure we should come nearer to the mark were we to say that one half of the mortality depends upon affections of the pulmonary or cardiac systems.

In the next place, their complication with other diseases is a subject of great importance ; for you scarcely find any general disease, especially of a febrile kind, in which the lungs and heart do not more or less participate. So sure as the skin becomes hot or cold, the mucous surfaces of the tra-

chea and bronchi will sympathise with it. You will find no case of fever in which there are not some signs of pulmonary lesion. Chronic diseases occasioning great debility, often predispose to phthisis; and I need not say how the motions of the heart are accelerated in fevers, or are influenced by many other affections.

But to us, as medical men, there is another important reason why we should study these diseases. The signs by which we have hitherto judged of them, have been, as I shall show you, extremely equivocal. I say *equivocal*, because one sign may be common to various conditions of the lungs and heart. In the middle of the last century a discovery was made by Avenbrugger, by which these diseases could be more specially determined; and the means of doing so were enlarged by the still more brilliant discoveries of Laennec. He, or rather I may say, they, have rendered the diagnosis of the diseases of the heart and lungs almost as clear, almost as distinct, as of the external or surgical diseases of the body.

When you reflect, therefore, upon the extreme frequency of pulmonary disorders — of the mortality they occasion — of their complication with other diseases — and, finally, of the extreme importance of the new modes of investigating them — I need say no more to stimulate you to the highest exertion in prosecuting the study of this subject.

It might not be, perhaps, uninteresting at this moment, to cast a general view over the causes of this extreme frequency of thoracic disease. I do not mean to enter here into a minute detail, because I must do that when I arrive at each parti-

cular subject. In the first place, in the lungs you have a most delicate organisation, consisting of an immense number of cells, extremely thin, so that from their mere texture it is not a matter of surprise that these organs should become frequently affected. But when you superadd, that that texture is in constant movement — a movement which commences at the moment of birth, and only terminates in an existence of longer or shorter duration — a movement proceeding at the average rate of seventeen or eighteen respirations in a minute, and each of which, you will recollect, is not a single, but a double action, the one of inspiration, the other of expiration, so that you have no less than thirty-four or thirty-six movements going on in that time; — I say, therefore, that the frequency of these motions in so delicate a texture, must strongly predispose these organs to disease. Consider, also, the nature of the functions which the lung has to perform; on the one hand, receiving blood, varying frequently in quantity, varying in the force with which it is received from the heart, and varying also, doubtless, in its quality; and on the other hand, receiving atmospheric air, varying also in quantity, quality, and, above all, in temperature; — taking all these circumstances together, you see the numerous causes there are to account for the frequency of diseases of the lungs.

The heart, again, is an organ delicate in its texture, considering the function it has to perform. It has constantly to overcome a resistance, because its office is to propel forward a fluid; this office commencing at the earliest period of embryotic existence, and terminating only in a life of varied

duration. Too slow or too frequent a pulsation may equally be a cause of derangement.

We see that the average rapidity of the heart's contractions is seventy or eighty in a minute, and that each ventricular beat is followed by a ventricular dilatation, which I think I can prove hereafter to be effected by an active expansion; so that the movements are in reality double the number of the arterial pulsations, or 140 or 160 in that time. We see also that these motions are influenced by a variety of circumstances, as general disease, labour, exercise, emotions of the mind, &c.; some of the passions accelerating the heart's actions to an extraordinary rapidity; others, as profound grief, depressing them almost to cessation. Considering all these circumstances, it is a matter of surprise that this organ is not more frequently affected.

It might be expected, perhaps, that I should enter here into an historical account of the rise and progress of our knowledge of pulmonary and cardiac diseases. Were I to do so, I must suppose that all the gentlemen whom I have the honour to address are acquainted with the subject. I know that many of you are; but it is my business to suppose that I am addressing those who are perfectly ignorant of it; and were I to give a history, I must enter into details, and employ technical expressions, which I must assume you do not understand. I shall give as much of the literature as will be necessary to elucidate the subject, when I come to each particular part.

I now proceed to lay down the plan which I intend to follow in the course. I propose to

treat of diseases of the lungs, and diseases of the heart. The course, then, divides itself into two sections: — first, diseases of the lungs, which will comprise about twenty lectures; and, secondly, diseases of the heart, which will occupy about six or seven.

We commence with

DISEASES OF THE LUNGS.

This portion of the course may be divided into two parts: — first, the *Theory of the Signs* of pulmonary affections; secondly, the *Diseases* themselves; the latter section admitting the anatomical arrangement of diseases of the air passages, diseases of the parenchymatous substance, and diseases of the pleura.

THEORY OF THE SIGNS OF DISEASE OF THE LUNGS.

I shall now proceed to the consideration of the theory of the signs of the diseases of the lungs; and it may be asked, and very naturally, why I prefer commencing with this subject, rather than with the diseases themselves. It is because there are many generalities which can be made in relation to these signs, particularly on the subjects of dyspnœa, percussion, and auscultation, easy to be understood at once, but which involve many principles and technical expressions, which would constantly embarrass us were we to defer their explanation until we arrived at their application to each particular disease. Experience has shown this to me as the best plan, and I think you will soon discover this as we proceed.

Let us now endeavour to arrange the signs : I think the following order the best, as the lines of demarcation are tolerably distinct ; and it applies as well to the rest of the diseases of the body as to those of the lungs.

First, *functional signs*. By a functional sign I mean a sign which arises from a disordered condition of the function of the organ.—Secondly, *local signs*. A local sign is that which is detected by a local examination of the organ.—Thirdly, *general signs*. By these we mean, when two or more of the rest of the functions of the body are deranged in consequence of the disturbance of a particular organ. This arrangement, as I have just said, applies to all the diseases of the body ; but, as examples are infinitely better than mere definitions, I will give you a few to show what is meant.

A functional sign is a sign arising from the lesion of a function. For instance, I will suppose the eye to be inflamed. What is the first thing you observe ? Why, that vision is impaired. There is a change here in the function of the organ, and that would be a functional sign. I examine the eye ; I see, perhaps, redness of the tunica conjunctiva. This is a local examination of the organ ; and the redness of the conjunctiva is a local sign. Suppose, in consequence of this disease, febrile symptoms arise ; the pulse becomes excited, beating quickly ; the head affected, and the skin heated. Here are several functions of the body disturbed, in consequence of the derangement of a particular organ. These are the general signs. I will suppose a case of a common sore

throat — cynanche tonsillaris. The first sign is a difficulty of swallowing ; the function is disturbed, and that difficulty would be a functional sign. I examine the throat ; I see the tonsil swollen and red : this swelling and redness are local signs. General febrile action would be set up under such circumstances, or the rest of the functions of the body would be disturbed, in consequence of the lesion of the tonsils ; and these constitute the general signs. To exemplify it by a disease of the chest : suppose a case of peripneumonia, or inflammation of the substance of the lungs. The first thing I observe is, a difficulty of breathing, *dyspnœa* ; that is a functional sign. By percussion and the stethoscope I examine the chest, and I find certain sounds, and these would be local signs ; and the general febrile action would be the general signs.

I shall not fatigue your attention with more examples otherwise I might go through the whole of pathology, and give instances throughout of the general application of this simple division.

We will now commence with the theory of the signs of the diseases of the chest, by describing the

THEORY OF DYSPNŒA.

Dyspnœa is the first great functional sign of disease of the chest : to this I must request your especial attention.

By dyspnœa we mean an aberration from the normal condition of respiration ; or, to use language less scientific, simply, difficulty of breathing ; but I would rather extend the term dyspnœa. It is

generally used to express mere rapidity of breathing; but I would say that *any* aberration from the usual condition of respiration constituted dyspnœa: if the breathing were too slow, that would be to me dyspnœa, or difficulty of breathing.

Having given you this definition, I shall next attempt to establish a theory, by announcing the following proposition, and then I shall endeavour to prove it; which is, that dyspnœa is nothing more or less than an effort of nature to establish an equilibrium between the quantity of the air and the quantity of blood in the lungs.

Before I proceed to establish this proposition, I must, in the first place, recall your attention to some of the leading points connected with the anatomy and physiology of the lungs and heart. The lungs are to be considered as a double organ, one placed upon each side of the chest, connected together by means of a tube called the *trachea*, which splits at its inferior extremity into two branches, denominated *bronchi*, one of which goes to each lung, and each has for its function the carrying of air into that organ. These bronchi ramify, and terminate in capillary extremities in the vesicles or air-cells of the lungs. Between the lungs is placed the heart: it is situated in the lower part of the cavity of the chest, and inclining obliquely to the left side. The heart is of a conical form, having its base uppermost, and its apex below. This viscus is divided into two parts, or a right and left side, by a central septum: it is also divided into two parts, according to its transverse diameter, the upper and lower; the upper portion being called the *auricles*, the lower

part the *ventricles* ; and the terms right and left being applied to these cavities, according to their relation to the perpendicular septum.

Now, what is the use of this organ ?—The heart may be considered as a vessel divided into four parts or cavities, for the purpose of receiving the blood from all parts of the body, and for propelling that blood onwards. Let us trace the blood in its course.

The arterial blood, of a vermilion red colour, and fitted for the purposes of secretion and nutrition, is carried from the lungs by the four pulmonary veins into the left auricle, and thence into the corresponding ventricle, from which it passes into the aorta, and all its branches, to their capillary terminations; and, by the beautiful mechanical arrangement of the mitral and semi-lunar aortic valves, it is prevented from retrograding in its course in the heart. The arterial blood deposits its secretions, and adds nutriment to the living fibre at the extremity of the aortic system of vessels, and is now diminished in quantity in proportion to those deposits, and changed in other qualities: it becomes of a darker red hue, and is called venous; is unfitted for the purposes of secretion (excepting of the bile), and of nutrition, and requires renovation.

This renovation is effected in its transit back again to the heart, by the chyle and lymph being poured into the venous system, and by the blood being carried afterwards into the lungs, to be there exposed to the oxygen of the atmospheric air. By the united influence of these causes it is restored to its arterial qualities, and is again fitted for its

important functions. But how is it carried to the lungs?

From the arterial capillaries arises another system of vessels, equally minute, which unite in larger and larger tubes as they approach the heart, until they finally terminate in the right auricle by two large vessels, which are denominated *venæ cavæ*. These vessels are called veins; the blood within them flows more tardily than in the arteries; and, as if the force which moved the venous blood were insufficient to carry it to the lungs and round the pulmonary circulation, the right side of the heart seems placed intermediately to receive it, and to give it sufficient impetus to be transmitted to its ultimate destination.

The blood, then, of the *cavæ* is carried into the right auricle, ventricle, and pulmonary artery, to the minutest ramification of the latter vessel, and from thence to the system of pulmonary veins. A similar arrangement of valves exists in the right side as well as the left, under the names of tricuspid and pulmonary semi-lunar valves, and for a precisely similar purpose, — to prevent the retrogression of the blood.

You thus see here one pulmonary artery to carry the blood into the lungs, and one tube or trachea to carry air; but the venous blood, when changed, passes by a different set of vessels, the pulmonary veins, while the air returns by the same vessel, necessitating a double act in the latter, — the one of inspiration, the other of expiration.

Now, to examine the subject still farther, we must inquire, What are the elements essentially necessary to the healthy condition of the circu-

lation and respiration? There are two; there is the blood on the one hand, and the air on the other; there is blood to be changed, and there is air to be carried down to change it.

With regard to the blood, every time that the right ventricle propels it into the lung it must be effected in due *quantity*. I cannot state the quantity, for it must be various in different individuals, and at different ages; but there must be a due quantity in relation to the quantity of air; and therefore we will note down as our first element, *quantity*. In the next place, the blood must certainly be conveyed with due *force*. If the force be too feeble, it is very evident that it will not propel the blood sufficiently onwards; if it be too strong, the contrary will be the effect; and therefore *force* must essentially be an element here. In the third place, there must certainly be a given *time* for each pulsation. The heart may beat thirty or forty times in a minute, — that is too slow; or it may beat 150 times in a minute, — that is too quick; and therefore *frequency* of pulsation must be considered as an element. In the next place, although this is a subject which we do not yet well understand, there can be no doubt the blood shall be of a certain *quality*. And, finally, there must be a *vessel of due calibre* to convey the blood. *Capacity* of vessel may therefore be considered a necessary element.

These, then, are the necessary elements of the healthy circulation of the blood, — due quantity, a given force, a due relation of time, a given quality, and a proper capacity of the vessels.

Then, with regard to the air, you will find the

elements here exactly the same as in the case of the blood. There must be a due *quantity* of air to decarbonise a due quantity of blood. *Quantity*, then, will be an element.

We may give an instance of this. In breathing upon a plain, we will say at the level of the sea, where the atmosphere is of the greatest density, from receiving the greatest possible pressure, we will suppose that a cubic inch of air contains a given quantity of oxygen: but ascend a mountain; the pressure of the atmosphere becomes less, and consequently there is less oxygen in the same space: the breathing then becomes quick; and therefore it is evident that quantity forms an element. Then the respiratory acts must be effected with a certain *force*, otherwise the lung would receive too great or too small a quantity of air. There must also be a given *time*. The ordinary time is about eighteen respirations in a minute; but, suppose there be only one half that number, the patient will experience great difficulty. Try it yourselves. Reduce the respiration voluntarily to four or five times in a minute, and you will soon feel great distress; from which it is evident that time is an essential element. The *quality* of the air, no doubt, is another element. I am not speaking of poisonous gases, such as sulphuretted hydrogen; but of those which are noxious only from the absence of oxygen, such as carbonic acid and azote. Finally, there must be a due *capacity* of the trachea and the lungs, to admit of sufficient air to oxygenise the quantity of blood sent by the pulmonary artery.

Now, let this be understood, and what we are

about to say becomes exceedingly easy of comprehension. There must always be a due relation of these elements to each other; for if there be not, you will have dyspnœa as the result. If the quantity of blood in the lungs be increased, there will be an attempt to increase the quantity of air; and that can only be effected by accelerating the respiratory acts; and therefore I repeat, that dyspnœa is an effort of nature to establish an equilibrium between the quantity of air and the quantity of blood. But I will state a number of examples, which will incontestibly prove the truth of my proposition. They shall first be in relation to the heart, and next, to the lungs.

You know that, if a person runs, he soon gets out of breath, or into a state of dyspnœa. What is the cause of this? The venous blood is now precipitated with greater rapidity to the right side of the heart, and from thence to the pulmonary artery and lungs, so that the force and velocity of the circulation is increased: by consequence, the quantity of blood in the lungs is also increased in a given time, so that we have here the elements of force, velocity, and consequent quantity, in excess. What results? The respiratory acts become also accelerated; they are effected with greater force and rapidity, and more air is received into the lungs, in a shorter time. Nature here, then, endeavours to create a due relation between the force and velocity of the movements of the two organs, for the purpose of establishing an equilibrium between the quantity of the blood and the quantity of the air.

Let us now suppose a case of diseased heart —

that the caliber of the aortic orifice be diminished, either by congenital error of formation, or by diseased valves. The blood arrives in due quantity to the left ventricle, but the aorta, or discharging tube, is of too small a *capacity* to allow it to freely pass : what takes place ? The blood must accumulate in a direction posterior to the obstruction, and retrograde to the current of the circulation. Thus, then, the left ventricle and auricle become habitually too full, and finally, as we shall hereafter describe, diseased in their parietes : the pulmonary veins to their capillary extremities become also gorged with blood, thus opposing obstruction to the current flowing in the pulmonary artery and the right side of the heart. The result of all this is, that the blood exists in the lungs always in too great *quantity* — the original cause being the obstruction from the diminished caliber of the aortic orifice. Observe persons affected in this way ; they are in a state of habitual dyspnœa ; their respiration is constantly accelerated ; they are always endeavouring to increase the quantity of air, to establish a relation with the quantity of the blood. Increase the force and velocity of their circulation, by making them ascend an inclined surface : although but a small additional quantity of blood be sent to their lungs, yet their breathing becomes quickened to a sense of suffocation. They are obliged to stop ; nature here makes a great effort to restore the balance.

We now proceed to the lungs, to exemplify the same principles ; and we will suppose that the circulation is perfectly healthy. You know that one of the most formidable diseases we have to treat,

though fortunately not a very frequent one, is inflammation of the rima glottidis, or opening of the larynx. The rima glottidis is but a narrow orifice; but the result of inflammation there is to contract it very considerably, and the *capacity* of the air tube is diminished at its very commencement, and consequently the diameter of the column of air inspired; so that the quantity of air is insufficient for the purposes of respiration at the usual rapidity of seventeen or eighteen movements in a minute. Dyspnœa arises; the respirations increase to thirty, forty, or fifty, in the same time. We endeavour here to establish an equilibrium in quantity, by multiplying the inspirations in proportion to the narrowness of the column of air. Is not this, then, an effort of nature to establish an equilibrium between the quantity of air and of blood in the lungs?

Croup is an inflammation of the tracheal mucous membrane, having for its result a secretion which concretes, and forms an inner mould for the larynx and trachea, or a tube within a tube. The column of air must here be diminished in diameter, and is insufficient for the purposes of respiration. It is renewed then with a doubled or tripled velocity;—in other words, there is dyspnœa, and for the same reasons I have already mentioned.

I will suppose a case of peripneumonia arrived at the second stage, or hepatisation, and that it affects half the lung on one side; the *capacity* of the whole of the lungs would be diminished one quarter; or if it affect the whole of the lung on one side, then the diminution of capacity would be one half. Under such circumstances, the ra-

pidity and force of the respirations would be proportionately increased ; so that, here, the elements rapidity and force are in excess, in consequence of diminished *capacity*.

Any circumstance preventing the free expansion of the lungs, may be said to act by diminishing their capacity ; thus, fluid in the cavities of the pleura, or in the pericardium, aneurisms of the thoracic aorta, enlargements of the heart, tumours in the chest, all prevent these organs from expanding, more particularly in the transverse or antero-posterior directions ; whilst abdominal tumours, gestation, dropsies, &c., prevent the free descent of the diaphragm, and consequently the increase of the perpendicular diameter of the thoracic cavity.

I need hardly say, that the *quality* of the air influences the respiration. Confine an animal in a vessel in which the air cannot be renewed ; and in proportion to the diminution of the oxygen dyspnœa comes on — that is, it increases the frequency and force of its respirations in proportion to the decrease of that gas.

I might, at the expense of fatiguing your attention, easily increase the number of examples ; but I hope these are sufficient to prove the proposition I originally advanced, that dyspnœa is an effort of nature to establish an equilibrium between the quantity of the air and of the blood in the lungs.

Are there any exceptions to this theory ? There are apparently two ; but I think they are only *apparent*, and not real. In some instances, particularly in low fevers, you will find that the pulse

is very rapid, but that it is small, and there will be no dyspnœa. You have two elements here, rapidity and quantity, in fault ; but if you have the quantity diminished at each stroke of the heart, as the smallness of the pulse would indicate, the increased rapidity may make up for the difference of the quantity which should be sent into the lungs each time ; and therefore there is a compensation, and the real quantity is not greater than it should be, and consequently there is no necessity for a greater quantity of air than usual, and there is no dyspnœa.

The next objection would be, that in some cases the pulse is full and quick, and yet there is no dyspnœa : you see this particularly in cases of inflammatory fever ; but observe the blood as it flows from a vein ; it appears half-arterialised ; it is much redder than usual, especially if the febrile action be high. What has occurred in this case ? the blood seems to be already, to a certain extent, decarbonised. The quality is changed ; it does not require so much oxygenation. I only offer this as an hypothesis ; but I believe that in consequence of the high degree of the oxygenation of the venous blood, it requires less decarbonisation when it arrives at the lungs, and therefore the respiration is not increased in frequency.

What is the value of the sign of dyspnœa ? In one sense it has no value ; in another it is of the highest importance. Dyspnœa never determines the nature of the lesion ; it does not determine whether the lungs be affected, or the heart, for you see it is common to them both ; it is common to every disease of the lungs, and to a

certain extent to every disease of the heart; and therefore it is an *equivocal* sign; nay, it even occurs in diseases that are placed out of the cavity of the chest — in dropsy, and even in pregnancy, and therefore I say it is of no value in determining a specific lesion. But it is of the highest importance in a practical point of view, as expressing the degree of the loss of equilibrium, and therefore gives the most valuable indications. Such, then, is the general theory, as I apprehend, of the sign dyspnœa.

LECTURE II.

THEORY OF THE SIGNS OF DISEASES OF THE LUNGS — (*continued*).

MECHANISM OF THE CHEST.

Objects to afford Protection and Movement — Position and Form of Chest — Structure — Sternum — Ribs — External Surface of Chest — Internal Surface — Superior and inferior Circumference. — Functions of Chest — *Protection* — Posteriorly, laterally, anteriorly — Resistance — Yielding — Illustration. — *Movements* — Diameters — Perpendicular — Antero-posterior — Transverse — Their Measurements — Mechanism of ordinary Inspiration by the Diaphragm — Of extraordinary Inspiration — Increase of all the thoracic Diameters — Least Muscular Power where the Ribs are most movable; most, where they are the least — *Varieties* of Dyspnœa, abdominal, thoracic, complete, incomplete, equal or unequal, frequent or slow, high and sublime — Orthopnœa. — Mechanism of Expiration — Natural — Forced. — Cough — Its Causes — Contraction of the Thoracic Diameters — and of the Rima Glottidis. — Varieties.

WE now proceed to the consideration of a subject immediately connected with that which we discussed in the last lecture; namely, the examination of the structure by which the important organs of the lungs and the heart are protected, and of the mechanism by which that structure adapts itself to the constantly varying capacities of the lungs.

When we reflect upon the great importance of the functions performed by the lungs and the heart, so important that their cessation for a very short period would be productive of death, we must naturally suppose that there should be some protecting envelope or case for them. When we recollect,

also, that the lungs particularly are constantly varying in their capacity; that at one moment they enlarge, that at the next they diminish in performing the acts of inspiration and expiration, we see that it is necessary that the case which protects them, should also have the power of dilating and contracting to follow these movements. We find, therefore, that the double function of protection and mobility is beautifully effected by an organisation of bones, muscles, and cartilages, known under the appellation of *the chest*.

Before we proceed to explain the functions which the chest performs, we must first take a glance at its structure. The gentlemen around me must be generally acquainted with that structure, but still it is requisite that we should take a general view of the subject.

Position and form of the chest. — The chest is placed at the upper part of the trunk, between the cavities of the cranium and abdomen. Its form is that of a cone, whose apex is above, and truncated, and whose base is below, large and open; at least it should be so in a well-formed subject: if, however, you were to examine the chest in a living individual, you would form a very false notion of its shape. It would then appear that the base of the cone was above, and the apex below. This arises from the length of the clavicles, and from the shoulders, which give a breadth at the upper part, and from the narrowness of the waist below: in some persons, particularly in females who have acquired the unfortunate habit of binding the waist too much, this is still more apparent.

Structure of the chest. — We will now examine the structure of the chest. We have it formed anteriorly of the sternum and costal cartilages; posteriorly, of the vertebral column, particularly of the bodies of the vertebræ; and laterally of the ribs.

Sternum. — The sternum consists in early life of five bones, and on its anterior surface there are four transverse lines indicating their former separation. Anteriorly, it is slightly convex, and covered merely by the aponeurotic expansion of the sterno-mastoid, and pectoral muscles, and by the skin; posteriorly, the bone is a little concave. At the superior portion there are articulating surfaces for the clavicles, and also for the first ribs. The inferior portion is terminated by a point, called the ensiform or xiphoid cartilage. There are, laterally, seven depressions for the seven corresponding ribs.

Ribs. — The ribs are twelve in number on each side: the seven superior are called thoracic or true ribs, and are articulated by the intermedium of cartilage directly to the sternum; the five inferior are termed false or abdominal ribs, each being articulated anteriorly to the one above it by cartilage, and thus indirectly with the sternum. The two last ribs have merely cartilaginous tips, and are called the loose or floating ribs. The ribs are flattened in front, and gradually become more rounded behind. Their length is various; the first is the shortest; the second is nearly double the length of the first: they progressively increase to the eighth, and then gradually diminish until the last. Their breadth also varies: the first may, perhaps, be considered the broadest, the second is some-

what less, and we find a gradual diminution as we descend.

Direction of ribs. — The next point to which I shall call your attention, is the direction of the ribs, and this is a matter of considerable importance in relation to their movements. The first rib arises nearly at a right angle with the vertebral column, and the acuteness of the angle increases as we descend from the first, so that each rib being inclined outwards and downwards increases also in the obliquity of its course, and the result of the particular direction is, to give to the whole lateral parts of the chest a general inclination downwards and forwards. Each of the ribs taken separately presents a twisted form, the point of torsion being at its angle. Anatomists describe each rib as having a posterior or vertebral head, a cartilaginous end, and a body. The head of the bone is articulated with the sides of the bodies of the two corresponding vertebræ, with the exception of the first, eleventh, and twelfth, which are each connected to a single vertebra. The head of the rib is supported by a short neck. External to the neck, there is a process called the tubercle, which has an articulating surface, and is connected with the transverse process of the vertebra below it. This is the case with all the ribs, except the first and the two last. The bone then becomes somewhat flattened, and is bounded by an angular projection, which is removed farther and farther from the tubercle as we descend towards the last rib: this is called the angle, and is, as I have already said, the point of torsion or twisting of the bone. The sternal extremity is hollowed out and thin, for

the insertion of the cartilage. The external surfaces of the ribs are convex, and covered by muscles and skin; their internal surfaces are concave, and lined by the pleura. They have a superior and inferior edge. Observe the grooves in the inferior edges for the intercostal arteries, arteries which might possibly, but not probably, be wounded in the operation of paracentesis thoracis.

Generalities; external surface of the chest. — Having considered the chest in its separate bones, let us now examine it generally. In the first place, it has an external and an internal surface; a superior and an inferior circumference. The external surface may be considered in four points of view, in its posterior, anterior, and two lateral regions. In the posterior region, you see the spinous processes of the dorsal vertebræ, and the gutter on each side of them; the transverse processes of the vertebræ; the tubercles of the ribs; the spaces between the tubercles and angles, enlarging as we descend, and the angles forming an oblique line from above downwards and outwards. On examining the chest anteriorly, we first notice the sternum in its centre, and on each side there are the cartilages of the ribs. These cartilages gradually enlarge as we descend, and form an oblique line from above downwards and outwards. The lateral regions are convex, and formed by the bodies of the ribs.

Internal surface of the chest. — Internally, the chest is to be considered in relation to the same positions as externally; that is, the posterior, anterior, and the two lateral regions. Posteriorly, we have the convex surface of the bodies of the dorsal vertebræ. Observe that the dorsal vertebræ

decrease in size from the first to the fourth, by which the antero-posterior diameter is there so much increased: the vertebræ then gradually enlarge to the twelfth. In this region, too, you perceive the articulation of the heads of the ribs. Anteriorly, there is the posterior surface of the sternum corresponding to the anterior mediastinum and the cartilages of the ribs. The lateral region presents a concave surface, covered by the pleura.

Superior and inferior circumference of the chest.

— The superior circumference is formed by the sternum, by the first rib on each side, with its cartilage, and by the anterior part of the body of the first dorsal vertebræ. The inferior circumference is bounded by the last dorsal vertebra, by the last or floating ribs, by the successive edges of the cartilages of all the false ribs, and by the xiphoid cartilage.

Such is a general view of the anatomy of the bones of the chest.

FUNCTIONS OF THE CHEST.

Protection.—We now proceed to examine the functions of the chest, and the first to which we must advert is the protection it affords to the thoracic viscera. Let us consider this subject in relation to the regions we have just described. We will suppose the case of a force being directed against each of these parts. Posteriorly, great protection is afforded to the chest by the spinous processes and bodies of the vertebræ, and also by the large mass of muscles of the back. Two modes of protection are afforded by the upper part of the lateral regions, one only by the lower: thus, the seven

superior ribs, arched, and consequently of the best form to afford a resistance, have each two abutments, the one vertebral, and the other, by the intermedium of a cartilage, sternal. When a blow is expected, a strong inspiration is made, the ribs are then carried outwards, their extremities become more fixed, and their arches are rendered firmer, and the force is received with a *resistance*; but when the blow is unexpected, when there has not been time to effect a deep inspiration, the whole chest *yields* by its admirable elasticity, and the force of the shock is disseminated and lost over its whole surface, often with perfect impunity to its structure: but the five inferior ribs being so indirectly connected with the sternum, cannot have their anterior extremity fixed in the same firm manner as the seven superior by the deepest inspiration; they protect not, therefore, by *resisting*, but by *yielding*.

The anterior region affords also two modes of protection; namely, by resistance and by yielding: the sternum is slightly convexed or arched: its two extremities are movable, the inferior most so; and when a strong inspiration is made, the sternum is elevated, particularly at its xiphoid portion, and the bone is thus firmly fixed, and *resists* a force applied to it; if, however, that force be unexpected, it *yields*, and the shock is diffused over the chest, and lost.

An illustration of the two modes in which a force may be received is, I dare say, familiar to most of you: thus, in catching a ball, struck, I will suppose, from a bat, a tyro at the game will advance his hand to receive it, and will suffer

according to the resistance he gives ; while the experienced player allows his hand and arm to yield, and the force of the ball is diffused over the whole of his body.

Movements. — The next important function which the chest has to perform relates to its movements. In order to understand the movements of the chest, we must first study it in its various diameters : these are three. In the first place, we drop a line perpendicularly at the upper aperture of the chest, midway between the sternum and vertebral column to the diaphragm, and we call it the *perpendicular* or *diaphragmatic* diameter. We then take the measurement from the posterior part of the sternum to the anterior part of the bodies of the dorsal vertebræ, and term it the *antero-posterior* or *sterno-vertebral* diameter. We then measure the chest across from rib to rib, and denominate it the *transverse* or *costal* diameter.

In consequence of the conoid form of the chest and of the oblique direction of the diaphragm, which is the base of the cone, these diameters vary in different parts : thus, the perpendicular is greater at the posterior portion of the chest, and shorter as we advance towards the sternum ; the antero-posterior is longest from the xiphoid cartilage to its opposite dorsal vertebra, and shortest from the first bone of the sternum to the first dorsal vertebra ; the transverse is broadest at the lower part of the thorax, and becomes gradually narrower as we ascend.

Movement of inspiration. — There are two movements of the chest, the one by which we

inspire a certain quantity of air, and consequently increase the capacity of the lung; and the other, by which we expire, and thereby diminish the capacity of the organ. Every time we inspire, it is necessary that the chest should enlarge in one or other of its diameters, in proportion to the quantity of air inhaled: this is self-evident. What, then, is the diameter which you would, *à priori*, suppose would first increase? Of course, where the resistance is the least, and that is the diaphragmatic. In the act of expiration, the diaphragm is convex towards the lung, and concave below; but in proportion to the quantity of air inspired, it descends and presses the abdominal viscera before it, and this increase in the capacity of the chest is sufficient for the ordinary inspirations in a healthy person; indeed, in old age, when the ribs become more fixed, the respiration is carried on alone by the diaphragm. When, however, the respiration becomes difficult, it is then necessary that all the diameters of the chest should be increased to their full extent: to understand this, let us suppose a case of extreme dyspnœa, as that arising from severe peripneumonia; on observing the patient, you will see that he is seated in nearly an upright posture, with his head thrown back, and his arms widely extended.

The head and neck are directed backwards, and are firmly fixed by the action of the muscles arising from the back, and inserted into the occipital bone: these parts becoming thus firmly fixed, the sterno-cleido-mastoidei act from them upon the sternum and clavicles, and draw the chest directly upwards. The clavicles being thus rendered firm,

the subclavii muscles passing from them to the first ribs, elevate those ribs, and render them fixed points also: but you are aware that these ribs are not only elevated by the subclavii, they are still more powerfully so by the scaleni.

The first ribs being thus fixed, the second are elevated and fixed also by the action of the double layer of the intercostal muscles, the fibres of which decussate each other, so that the action of one series would be to elevate the second ribs *obliquely* upwards; but the result of their united actions would be to draw them *directly* upwards: the second ribs being thus fixed, the third are elevated in the same manner by their intercostals, and so on to the last, each rib becoming alternately a movable and a fixed point.

The whole of the ribs become thus elevated; but as they are directed obliquely downwards towards the median line; as the intercostal spaces, particularly of the false ribs, are wider anteriorly than posteriorly; and as these latter ribs are more movable from these circumstances, and from their greater length and mode of insertion of their cartilages, so the sternal extremities of them all, but particularly of the abdominal, are elevated more than their vertebral, and their bodies are consequently thrown outwards. You have only to place your hands on the sides of the thorax in making a deep inspiration, and you will distinctly feel this. If the ribs are thrown outwards, the transverse diameter must be increased in proportion.

The *antero-posterior* diameter is enlarged as a necessary consequence of the elevation of the ribs; for, at the moment of that elevation, the sternum

is pushed upwards particularly by the cartilages of the false ribs ; consequently, the space between that bone and the bodies of the dorsal vertebræ must be augmented.

I have stated to you that the arms are also extended in extreme cases of dyspnœa ; the superior extremities are thrown out, at acute angles, from the body, or are elevated above the head, to become fixed points, from which the ribs may be acted upon, and drawn outwards, to increase the transverse diameter of the chest. Thus, the pectoralis major being connected with the outer edge of the bicipital groove of the humerus, spreads broadly upon the upper parietes of the thorax, and acts upon the cartilages of the true ribs ; the pectoralis minor, attached to the coracoid process of the scapula, elevates obliquely upwards the second, third, and fourth ribs ; and, finally, the serratus magnus, passing from the inside of the base of the scapula, and fixed by nine digitations into so many of the corresponding upper ribs, most powerfully draws them directly outwards, the scapula at the same time being fixed by the action of the trapezius, rhomboideus, and levator-anguli scapulæ.

You may see, that by a beautiful compensation, the lower ribs, from their greater length of cartilage, and from their greater obliquity of course, are by far the most easily moved, and have therefore fewer muscles to effect their movements ; whilst the upper or thoracic ribs, from their greater fixation, have a number of powerful muscles to act upon them.

Varieties of dyspnœa. — Authors have made

several varieties of dyspnœa, which I shall now notice. We have first what is called *abdominal* respiration, by which we mean the moving of the abdomen to a great extent, while the chest is immovable. When any cause prevents the extension of the transverse or the antero-posterior diameters, then the perpendicular diameter alone is susceptible of increase. Suppose the case of a patient labouring under a violent attack of pleuritis, every time he attempts to expand the chest by increasing the transverse diameter, he presses the lungs against the inflamed part: he endeavours to avoid this by breathing by the diaphragm alone. Suppose, on the other hand, there be a difficulty of moving the diaphragm, as in peritonitis, in that case every time the perpendicular diameter is increased, the viscera are pressed against the inflamed part, and the pain is aggravated: the patient therefore tries, by elevating the chest, to increase the antero-posterior and transverse diameters only. In cases also of ascites, or pregnancy, or large tumours in the abdominal cavity, the perpendicular diameter is increased with difficulty, in consequence of the mechanical obstruction to the descent of the diaphragm: patients then breathe principally by elevating the chest. This is called *thoracic respiration*.

We have respiration which is called *complete* or *incomplete*. It sometimes happens that there is an effusion of fluid in large quantity into the cavity of one or other of the pleuræ, in consequence of which the lung becomes compressed. It cannot dilate, and the ribs do not move on

that side, though they are active on the other, and hence the term *incomplete respiration*.

We have also *equal* and *unequal* respiration. These terms have merely a reference to time: thus, the inspiration may be shorter than the expiration. In acute pleuritis, the inspiration is short, and the expiration long.

Sometimes we meet with the expression *frequent*, or *slow*, respiration; but these words do not require any definition.

We have also *high* and *sublime* respiration. This occurs in extreme cases, where the patient endeavours to encrease the diameters in every way.

There is another kind of respiration, denominated *orthopnœa*, by which we mean that the patient is enabled to breathe only in an upright posture.

Movement of expiration. — The act of expiration is performed in the healthy state by the diminution of the perpendicular diameter of the chest, produced by the elevation of the diaphragm; but when we have a forced expiration, then all the diameters are diminished: this will be best understood by explaining the mechanism by which coughing is effected.

Forced expiration — cough. — A cough is commonly an effort of nature to remove some cause obstructing the passage of the air into the lungs: thus, the tracheal and bronchial mucus, if not absorbed or evaporated in proportion to its secretion, as in catarrh, accumulates, obstructs the air-passages, and we cough to remove the obstruction. In old catarrhal persons, the power of coughing is often lost, and they become suffocated. Persons

dying of other diseases lose this power, and the mucus accumulates, and the well-known and fatal tracheal rhoncus, or rattling sound, is then heard. Other obstructing causes may exist in the lungs, as, the matter of abscesses, of gangrene, of softening tubercles, &c.

Another very frequent cause of cough arises from irritations in and about the rima glottidis. You all know the extreme irritability of that part : it often happens that it becomes slightly inflamed, and then it is in the beginning dry, and shortly afterwards it throws out a thin and acrid secretion, as we shall describe when we arrive at that subject. A violent cough is the consequence, and it is continued until sufficient mucus is torn from the tracheal and bronchial surfaces to moisten the dry state of the membrane, or to soften the acrimonious quality of the irritating secretion. This cough, from its violence, has been called "tussis ferina." Whatever, however, be the cause of coughing, the mechanism is the same : it consists in a sudden contraction of all the diameters of the chest, for the purpose of pressing the air through the air-passages with the greatest force, to carry the mucosities with it ; accompanied at the same time by a *contraction of the glottis*, to afford a greater impetus to the air by diminishing the diameter of its column.

The recti-abdominales, arising from the pubes, and inserted into the sternum and cartilages of the fifth, sixth, and seventh ribs, draw the chest directly downwards, and thus act as the antagonists to the sterno-cleido-mastoidei ; the two oblique and transversales, having their attachment at the

cristæ of the ilia and Poupart's ligament, pass to the cartilages of the whole of the false ribs, and draw them also towards the pelvis; the quadrati lumborum attached below to the posterior part of the cristæ of the ilium, and to the ilio-lumbar ligaments, are inserted into the last ribs, and, by their contractions, pull those ribs downwards, and fix them, and thus become the antagonists to the scaleni. The last ribs becoming thus fixed points, the last row of intercostal muscles acting from the twelfth ribs, depresses the eleventh, and fixes them; the tenth are also acted upon and fixed by their intercostals in the same manner; and so on successively to the first; so that you perceive the functions of the intercostals are reversed in the act of expiration, that difference depending upon the position of the fixed points. The triangulares sterni also operate by drawing down the cartilages of the third, fourth, fifth, and six ribs. The serrati, postici inferiores, the sacro-lumbales, &c., all depress the ribs.

The result of these actions is to approximate the chest to the pelvis: in doing so, the ribs are also drawn inwards and the sternum downwards, as may be distinctly felt on applying the hands to the chest during coughing: thus, the transverse and antero-posterior diameters are lessened, and, as the diaphragm is elevated at the same moment, the capacity of the thorax is diminished in all its dimensions, the lungs are compressed, and the air driven out with force.

Contraction of the glottis. — At the instant the contraction of the chest takes place, the rima glottidis contracts also, so that the column of air

ascending the trachea, acquires additional force to carry the obstructing secretions with it, on passing through the narrow orifice of the opening of the larynx. The peculiar sound of cough is produced, too, by that contraction: such is the case also in spitting: however forcibly you make an expiration, not a particle of saliva will pass the mouth; the lips must be previously contracted, the fluid is then projected, and the sound of spitting is heard at the same moment.

There are several varieties of cough: we shall speak of them when we consider their particular causes.

LECTURE III.

THEORY OF THE SIGNS OF DISEASES OF THE
LUNGS — (*continued*).

PERCUSSION.

Obscurity of Signs of Diseases of the Chest — Why? — Signs mistaken for Diseases — Equivocal Nature of Signs — Notice of Avenbrugger — *Percussion* — Principle upon which it depends — Experiment — Application to the Chest — Position of Patient — Mode of Percussing — *Plessimeter* — Variations in Sound at different Ages and Temperaments — Variations of Sound at different Parts of Chest.

Regions of the Chest — *Anterior*, Clavicular — Anterior Superior — Superior Mammary — Sub Mammary — *Lateral* — Axillary — Superior Lateral — Inferior Lateral — *Posterior* — Supra and Infra Spinal — Interscapula — Dorsal — Character of Sounds in different Regions.

Inferences — Diminution of Sound — Why? — Increase of Sound — Why? — Value of Percussion — *Succussion*.

WE now proceed to the subject of Percussion.

The diseases of the chest were involved in great obscurity from the earliest periods of medicine up to the middle of the last century. The reason of this appears obvious. In the first place, the lesions of the particular textures which enter into the composition of the lungs and heart, were not then well understood. An acquaintance with morbid anatomy in general had not arrived at the point which it has attained at the present day; and it follows as a matter of necessity that if these morbid lesions were not known, neither could the signs be to which they gave rise. We have been too much, also, in the habit of satisfying ourselves

with the study of signs alone, and considering them as diseases, instead of reflecting upon the alterations of structure which give origin to them. Thus we find that Cullen has established a genus which he calls *dyspnœa*. Now difficulty of breathing is only a sign of a particular condition or conditions of the chest, and is not a disease: it is the internal cause which produces the dyspnœa that is really the disease. Thus, again, Cullen and other authors have defined *asthma* to be a difficulty of breathing occurring at intervals, without fever; but this, which they call a disease, is merely a sign, and a sign depending upon various causes.

Bichât, by his views of general anatomy, gave a new impulse to the study of morbid anatomy. He, from a hint given by Pinel, no longer regarded an organ as a homogeneous structure, but as composed of a variety of different tissues. He analyzed these tissues, and pointed out that they were subject to different physiological and pathological laws. Since that period, morbid anatomists have taken the same view, and followed up their investigations according to the same principles. They have unravelled (if I may so express myself) the diseases of the various elementary structures of a viscus, and have shown them in their isolated and combined states. To take an example from the lungs: we have here an organ which does not consist of a homogeneous structure, but of air tubes formed of cartilage and mucous membrane, &c.; of cellular tissue and vesicles, of blood vessels, of nerves, and, finally, of a serous membrane, which envelopes the whole. All these have diseases peculiar to themselves, which may exist in an iso-

lated or combined manner: thus we may have pleuritis without catarrh, and *vice versâ*. As we proceed we shall have constantly-recurring evidence of these facts. Morbid anatomy is advancing most rapidly, and consequently affections of the chest, like other diseases of the body, are really becoming better known.

In the next place, diseases of the chest were not understood, on account of the equivocal nature of their signs. Dyspnœa, a great and leading sign of these affections, is equivocal, inasmuch as it is common to every disease, both of the lungs and the heart, when it has reached to a certain point; and it also occurs in some diseases external to the thoracic cavity. Cough, too, is equally equivocal, for it may occur in all diseases of the chest, and even in some abdominal disorders; nor is the violence of a cough any proof of the gravity of the disorder, as the severest may happen from comparatively trifling affections, as a slight inflammation attacking the larynx, whilst the lung may be profoundly lesed, as in peripneumonia, and cough may be absent.

There can be no doubt that the inadequacy of these signs to determine any specific disease first led Avenbrugger to think of percussing the chest. But, before I speak of his discovery, I will mention a few particulars relating to himself. Leopold Avenbrugger, or Auenbrugger, was born at Graets, in Styria, in 1722: he became physician to the imperial hospital of the Spanish nation at Vienna. There it was, as he expresses it, *inter tædia et labores*, that he prosecuted his studies on this subject for a period of seven years; and in the year

1763 he published a work which he called "*Inventum novum ex Percussione Thoracis humani, ut signo, abstrusos interni pectoris morbos detegendi.*" No notice whatever was taken of his book until the year 1770, when Rozière de la Chassagne, of Montpellier, translated it, avowing he knew nothing of the subject practically; and the inference which he would lead you to draw is, that he did not think much of the discovery himself. The pamphlet seemed now consigned to oblivion: however, Stoll, Van Swieten, and Cullen, in his First Lines, make some mention of it; but, like Rozière de la Chassagne, declared they knew nothing of it practically. Corvisart, when preparing his celebrated clinical lectures, first met with the subject in reading Stoll's work. He immediately commenced a series of experiments, which he continued for twenty years; and in 1808 he published a new translation of Avenbrugger, with his own commentaries; a work that has been translated, I believe, into every European language.

Principle upon which percussion depends.—Let us, in the first place, speak of the principle upon which percussion depends. Suppose a hollow ball, or a vessel of any kind, made of wood, glass, metal, or any other material, and containing atmospheric air only: strike it with the finger, and a certain sound will be produced, varying, of course, in kind and intensity, according to the size of the vessel, the nature of its parietes, and their thickness. Half fill this vessel either with a fluid or solid matter, percuss it, and there will be a difference in the sound. The sound will be duller than it was before; and the lower part, containing the fluid

or solid, will be duller than the upper, which contains air only. Fill the vessel completely, and the sound of course will be duller than in the first instance. This is so perfectly obvious that no experiments can be necessary to prove it.

What is the human chest? It is a hollow cavity, containing within it two organs; the one solid, at least so far as regards percussion — the heart; and the other the lungs, an organ containing a quantity of atmospheric air. Hence the chest will give a louder sound in the healthy state, on percussing it, than any other part of the body.

Before we proceed to elicit the various sounds which may be produced by the percussion of the chest in the living subject, let us first describe some preliminary steps relative to the position of the patient, &c.

Position of the patient. — It is best to place the patient in a chair with a wooden seat: if, however, he be in bed, let the curtains be widely opened, that the sound be as little deadened as possible. The chest may be examined in reference to its anterior, lateral, and posterior regions. If the anterior, the head and shoulders should be thrown well back, and the arms brought behind the chair: in this manner the pectoral muscles are put upon the stretch, and a better sound is produced. If the lateral region is to be examined, the body should be inclined to one side, and the arm passed over the head, and then percussion is easily performed from the highest point of the axilla to the lowest rib: if the posterior region, let the patient fold his arms, incline his shoulders inwards, and his head forwards; the muscles of the back will thus be

rendered tense and firm, and the sounds may then be easily obtained.

You will observe that the objects we have in view in these positions are to place the patient in a convenient posture for the operator, and to render the external parietes of the chest of the patient as firm as possible, by putting the muscles upon the stretch.

Mode of percussing. — With regard to the mode of percussing, you should never strike the patient with much force, for by that means you give pain, and do not elicit a good sound. The object is to percuss smartly and quickly; the fingers should remain on the chest but an instant. To percuss effectually, the four fingers of the right hand must be employed; their extremities should be brought into the same line, the index being supported by the thumb. When you percuss a rib, strike parallel to it; for if you percuss it transversely, your fingers will then come upon the intercostal spaces above and below it. Always be careful, too, to percuss at the same angle, and with the same force, in comparing the opposite sides of the chest, as a slight difference will occasion a difference in sound, which would not occur if the percussion were properly effected.

It oftentimes happens that the parietes are flaccid, or fat, or the skin of the patient is so exceedingly irritable and tender, that he cannot bear the blow of the fingers, however slight. These difficulties are obviated by the instrument invented by Piorry, called the plessimeter, being, as you see, a round piece of ivory of about one inch and a half diameter, and two or three lines thick. This may

be placed on the chest, and a good sound may be obtained by striking it with the fingers, without occasioning any inconvenience. I often place the fingers of the left hand firmly upon the chest, and percuss them with those of the right. Laennec frequently struck the back of the patient by a slight but quick blow with the stethoscope.

Variations in sound at different ages, and in different temperaments. — A very important question arises here, viz. whether the sound elicited from the chest is the same at all periods of life? Certainly not. The sound produced from the chest of a child is infinitely louder than that of an individual in the middle period of life; and that of the latter is also much louder than from an old person. The reason of this we shall inquire into hereafter.

Are there any differences with regard to sex? As a general rule, a louder sound may be obtained by percussing the female than the male chest. The sound is more distinct also in persons of a nervous or irritable temperament, than in those of an opposite character, although of the same age. These circumstances lead to this conclusion, that you should never compare the sounds produced from the chests of two or more individuals, to arrive at a diagnosis, since these persons may differ naturally; but you should compare the different parts of the chest of the same individual.

Variations according to situation. — It then becomes a matter of importance to determine, whether the different parts of the chest of the same individual produce the same sounds: they do not. Laennec, to facilitate the study of this subject, has

divided the surface of the chest into the following regions.

Regions of the chest. — The *clavicular* region comprises the length of the clavicle: this region is subdivided into the sternal, middle, and humeral portions. The *anterior superior* region extends from the first to the fourth rib; the *superior mammary* from the fourth to the eighth; the *sub-mammary* from the eighth to the twelfth.

That part of the chest which is occupied by the sternum is called the *sternal* region; it is subdivided into the *superior*, *middle*, and *inferior*.

At the lateral parts of the thorax we have three regions: the *axillary* from the highest point of the axilla to the fourth rib; the *superior lateral* from the fourth to the eighth; then the *inferior lateral*, from the eighth to the twelfth.

The posterior parietes have the following regions: the *supra* and *infra spinal*, corresponding to the supra and infra spinal fossæ of the scapula; the *interscapular*, between the bases of the scapula and the vertebral column; and the *dorsal*, extending from the angle of the scapula to the last rib.

Nature of sound elicited in the various regions. — The sounds elicited by percussion vary exceedingly in the different regions. Thus the sternal extremity of the clavicle gives a loud sound, the middle less, the humeral portion the least; the sound diminishing in proportion as the space becomes greater between the clavicle and the thorax.

The sternum varies in sound according to the region percussed; thus the superior yields the loudest, the middle somewhat less, the inferior the

least, — the latter in consequence of the heart being placed beneath it.

The anterior superior region is very sonorous; the mammary nearly equally so in the male; in the female the sound is dull, from the presence of the mamma. The sub-mammary is dull also on the right side, from the liver being placed beneath it; it is often very loud on the left, from the occasional presence of gases in the stomach.

The axillary and the superior lateral regions sound well; but the inferior lateral does not on the right, from the presence of the liver; and on the left it is frequently too sonorous, from flatus in the stomach.

The supra and infra spinal, and the interscapular regions give fleshy sounds; but the spine of the scapula, and the spinous processes of the dorsal vetebræ, yield them louder.

The greater part of the dorsal region is very sonorous; but the lower portion is dull on the right side, and often very loud on the left, for the reasons I have assigned in speaking of the inferior lateral and sub-mammary regions.

Inferences. — What inferences are we to draw from these examinations? First, that any cause which tends to diminish the quantity of atmospheric air in the lung must tend to diminish the intensity of the sound produced by percussion, the sound being dependent upon the presence of air. Thus, when a portion of the lung is consolidated, as in hepatization, the sound given by the parietes of the chest over the diseased part is dull, in consequence of the total absence of air in that part. When a mass of tubercles exists in the lungs, there

the sound will also be dull, and for the same reason. Again, when fluid accumulates in the pleuritic cavity, it compresses the lung, and diminishes its volume in proportion to the quantity effused; and in proportion to the diminution of the lung, so will the quantity of air be lessened, and the sound elicited from the side affected will be consequently obscure.

Our second inference will be the converse of the last, viz. that any cause which tends to increase the quantity of atmospheric air in the chest, must increase the intensity of the sound on percussion. We have two remarkable instances of this; the first in emphysema of the lungs, of which disease I shew you a splendid specimen. Here you see the air cells are dilated; some of them have ruptured and communicated with each other, forming the appearance of blisters upon the surface of the organ: here the quantity of air is beyond the normal proportion; and the part of the chest situated over the portion of the lung affected produced a very loud sound.

The next case is that of pneumo-thorax. From cases which we shall hereafter examine, we occasionally find an accumulation of air in the cavity of the pleura. The chest then sounds like a drum on percussing it.

As I have before stated, dyspnœa and cough are signs totally inadequate to the determination of any *specific* lesion of the pulmonary structure, since they are common to them all. By percussion, however, we can discover the locality of the affection, whether it be in one lung or the other, and in what part of the lung; and still more, we can

distinguish whether the part be permeable to air or not ; but it affords no demonstration of the cause of the impermeability of the organ, as the same dull sound is produced in hepatization or effusion of fluid into the pleura, &c. ; nor can we say, when the sound is louder than natural, whether it depends upon emphysema of the lung, or pneumo-thorax. Auscultation overcomes these difficulties.

You thus see, gentlemen, that the method of Avenbrugger affords most important signs of the diseases of the lungs ; and it is very probable that Laënnec would never have been led to the discovery of auscultation, had it not been for his previous perfect knowledge of percussion.

It is the fate of all discoveries, however great, to meet with objections ; not that I think opposition is of any disadvantage to them, for it forces the discoverer to consider his subject on every side, even if it be only for the purpose of its more complete demonstration ; and in doing so, new views often arise, leading him to further discoveries. Percussion has met with its opponents ; but the facts are now too well attested to admit of any doubt ; and I recommend your constant practice of it — for it requires it — for the purpose of acquiring an early facility.

SUCCUSSION.

We shall defer the subject of Succussion, as it applies only to the particular case of pneumo-thorax, with effusion into the pleuritic cavity, until we arrive at the consideration of that disease.

LECTURE IV.

THEORY OF THE SIGNS OF DISEASES OF THE
LUNGS — (*continued*).EXAMINATION OF THE SIZE, MOVEMENTS, ETC. OF
THE CHEST.

Signs derived from the Examination by the Touch, and of the Size, Movements, &c. of the Chest.

Examination by the Hand — Abdominal Diseases — Vibration of the Voice — Presence when — Absence — Conclusions — A Multilocular Cavity — Rhoncus sonorus gravis — Emphysema — Fluctuations — Aneurisms.

Movements of Chest — Complete, incomplete, Causes — Pleuritic Effusion — Acute Pleurisy — Pleurodynia.

Dimensions of the Chest — Dilatation of one Side — Cause — Contraction — Cause — Mensuration. — Abdominal Pressure — Pulmometry — Biographical Notice of Laënnec.

I now proceed to the consideration of some other signs of disease of the chest, which we shall class under the following heads: — in the first place, those derivable from the touch; secondly, from the examination of the external parietes of the chest; thirdly, that from pressing the abdomen, a means imagined by Bichât; and, fourthly, of the mode of measuring the capacity of the lung, proposed by the late Mr. Abernethy.

EXAMINATION BY THE HAND.

First, with regard to touch. The sign afforded by the touch generally gives to the physician but

very insufficient data ; the hand is infinitely more important to the surgeon in detecting disease : however, we derive some advantage from it, particularly in affections of the abdomen. Thus we can discover peritonitis, and determine its extent, its situation, and, indeed, its degree, by the amount of pressure which the patient can bear. When the liver or the spleen are enlarged, we can determine, to a certain extent, their volume, by the hand. Dropsies, or tumors formed in the mesentery, may be detected by the same means. Upon one occasion, I ascertained by the hand the presence of a calculus in the pelvis of the kidney. It was in a very thin subject. Certain affections of the uterus may frequently be distinguished by the touch ; and, in fact, it is much more easy to distinguish diseases of the abdomen by the hand, in consequence of the softness and pliability of its parietes, than the diseases of the lungs, surrounded, as those organs are, by the comparatively unyielding surface of the chest.

There are, however, some signs of diseases of the lungs that may be derived from touch. Thus, for instance, if a person, with a very grave voice, speaks or sings, upon applying your hand to the parietes of the chest, particularly at the upper part, you will very distinctly feel a vibration all over it ; but it only occurs in persons with a large chest, and in whom the voice is grave. Can we make use of this sign in diseases of the lungs ? To a certain degree we can. You may be sure that wherever you feel this vibration, the lung is permeable at that part, and therefore it is useful as a confirmative sign. On the other hand, you must

not conclude that, because you do not feel the vibration, the lung is not sound, unless there be some other symptom indicative of disease: as if, upon percussing a certain part, you have a dull instead of a hollow sound, and are thereby led to infer the existence of disease, then, if, on applying the hand, you do not find a vibration, you have a confirmative sign of the impermeability of the lung. In empyema, the touch occasionally is of use. Suppose a collection of pus commences in the cavity of the pleura, and makes its way between the intercostal spaces, forming a projection between the ribs, you can then feel the tumor; and if, on pressing it, the fluid recedes, and then, by coughing, the swelling returns, you ascertain the nature of the affection by the tact. Sometimes a large cavity forms in the apex of one or other of the lungs, which is called a *tubercular excavation*; in the first instance, this cavity is not complete, but is usually traversed by a number of bands, of pulmonary structure, not yet destroyed, and in this state it has been called a *multi-locular* cavity. When this multi-locular cavity is near the surface of the chest, the matter contained within it becomes entangled, and every time the patient coughs, the secretion strikes the sides, and gives a feeling of gurgling, which gurgling may be distinctly felt by the hand. Again: there is a sound with which many of you are perfectly familiar — the deep sonorous wheeze, or rhoncus — which is heard in chronic catarrh. This sound is so loud and intense, that it communicates a vibration to the parietes of the chest; so that it is a common practice of mine to pass my hand round

the patient's body and to make him take a deep inspiration, for the purpose of determining the extent of this species of rhoncus. I do not recommend you to employ this means alone, because the stethoscope will determine the affection much better. *Emphysema pulmonum*, a disease which we shall see consists in a permanent dilatation of the air cells, or in the infiltration of air into the cellular substance which divides the lungs into lobules, often communicates a crepitation to the parietes of the chest, which may be felt by the hand; it is a crepitation like that which a thin dried bladder would give upon pressure. The *fluctuation* upon movement in cases of pneumothorax, with effusion into the cavity of the pleura, can readily be detected by the tact. Aneurisms of the thoracic aorta, when they form external tumors, may be distinguished, of course, by the same means.

MOVEMENTS OF THE CHEST.

We now proceed to the examination of the signs to be derived from observing the parietes of the chest; and, first, in relation to its movements.

There are three diseases by which that state of respiration is produced which we have called *incomplete*, a state in which the ribs may be seen immoveable on one side of the chest, and moveable on the other.

The first case is that in which an accumulation of fluid takes place in the pleuritic cavity of one side; the lung then becomes compressed by the increased secretion against the vertebral column,

so that it is often reduced to a very small volume ; the respiratory act ceases on that side ; the ribs are there, consequently, perfectly quiescent, while you will see on the opposite or healthy side, that they move with a rapidity proportionate to the dyspnœa.

The second case is that of very acute and extensive pleurisy ; the patient then experiences great pain during the act of inspiration ; he consequently avoids elevating the ribs on the side affected, so that they become immoveable, or nearly so. This may be easily distinguished from effusion ; inasmuch as in acute pleurisy the disease occurs suddenly, and there is much pain, neither of which happens when effusion takes place ; and in the latter case also the diseased side of the chest becomes enlarged.

The third instance of incomplete respiration arises from rheumatism of the muscles of the chest, or, as it has been called, *pleurodynia*. Such is, then, the pain occasioned by throwing the inspiratory muscles into action to elevate the ribs, that the patient avoids doing so as much as possible, and breathes only by the healthy side. This state may easily be distinguished from the two others, by the pain being increased by elevating the arm of the affected side, by the patient not bearing pressure upon the ribs, and by co-existing rheumatism of some of the other muscles of the body.

DIMENSIONS OF THE CHEST.

Let us now examine the chest in relation to the capacity of its two sides, for differences may occur

in that respect, rendering most important signs. We will proceed to explain the causes of these differences ; and first, of

Dilatation of one side of the chest.—We shall see, when we arrive at the subject of pleurisy, that, in the second stage of that disease, more or less fluid is accumulated in the cavity of the pleura ; sometimes the quantity is very small, occasionally it amounts to many pints ; I have seen as much as nine pints.

You cannot but suppose that this large quantity of fluid must produce some effects by its presence upon the parietes of the cavity in which it is contained ; and it is to those effects I wish, at this moment, to draw your particular attention.

The pressure of the fluid will first affect the parts which, from their structure, offer the least resistance ; thus the lung, being of a soft and spongy tissue, vesicular in its texture, and filled only with air, diminishes gradually in its volume, in proportion to the accumulation of the fluid ; so that at last it is reduced to a very small mass, which is pressed against the vertebral column. In the next place, the heart is driven out of its position by the fluid, so that if the effusion be on the left side, that organ is pushed towards the right, and its beatings can be heard on the right side of the sternum, and scarcely at all on the left : if the accumulation be on the right side, the heart is pressed still farther to the left. The diaphragm, forming the floor of the cavity, will be forced downwards ; but as the liver supports that muscle, particularly on the right side, so much resistance is afforded, that the effect of the compression is rarely

sufficiently considerable to depress that organ below the margin of the false ribs ; it no doubt does so occasionally, but I think rarely. If, however, the fluid be in the left pleuritic cavity, the diaphragm will be rendered permanently convex towards the abdomen, will push forwards the stomach, &c., and form a diffused projection along the margins of the cartilages of the false ribs of the same side.

Observe the effects of the pressure upon the external parietes of the chest : the intercostal muscles are first acted upon ; they are pressed directly outwards, so that the inner surfaces of these muscles are projected to a level with the external surface of the ribs, — thus effacing altogether the appearance of intercostal spaces, and rendering the whole side perfectly smooth, while on the other side those spaces may be sufficiently well marked ; nay, some have said that the intercostals have so projected as to bulge beyond the ribs, so that the spaces have become elevated instead of depressed. I have often sought for this, but I cannot say I have ever distinctly seen it. Not only are the intercostal muscles acted upon, but also the ribs themselves ; they are pushed directly outwards, so that the transverse diameter of that side of the chest becomes increased, and its capacity enlarged. Nothing is more easy than the detection of this enlargement by the eye : a small difference may be perceived — a difference only of a quarter of an inch in the comparative measurement of the two sides, can be determined. Laennec was satisfied with the appearance alone ; but as the sign is rendered more perfect by measuring, I should recom-

mend you always to do so, more particularly as you occasion no pain or distress to the patient.

Mensuration of the chest. — For this purpose I generally use a measure, made of some inelastic stuff, graduated into inches and tenths of inches; I apply one end of it to the centre of the xyphoid cartilage, and pass it round to the spinous process of the dorsal vertebra exactly corresponding, observing the number of inches; I then measure the other side exactly in the same way, and note the difference. You may measure also higher up if you will, but I am generally satisfied with that which I have mentioned.

Contraction of one side of the chest. — After effusion has taken place from inflammation of the pleura, an albuminous matter separates from the fluid and forms a false membrane, which lines the parietes of the containing cavity. This membrane adheres to the costal pleura, is reflected over the diaphragm, and passes from thence over the lung. It varies considerably in thickness; sometimes it is a mere thin and semi-transparent pellicle, through which the colour of the parts beneath can be distinctly seen — frequently it is several lines thick, and perfectly opaque.

Nature often removes the effused fluid by the process of absorption, or art may effect that removal by the operation of paracentesis thoracis. In either case, in proportion to the evacuation of the fluid, the compressed lung gradually expands, and tends to resume its former volume. If the false membrane be thin, it will dilate with the lung, and allow that organ again to touch the ribs; but if it be thick, it will bind the viscus so com-

pletely against the vertebræ that no expansion can take place. Both these are, however, extreme cases; it generally happens that the false membrane allows a very considerable, although not complete, development of the lung.

A beautiful effort of nature follows this state of things; for when the lung cannot farther dilate — when it cannot touch the costal pleura — the whole of the ribs of the affected side fall inwards upon the organ, sometimes sufficiently to reach it; the false membranes of the pulmonary and costal pleura then adhere, and form a single membrane of a fibro-cartilaginous structure.

In these two casts you perceive well-marked instances of this *falling in*, or contraction of the chest. The operation for empyema was performed in both cases. In the smaller one, the measurement before the operation gave an inch and a quarter more on the diseased side than on the healthy. You now see that it is an inch less; giving a contraction altogether of two inches and a quarter. This large cast offers a still greater difference, and the depression of the side is here great indeed.

Consequent upon this depression of the ribs, the shoulder becomes depressed also — the inferior angle of the scapula is lower on the affected than on the opposite side — the last rib also approximates much nearer to the crista of the ilium.

The sternum and dorsal vertebræ deviate from their natural positions. If the contraction be on the left side, the sternum and vertebræ are pushed towards the right; so that the centre of the former bone is not on the median line, and the vertebræ

form a curve whose convexity is to the right side also : if the contraction be on the right, the sternum and vertebræ will deviate towards the left side.

ABDOMINAL PRESSURE.

Abdominal pressure was proposed by Bichât. He imagined that we could determine the capacity of the chest by pressing firmly upon the abdomen, so as to diminish the perpendicular diameter of the chest, and see how much the patient could bear before he felt a sense of suffocation. You may suppose that it must be impossible to do this — particularly under disease. Laennec justly remarked, in reference to this means, that we have no right to interrogate nature by putting a patient to the torture.

PULMOMETRY.

The late Mr. Abernethy proposed to determine the capacity of the lungs by making the patient breathe through a tube, the opposite extremity of which was passed into an inverted jar full of water, placed in a pneumatic trough, and to observe the quantity of water he displaced in the jar. This means looks very pretty in theory, but it is perfectly useless in practice ; for some persons, even of large chests, will displace less of the water than those of smaller proportions, from the greater determination of the latter in continuing to expel the air from their lungs ; but as it is the comparative capacities of the healthy or disordered states we wish to obtain, how can we possibly know what quantity the individual could have breathed in the

healthy condition? It is needless, also, to state how difficult it would be to force a person, labouring under dyspnœa, to make the experiment; sometimes, too, it would be even a dangerous one.

These are the modes of investigating diseases of the lungs, and I hope you have by this time been able to appreciate their various degrees of value; but we have one more means, of infinitely greater importance, by which we can determine the specific nature of the lesion, viz. by AUSCULTATION. But this is too interesting a subject to commence towards the close of a lecture; I shall therefore defer it till the next. I will, however, occupy your time with a few observations on the biography of Laennec.

René Theophilie Hyacinthe Laennec was born on the 17th of February, 1781, at Quimper, in Lower Brittany. His father was an advocate. Laennec was placed, early in life, under the care of two paternal uncles — the one an ecclesiastic, and the other a physician, Dr. Laennec, professor of materia medica at the secondary school at Nantes.

Laennec very early obtained a reputation for scholastic acquirements, more particularly in the Greek and Latin languages. He used to communicate with his pupils, and deliver his clinical lectures in Latin, for the double purpose, as he said, of familiarising them with that language, and preventing the patients under his care from hearing his opinion of their cases. He was also much attached to antiquarian pursuits, particularly relating to the dialect of his own province; and nothing

seemed to please him more than to meet with a Welchman, for the purpose of conversing with him, and comparing the Armorican with the ancient British tongue.

In the year 1799, Laennec was appointed assistant-surgeon in the army of the west, — an army directed at that time towards the Morbihan, for the purpose of quelling an insurrectionary movement made against the constituted authorities. In 1800, he arrived at Paris to complete his education. It was at that period, in France, and under the consular government, that the greatest encouragement was given to the study of all the sciences, and more particularly to those of medicine, surgery, and all their collateral branches. The professor's chair was then the reward of a hard-fought contest, and *merit* alone could by possibility insure the victory. I trust you will not consider it misplaced if I say, that I hope to live to see the day in this country, when no man shall be permitted to occupy any station in which he has to teach others, without having first passed through the ordeal of repeated examinations — examinations not only of the knowledge he possesses, but of his power of communicating that knowledge. Let me hope that the time will arrive when merit shall be the passport to posts of honour — when we shall no longer be obliged to canvass the suffrages of men who, however estimable in other respects, can be no judges of our professional qualifications — when the present system of election shall be totally abandoned, as a system utterly disadvantageous to the public, and degrading to every honourable aspirant.

It was at that time that the reputation of Corvisart was at its zenith ; the force of his example, and the value of his clinical instructions, carried enthusiasm into the minds of all his followers. The star of Bichât, too, was upon the ascendant, soon to shed a new lustre upon the face of anatomy and physiology. Bayle and Dupuytren were the contemporaries of Laennec ; and it is no wonder that, with masters like these, and a genius like his, he should soon be seen among the foremost in the rugged path to the temple of fame.

He gained the two chief prizes in medicine and surgery, granted by the Minister of the Interior through the Institute of France ; after which he became a critical writer ; and the first work upon which he exercised his talents was Benjamin Bell's Treatise upon Syphilis. Impressed, as he had been from the commencement of his studies, with the importance of morbid anatomy, he began early a course of lectures on that subject, which he continued for three or four years ; Dupuytren giving a course of a similar kind at the same period. Laennec was the first in France to describe the adventitious deposit called by him *melanosis*, and also the "*matière encéphaloïde* ;" but the latter had been previously made known in England by Burns, under the name of *spongoid inflammation* ; by Hey, of Leeds, under that of *fungus hæmatodes* ; and by Abernethy, who denominated it *medullary sarcoma*. Dr. Forbes, in his excellent translation, raises a question as to whether Laennec had any knowledge of the disease having been previously written upon in England, and he thinks he had not ; but considering that there was then a short peace, I think it

probable that the French anatomists must have known what had been going on in England.

But it is from the year 1816 that the great fame of Laennec is to be dated. At that time he was appointed physician to the "Hôpital Necker," an institution having similar objects to our Infirmary for Diseases of the Lungs. He had occasion at that period to examine a young female, who he supposed was labouring under disease of the heart. From that delicacy of feeling, which every medical man should possess, he did not like to apply his ear to the chest, though that idea first struck him. He recollected at the moment that, as solid bodies have the power of conducting sounds better than air, it would be possible by such a means to ascertain more than could be done by the hand. He procured a quire of paper, rolled it up tightly, tied it, and then applied one extremity to the patient's chest, and the other to his ear. To his great satisfaction, he heard the beating of the heart infinitely more distinctly than it could possibly be felt by the hand. A discovery like this was not lost upon Laennec, and he immediately conceived the possibility of applying the same means to diseases of the chest in general, but more particularly to the examination of the voice. He commenced a series of observations upon different patients merely in relation to the latter object. He then first discovered *pectoriloquy* and its cause. With unexampled ardour he continued his researches; and in the year 1818, only two years after his first discovery, he brought forward his extraordinary work on Diseases of the Chest; a work which, for elegance of composition, for correctness of anatomical de-

scription, and for originality of facts, is unparalleled perhaps in the history of medicine.

As you may readily conceive, great opposition arose against his discoveries: his facts were denied, his inferences refused, his instrument ridiculed; and, if it had not been for the manner in which the work was written, for the clearness of his anatomical details, and his personal efforts in proving the facts at the bed-side, no doubt auscultation would have fallen into the same oblivion as the percussion of Avenbrugger had done. Laennec's weak frame gave way under all these exertions, and he retired to his native country. His health to a certain degree became re-established, and he returned to Paris in 1821, to reap the fame of his discoveries, and to extend them. He was shortly afterwards appointed Professor of Medicine to the College of France, and Physician to La Charité. He then prepared the second edition of his work, to which he added the mode of treatment. While executing this task, he again fell into a state of great exhaustion; retired to his native home; and in August, 1826, expired, at the age of forty-five years.

Laennec died of consumption, and I have no doubt that he had long believed himself to be predisposed to that dreadful disease. You could not be with him for a very short time, without seeing him frequently expectorate, and examine the secretion with great minuteness: indeed, while I knew him, it was his constant habit. No doubt, a consciousness of this kind led him to his investigations of pulmonary disease; nor do we find him singular in this respect, for some of the best monographic

works we possess were written by authors subject to the diseases they have described. Thus, Corvisart died of disease of the heart; Bayle of an affection of the lungs; Sir John Floyer was himself the subject of asthma. The best work written upon asthma in the English language, is by Dr. Bree, himself an asthmatic. These instances might be greatly extended.

Thus, if I be not accused of using language somewhat too ambitious, I would say that Laennec fell a victim upon his own altar; that he suffered the lot of mortality from one of those diseases, the description of which has conferred upon him a crown of intellectual immortality.

LECTURE V.

THEORY OF THE SIGNS OF DISEASES OF THE
LUNGS — (*continued*).

AUSCULTATION.

Auscultation — Principles of — Instruments — Immediate Auscultation — Position of Patient — Application of Stethoscope. — Table.

Auscultation of the *Voice* — Theory of the Voice — Pectoriloquy, Causes of — Pectoriloquy, perfect, imperfect, and doubtful — Cause of — Voice in Trachea and Larynx — Voice in Bronchial Tubes — Bronchophony, Causes of. — Voice in Pulmonary Tissue. — Auscultation of *Respiratory Murmur* — In Pulmonary Tissue — Nature of — Varieties in different Ages and Temperaments. — *Puerile Respiration* — Inferences from the Presence and Absence of this Sound — Murmur in Trachea and Bronchi. — *Bronchial Respiration*, Causes of. — *Ronchus Mucosus* in Excavations — Cavernous Cough. — *Blowing Respiration* — Causes of. — “*Souffle Voilé*,” Causes of. — *Bourdonnement Amphorique*, Causes of. — *Rhoncus* — Definition — *Sibilans* — Causes — *Gravis* — Causes — *Mucosus* — Causes — *Crepitans*, humid and dry — Causes. — *Ægophony* — Nature of Sound — Causes. — *Metallic Tinkling* — Nature of Sound — Causes. — *Bourdonnement Amphorique* — Nature of — Causes.

THE subject to which I shall now call your attention is Auscultation.

Object. — What is the object of auscultation? To determine the specific lesions of structure either of the lungs or heart. We shall first speak of it in relation to the lungs.

Principles on which auscultation is founded. — What are the principles on which auscultation is founded? There are two: first, the lungs, during the acts of inspiration and expiration, produce certain sounds in the healthy and in most of their

diseased states, which sounds are inaudible through the medium of the atmospheric air. The second principle is, that, as solid bodies have the power of conducting sounds better than the atmospheric air, so if a solid body be applied upon the parietes of the chest, those sounds can then be most distinctly heard.

Instruments employed. — Various solid bodies have been used for the purpose of auscultation. The first instrument was made by Laennec at the moment, and consisted of a quire of paper rolled tightly. It is a good conducting body, and would form a fair substitute on occasion for the ordinary stethoscope. The next form was the stethoscope, also invented by Laennec. It consists of a cylinder of wood, a foot in length, and about an inch in diameter, perforated by a canal of four lines in breadth. It is hollowed out at one extremity into a funnel-like form, for the purpose of distinguishing delicate sounds. The funnel is fitted with an obturator, which added, the instrument may be used for the louder sounds, and for examining the movements, &c. of the heart. The stethoscope is divided into two parts, which may be united by this screw, merely for the convenience of carriage. Other forms have been invented, which are still more convenient. The instrument I commonly use is the one invented by Piorry. It is lighter than Laennec's, and answers the purpose equally well. You perceive that this is hollowed into a funnel-form at one end; an obturator is attached to it also, which is usually surmounted by the plessimeter. Sometimes an additional piece is fixed, for the purpose of lengthening

it; this, however, is, I think, unnecessary. Be careful that you choose an instrument the edges of which are rounded at the extremity to be applied to the chest; for they are often made sharp, and cause pain upon application.

Various materials have been used in constructing stethoscopes, as metals, card-board; different kinds of wood, as ebony, box, &c. They are now almost universally made of cedar, which is, perhaps, the best.

Immediate auscultation. — Another instrument, and one which you have always the advantage of carrying about you, is the naked ear; and in most instances it is as good a stethoscope as you can employ. But there are some cases in which it is difficult to apply the ear; as, for instance, at the humeral extremities of the clavicles, and in the axilla. Where the patient is a female, it is a matter, too, of delicacy to use intermediate auscultation in preference.

Position of the patient. — The objects you are to have in view, in relation to the position of your patient, is, to place him in a situation convenient to yourself, and to render his muscles as tense as you can, by putting them upon the stretch. Thus, if you wish to examine anteriorly, you must place him in a chair; let him incline the head backwards, and throw his arms behind the chair: if laterally, then he should incline to one side, and place the arm of that side over the head, and you can then easily examine from the axilla downwards: if posteriorly, then the body must be bent forwards, and the arms firmly crossed in front.

Application of the stethoscope. — There are a few

points also to be attended to in the application of the instrument. It should be held as you would hold a pen ; facility must be acquired in holding it in either hand, and it must be applied firmly and steadily to the chest, otherwise a rustling noise is created, which often masks the sounds produced by the lungs. You should also apply it perpendicularly to the surface, and not at an angle ; because, in the latter case, there will only be a single point of the instrument in contact, and not the whole circumference. Take care that you do not allow it to move backwards and forwards, for, in doing so, you create sounds which obscure those arising from the lungs. It is also advisable, particularly in a thin person, to interpose something between the instrument and the chest itself. The shirt of the patient is generally sufficient ; if not, you may employ a linen cloth or a little lint. Be careful, also, not to examine a patient in a silk dress ; for silk causes a rustling sound, which will deceive you. Stuff and flannel dresses are objectionable on the same account : they occasion noises very analogous to the “*rhoncus crepitans* ;” and I have occasionally seen beginners diagnose softening tubercles beneath the clavicles, when the sounds have altogether depended upon the sort of dress worn by the patient, and the loose manner in which the auscultator has held the stethoscope. The extremity of the instrument, on which the obturator is fixed, should always be applied to the chest ; the ear, of course, upon the opposite end.

We shall first consider auscultation in reference to the voice ; secondly, to the respiratory murmur ;

thirdly, to the various sorts of wheezing or rhoncus heard in the chest ; and, fourthly, in relation to certain sounds occasioned by diseases of the pleura. Perhaps the following tabular view of the sounds will assist your recollection : —

Auscultation of	The voice in...	Excavations	Pectoriloquy . .	{ Perfect. Imperfect. Doubtful.
		Trachea and larynx. Bronchi	Bronchophony.	
		Pulmonary tissue.		
	The respira- tory murmur in	Pulmonary tissue . .	Puerile respiration.	{ Cavernous respiration. Blowing respiration. Souffle voilé. Bourdonnement amphorique.
		Trachea and larynx. Bronchi	Bronchial respiration.	
		Excavations		
	The rhonchus in	Bronchi	{ Rhonchus sibilans or sibilation. Rhonchus sonorus, grave, or deep sonorous wheezing. Rhonchus mucosus, or mucous rattle.	
		Pulmonary tissue...	Rhonchus crepitans { humid. dry.	
		The sounds in diseases of the pleura	Ægophony . .	{ Simple. Combined with bronchophony.
Metallic tinkling.				
Ascending and descending rub- bing sound.				

AUSCULTATION OF THE VOICE.

We proceed to the study of the resonance of the voice : — 1st, in tubercular cavities or excavations formed in the lungs from any other cause ; 2dly, in the trachea ; 3dly, in the bronchial tubes ; and, 4thly, in the pulmonary tissue itself.

In considering the subject, we must revert to the principles of the instrument, and give some explanation of the theory of the voice. It is an experiment familiar to us all, that, if a beam of

wood be scratched at one end with a pin, the noise may most distinctly be heard by applying the ear at the other extremity of the beam, though you might not hear it at the same distance by the intermedium of the atmospheric air; — proving incontrovertibly that solid bodies convey sounds better than the air. I shall not enter into the theory of this, because it involves other questions with which we have nothing to do, but I state it as a matter of fact.

Let us now consider some points of the theory of the voice. The voice is produced in the act of expiration; the air then passes through a tube — the trachea having at its superior part a moveable orifice, called the rima glottidis, an orifice which can contract and dilate, and consequently vary its diameter. The air, in passing through the rima glottidis, produces a sound varying in its gravity, according to the diameter of the opening: if the opening be contracted, it will be acute; if dilated, grave; and I take the liberty of calling this sound the primitive note, in the same way as I should call the sound produced at the embouchure of the flute, when the fingers are off the rest of the holes of that instrument, its primitive note.

The primitive note being thus produced, its sound passes outwards, but, in its way, becomes modified by the action of the muscles of the pharynx, the velum pendulum palati, the tongue, palate, lips, teeth, &c., to form words named guttural, lingual, labial, palatine, dental, &c., according to the parts which principally operate in producing the modification.

Pectoriloquy. — The words thus formed pass

outwards to a distance proportionate to the force employed in forming the primitive note ; but, as the trachea is always filled with air, and is in direct communication with the mouth, there is no reason why the words should not descend into that tube. If that be the case, and as solid bodies convey sounds better than atmospheric air, so a solid body applied to the trachea at the moment a person is speaking should convey the voice to the listener with greater intensity than the air at the distance of the length of the instrument used. Try the experiment, gentlemen, with each other : you will find it is so ; nay, the voice is so completely conveyed through the stethoscope, that it entirely masks that from the mouth : it is true it is more hoarse, it is not so clear ; but it is perfectly distinct : and this sound is the type of that which we shall hereafter describe under the name of *pectoriloquy*.

You may, however, readily suppose that, for a tube to resound the voice, it is necessary that it should be of a certain diameter : thus the largest reed of the instrument called the pandean pipes can be easily sounded ; that facility diminishes as we ascend in the scale to the smaller ones ; and, supposing these to diminish still farther until they became capillary tubes, no sounds could be elicited from them.

We have seen that the trachea resounds the voice : its two primitive divisions are of sufficient diameter to do so likewise ; so that if the stethoscope be applied in a thin person, upon the second or third dorsal vertebræ, the words can still be heard, although not so distinctly ; but when these pri-

mitive bronchi split into smaller branches, which plunge into the substance of the lung, the resonance becomes less and less, and that even is effaced by the respiratory murmur; and when the tubes become capillary, no sound whatever is produced by them.

The consequence that may be drawn from these premises, is, that, as the mass of the lungs consists of capillary bronchial tubes and air cells, the *voice should not* be heard at any point of the chest excepting at the second and third dorsal vertebræ of thin persons. Try the experiment, and you will find it as I have stated. We will now endeavour to explain the cause of pectoriloquy.

We shall hereafter have occasion to enter largely into the consideration of an adventitious deposit in the lung, called tubercle — the proximate cause of phthisis. Tubercles exist at first as isolated bodies, gradually increasing in size and numbers, until they unite and form solid masses: these masses soften, and are expectorated; but, as they cannot be expectorated through capillary tubes, they burst into those of a larger caliber; and it follows of course that, as this softened matter becomes evacuated, it must leave a cavity proportionate in size to the quantity of the tubercular mass: if, then, a cavity be thus left, it follows also that we have now a space of sufficient diameter to resound the voice in a spot where it *could not* have been heard before. This resonance is called *pectoriloquy*.

All causes producing excavations of the lungs will occasion pectoriloquy — as gangrene, peripneumonic abscesses, &c.

As there are great variations in the size and form

of cavities,—as their parietes vary in consistence,—as they are more or less near to the surface of the lung,—so the pectoriloquy will be more or less complete. Thus Laennec has considered this sound under the three heads of *perfect*, *imperfect*, and *doubtful*.

The pectoriloquy is *perfect* when the words are distinctly articulated through the stethoscope, and when the sign is exactly circumscribed to a spot.

The circumstances necessary to perfect pectoriloquy are, 1st, a cavity of moderate size: if the excavation be immense, the voice is lost within it. 2dly, That the cavity should be empty. 3dly, That its parietes should be firm: thus, in this preparation, where you see the excavation is lined by a membrane of almost cartilaginous density, the pectoriloquy was complete. 4thly, That the bronchial opening should be sufficiently large. 5thly, That the excavation be near the surface. Even under these favourable circumstances, the phenomenon may be lost for a time, in consequence of a momentary obstruction of the communicating bronchial tube, from sputa: let the patient cough, the secretion will be removed, and the sign restored.

The pectoriloquy is *imperfect*, when the voice is not distinctly articulated through the instrument.

The circumstances which cause this imperfection are, 1st, the cavity being multilocular; 2dly, its parietes being soft; 3dly, its incomplete evacuation; 4thly, its distance from the surface of the lung; and, finally, the smallness of the bronchial opening into it.

The pectoriloquy is *doubtful* when the voice is resounded feebly—when we cannot, except with

difficulty, distinguish it from bronchophony. We must then bring to our aid the functional and general signs to form our judgment.

Voice in Trachea and Larynx. — The voice, as I before stated, in speaking of the theory of pectoriloquy, is perfectly articulated through the stethoscope when it passes from these organs.

Voice in Bronchial Tubes — Bronchophony. — The two primary bronchial divisions yield articulated sounds over the second and third dorsal vertebræ, in very thin persons. A very strong resonance is also often heard at the upper extremity of the sternum, from the presence of the trachea beneath it; and, as Laënnec observes, you should be careful not to confound it with a doubtful pectoriloquy: but, in fact, it is often impossible to distinguish them.

The larger bronchial tubes, as they plunge into the substance of the lungs, produce no resonance; no doubt, because the air contained in the cells is an insufficient conductor of the sound. Thus we find that any cause tending to solidify the lung, so as to render it a better conductor, will allow the resonance of the voice in these vessels to be distinctly heard. These causes are hepatisation of the organ, extensive pulmonary apoplexy, and accumulation of tubercles. This sound has been called, by Laënnec, *bronchophony*. It differs from pectoriloquy, inasmuch as the voice does not penetrate the instrument, nor is it heard articulated; its tone is different, and the sound is more diffused.

Voice in Pulmonary Tissue. — For the reasons I have before mentioned, the voice cannot be heard in the pulmonary tissue.

RESPIRATORY MURMUR.

The sounds produced by the passage of the air in inspiration and expiration, or the *respiratory murmur*, should be studied in the pulmonary tissue, the trachea, the bronchi, and in pulmonary excavations.

Respiratory Murmur in the Pulmonary Tissue.
— If you apply the stethoscope, or the naked ear to the chest, particularly of a child of six or seven years of age, you will there hear distinctly, during expiration and inspiration, a perfectly soft murmur. It is impossible to convey by words the nature of sounds, for these are sensations that must be acquired by and for yourselves. I can only speak analogically, and say a sound is like this or that; for simple sensations admit of no definition. In the case under consideration, you hear a slight noise both in the act of expiration and inspiration — something like the soft murmur of a pair of bellows, in which the valve does not click. Laënnec has well expressed it, by saying that it is like the sound produced by the occasional deep inspirations made by a person profoundly sleeping. Unless you obtain a knowledge of this normal sound, you will make no progress in auscultation. The respiratory murmur has nearly the same intensity over every part of the chest: however, it is less loud upon the scapulæ and vertebræ, because of the intervening bones, and most distinct in the axillæ, where there is the least mass of interposing muscles.

It is important, here, as in percussion, to know whether the sounds are alike at all periods of life. They are not; for they are infinitely more audible

in the earlier periods than in old age, and for the same reason I have mentioned before; namely, that digestion, nutrition, circulation, and consequently respiration, are more active in children than in grown persons. It is on this account that I recommend you to study the sound first in a child.

Differences, also, in the intensity of the respiratory murmur occur in persons of the same age: thus you will find in some it is very indistinct, in others very loud — so loud, that it is like that of a child, and has been consequently called *puerile* respiration. The latter particularly obtains in women, and in men of nervous temperaments. This important consequence follows,—that, for the purpose of detecting disease, you should not compare the respiratory murmur of two individuals, but you should compare it in the different parts of the chest of the same person.

Some few practical points are necessary to attend to in examining a person for this sound. In the first place, you should use the stethoscope without its obturator; and, although the murmur may be heard through the clothes of the patient, yet the fewer they have on the better: indeed, if they be of silk, or certain sorts of stuffs, it is impossible to hear so delicate a sound at all. Sometimes, too, the patient is alarmed at the examination — especially if it be the first; they will suspend, interrupt, or precipitate their respiration. Wait a few moments patiently; desire them to breathe naturally, without effort, and then you will, commonly, hear the murmur very well. If it be not distinct, tell them to make a deep inspir-

ation, or sigh. I often find that the inspiration following a cough gives the murmur better, let that cough be voluntary or involuntary. No act of volition on the part of the patient can increase the sound to the puerile character.

What are the inferences we are to draw from this respiratory murmur? Whenever it is heard in a given portion of the lung, you may be certain that there the organ is permeable to the air, and not diseased; but whenever it is not heard, you may be then as certain that the lung is impermeable and diseased. The causes of the impermeability of the lungs to air, are various: it is impossible I can detail them now, as I should too much anticipate my subject; but the most common are, hepatisation, apoplexy of the lung, the accumulation of masses of crude tubercles, and effusions into the pleura, compressing the viscus.

It is important to observe, that, whenever the respiratory murmur is absent to any considerable extent, the energy of the respiration increases in the healthy parts of the lungs, and the murmur there puts on the character of that of childhood, or it becomes *puerile*; and you draw the inference, that, whenever the puerile respiration is heard, there is disease in some other part of the organ. This is a rule having but one exception, which we shall speak of in a future lecture.

Respiratory Murmur in Trachea and Bronchial Tubes. — If the stethoscope be applied to the trachea, or to the sides of the neck, a loud sound is heard during respiration; the same may be distinguished, although not with equal intensity, in thin persons, at the upper part of the sternum and

at the root of the lungs ; but when the bronchial tubes divide, and pass into the substance of the viscus, this sound is no longer to be heard, in consequence of the indifferent capacity of the air in the air-cells to conduct it, and from its being masked also by the respiratory murmur of the pulmonary tissue.

Bronchial Respiration. — But when a portion of the lungs becomes solidified by hepatisation, or by the presence of tubercles in masses, then, as no respiratory murmur is caused by the impermeable air-cells, and as the solid organ is a better conductor of sound, the respiration is distinctly to be heard in the bronchial tubes ; and, of course, the sounds are loudest where these vessels are nearest the surface. They are, therefore, of the greatest intensity at the root of the lungs and at the points corresponding to the upper lobes, at the anterior superior parts of the chest, and in the axillæ. This sound is called the *bronchial respiration*.

Respiration in excavations, or cavernous respiration. — The sounds produced by the respiration in cavities formed either by tubercles, peripneumonia, or gangrene, are very loud, and of the bronchial character ; but they evidently give to the ear the feeling of a resonance in a capacity of much larger dimensions than that of any bronchial tube : these sounds may be considerably increased by desiring the patient to cough. Nothing can be more decisive of the presence of an excavation than the noises produced in this manner.

Blowing respiration. — This is a modification of the bronchial and cavernous respirations, in which,

during inspiration, the patient seems to draw the air from the ear of the observer through the stethoscope, and blow it back again with force during expectoration. This takes place during quick respirations, or when the patient coughs or speaks.

The physical causes of this phenomenon are, —
1. The presence of an excavation, which approaches closely to the walls of the chest, in consequence of the thinness of the parietes of that excavation; this I have noticed frequently. 2. Laënnec says that the same sound is produced from a large bronchial tube near the surface, being surrounded by condensed substance, as when the lung is consolidated from peripneumonia, or compressed by pleuritic effusion. I am but little inclined to doubt Laënnec's correctness; but I confess I have often sought for the sound from this cause, but hitherto without effect.

Souffle voilé. — Laënnec also describes a modification of the blowing respiration, in which a moveable veil seems to be interposed between a pulmonary cavity, and the ear of the auscultator. This is heard during coughing, speaking, or respiring.

The causes of the *souffle voilé* are, according to the same author, — 1. A superficial cavity, with thin and supple parietes, not adhering to the costal pleura; so that whenever the patient speaks, coughs, or inspires, the loose and thin portion of the walls of the excavation flaps against the ribs. 2. A peripneumonic abscess, whose parietes are generally hepatised, but presenting here and there some points of engorgement; and, 3. Where bronchophony is given by a large bronchial tube in

peripneumony, part of the course of that tube having the lung between it and the ear of the examiner, in a healthy state, or only slightly engorged.

Bourdonnement amphorique. — Finally, you occasionally see an immense cavity, in which scarcely any of the lung is left, except a portion at the root, and a thin layer of its substance adhering to the costal pleura. Here pectoriloquy is lost; the cavity is too large to admit of it, but the respiration puts on a peculiar character. It is no longer a cavernous respiration, but something like a sound produced by blowing into a large empty bottle, or like the buzzing of a bee in a vase, whence Laënnec has termed it *Bourdonnement amphorique*.

RHONCHUS.

We now proceed to the description of the various sounds known under the general appellation of Rhonchus.

Definition. — All noises which may be made during the respiratory act, produced by the air traversing fluids, or by a partial contraction of the aërian tubes; they accompany, and are rendered more evident by, coughing. There are various species of this sound.

Rhonchus sibilans, or whistling sound: its characters are various; it is sometimes a prolonged whistle, of divers tones; sometimes of very short duration, and like the chirruping of birds: it is occasionally similar to the clicking sound produced by the opening and shutting of a small valve.

Causes. — It appears to depend upon a small

quantity of viscid tenacious mucus obstructing more or less the smaller bronchial ramifications, or from a swelling of the internal membrane of a bronchial branch of small or middle size ; the first cause produces the clicking noise, and occasionally the sibilation, either of which may be made frequently to disappear by desiring the patient to cough, by which the obstructing mucus is dislodged. The permanent whistle depends upon a thickened state of the mucous membrane.

Rhonchus sonorus gravis, or dry sonorous wheezing. The sound in this case is grave, and often very loud — frequently like that of snoring, or of the bass string of a violoncello when rubbed by the finger : it is often similar to the cooing of a dove.

Causes. — This sound is occasioned by some change in the form of the bronchial tubes, as their compression by a tumor or engorged gland, or by a local and slight inflammation of the pulmonary tissue ; or it may be produced by the presence of very tenacious mucus ; and, finally, from a swelling of the mucous membrane at the commencement or opening of a bronchial tube, whereby its diameter is rendered smaller at its orifice than in the rest of its course. The two last causes are the most common.

Rhonchus mucosus is a sound like that produced by the blowing of bubbles in a solution of soap in water. The ear can recognise most accurately the various sizes of these bullæ, and the tenacity of the mucosities in which they are formed. Their quantity and their situation in the lung can easily be detected by a slight habitude : in fact, this sound

is so very distinct, that I find it usually the first distinguished by the student.

Causes.—The rhonchus mucosus is produced by the air passing through fluids accumulated in the trachea or bronchi, or through purulent or softened tubercular matter. Thus it occurs in catarrhs, where the secretion is considerable, or in hæmoptysis, when blood obstructs the air passages; it exists also in tubercular excavations, containing a certain quantity of mucus or softened tubercular matter; it is found also in gangrenous or peripneumonic abscesses, when they burst into one or more bronchial tubes, so that the air communicates freely with their cavities; but in these three last cases this sound is modified in its character; the bullæ are larger; they are evidently contained in a more circumscribed space; a cough, or a forced inspiration, conveys to the ear the sensation of the rhonchus being formed in an excavation in the lung. The loudness of the rhonchus mucosus, and the largeness of the bullæ, would then, perhaps, be better expressed by the term gurgling than any other,—a term equivalent to the "*gargouillement*" of Laënnec.

This gurgling sound is occasionally distinguished by the patient himself, and he will point to the situation of the cavity as being the part of the lung from whence the expectoration proceeds: this is, however, somewhat rare; but I have met with it. Laënnec describes instances in which the pulsation of the subclavian artery produced a slight mucous rattle, from the vessel striking the parietes of the excavation. This is certainly a very rare circumstance; I cannot say I have observed it.

This rhonchus may occasionally be heard at a distance from the chest, or, which is more frequent, be felt by its communicating its vibrations to the hand; thus, as in this preparation, the tubercular matter found its way through the parietes of the chest, formed a tumor which, as you perceive, has burst externally; this tumor gave upon pressure a gurgling sound. A second case mentioned by Laënnec is, where a tubercular excavation bursts through the pleura, whose two laminæ have been previously united by an abundant cellular tissue, yet sufficiently loose to allow of the infiltration of air and pus within it. A third case, is of an extensive multilocular cavity, half full of pus or softened tubercular matter; this also occasionally produces a rhonchus sensible to the hand. He had not sufficiently verified this sign. I have no hesitation in saying, that I have distinguished it now frequently.

The intensity of the mucous rattle varies, of course, according to the quantity of fluid which the air traverses, according to the force with which a patient is made to respire or cough, and the distance at which the accumulated secretion is placed from the ear of the observer, so that the sound may be *distinct* or *obscure*; these differences are very easily determinable by practice.

Rhonchus crepitans. — There are two varieties of this sound, the one described by Laënnec as the humid crepitation, the other as the dry.

The humid crepitation. — This is very analogous to the sound produced by the crepitation of salt exposed to a moderate heat, or to that made by the insufflation of a dried bladder, or still more to

the sensation given to the hand when compressing a healthy lung; it carries with it also a distinct feeling of humidity.

Causes. — Whenever air and fluid coexist in the ærian cells, humid crepitation may be heard; thus it is the pathognomonic sign of the first stage of peripneumonia, or inflammatory engorgement of the lung. It is very distinct when that organ becomes œdomatous; it is also occasionally found in hæmoptysis. It is a sound of great practical importance, and deserves your fullest attention.

The dry crepitation, or crackling sound, is heard principally during inspiration. This sound communicates to the ear the sensation of dryness and unequal dilatation of the pulmonary cells; it is very like the noise produced by blowing into a dried bladder, and the bullæ of the crepitations are much larger than in the preceding species.

Causes. — The dry crepitation is the pathognomonic sign of vesicular and interlobular emphysema of the lungs, particularly the latter.

ÆGOPHONY.

This sound may be easily confounded with pectoriloquy and bronchophony, and consequently requires the greatest attention on the part of the student.

Laënnec gives the following description of its characters:—Simple ægophony consists in a particular resonance of the voice, accompanying or following the articulation of the words. The voice appears more acute and silvery, and trembles upon the surface of the lung; it is rather like an echo,

repeating the words and final sounds, than the voice itself. It is rarely introduced into the tube of the stethoscope, and still less frequently traverses it completely. It has another constant character; it is trembling and saccaded, like that of a goat, and its tone is very similar to that animal's. When the œgophony occurs near a large bronchial tube, particularly at the root of the lungs, then a bronchophony more or less marked is generally superadded; and this combination gives rise to various modifications of sound. Thus it may be like the voice transmitted through a metallic speaking trumpet, or a broken reed, or a comb, or like a person speaking with a piece of money placed between the teeth and lips; and, finally, it is often exactly similar to that of the personage familiar to us under the denomination of Punch.

To hear this sound distinctly, the stethoscope should be applied firmly upon the parietes of the chest, and the ear lightly upon the instrument.

Causes. — It exists only in those cases in which there is a slight effusion into the chest, either from an attack of acute or chronic pleurisy, or hydro-thorax; and in proportion as the effusion increases, the œgophony disappears; as the fluid is absorbed or evacuated, the sign comes on again, evincing that it depends upon the presence of a thin layer of fluid between the pleuræ.

The œgophony is never heard in a single point, like the pectoriloquy, but is always more diffused; thus it is commonly heard between the base of the scapula and the corresponding part of the vertebral

column, in the whole contour of the inferior angle of that bone, and in a zone of three fingers' breadth, extending from the middle of the scapula to the nipple. Occasionally, however, the sound may be heard all over the side affected; it then evidently depends upon adhesions between the two pleuræ, preventing the lungs receding from the ribs, adhesions which admit, nevertheless, of an insinuation of fluid sufficient to produce the phenomenon.

Œgophony seems, then, to depend upon the natural resonance of the voice in the bronchi, transmitted by the intermedium of a thin and trembling layer of effused fluid, and rendered more sensible by the compression of the pulmonary tissue, by which that tissue becomes a better conductor of sound.

It is at the upper portion, or edge of the layer of fluid, that the œgophony is most distinct: thus the extent of the sound above described supposes the patient to be seated; but if he be placed on the face, then the fluid will gravitate towards the sternum, and the sign will almost entirely disappear from the space between the base of the scapula and the vertebral column, and will be heard at the side only, and in a direction transverse to the ribs. It will be found, too, that as the absorption of the fluid continues, the œgophony leaves the upper part of the chest, and is heard lower down, following evidently the course of the fluid.

The œgophony may be always considered as a favourable sign, because it proves that the effusion of fluid is but slight: its persistance many days, after the acute period of the disease, demonstrates that it does not increase in quantity, and,

generally speaking, shows us that the affection will not become chronic.

The œgophony, like pectoriloquy, may be sometimes suspended, when the bronchial tubes are obstructed by accumulated secretions, preventing the voice descending through them; but, as catarrh does not very commonly co-exist with pleurisy, the sign rarely ceases suddenly.

The argentine and trembling tones of œgophony are generally most perfect at the anterior and lateral parts of the chest; for when the phenomenon occurs between the base of the scapula and the vertebral column, it is almost always joined with the natural bronchophony, in consequence of the bronchial tubes being there of a larger size. It is in the latter part that the voice occasionally completely traverses the stethoscope.

Ægophony and bronchophony are combined in cases of pleuro-peripneumonia.

Ægophony, bronchophony, and pectoriloquy, may be all united in cases of pleuro-peripneumonia, combined with abscess of the lung.

The theory of œgophonism is not yet well understood: in all probability it depends upon a partial flattening of the bronchial tubes by the compression of the effused fluid, and that the voice is modified in passing through them in that state, and subsequently in passing through the secretion itself.

METALLIC TINKLING.

Laënnec so denominates this sound from its analogy to that produced by slightly striking with

a pin a cup of metal, glass, or porcelain, or by dropping a few grains of sand into such vessels. The sound is heard during respiration, coughing, or speaking; the sound is feeblest, however, during respiration, loudest when the patient coughs: it sometimes happens that it is heard only during the respiratory act, and not at all while coughing or speaking; but this is a rare circumstance.

Causes. — The metallic tinkling is caused by the resonance of the air, agitated upon the surface of a liquid contained in a preternatural cavity formed within the chest. It occurs, therefore, in two cases: — 1. In pneumo-thorax, combined with effusion. 2. In a large tubercular excavation, containing a certain quantity of very liquid pus.

In cases of pneumo-thorax, combined with effusion of the cavity in the pleura, Laënnec asserts that it is necessary for the production of the metallic tinkling that there should exist a fistulous communication between the pleura and bronchi, such as occurs in tubercular or peripneumonic abscesses, or from gangrenous eschars opening at once into both; that it constantly happens under these circumstances, and in the greatest perfection, is incontestible; but that such a communication is essentially necessary I doubt, inasmuch as I have, in a great many cases in which the metallic sound was most distinct, endeavoured to discover, post-mortem, a fistulous orifice without success. We shall revert to this subject when we arrive at the description of the specific diseases of the lungs, in which this sign may be heard.

FROTTEMENT ASCENDANT AND DESCENDANT.

These are sounds so called by Laënnec from their causing, during inspiration and expiration, a sensation to the ear as if a bone were rubbed by the finger, and heard through the stethoscope, giving the feeling of substances ascending and descending, and rubbing against each other with a certain degree of roughness. The same kind of sensation is also distinguishable by the hand, but infinitely more obscurely.

Causes.— This phenomenon depends, at least in the greatest number of cases, upon interlobular emphysema of the lungs, and is combined with the dry crepitating rhoncus. It is supposed, also, that the same sound might be produced by the projection of osseous, cartilaginous, tubercular, or scirrhous deposits, from the surface of the lungs or pleura ; but this has not yet been proved.

Oct 10/40

LECTURE VI.

DISEASES OF THE LUNGS.

Classification of Diseases of the Lungs — Method of describing each Disease.

Diseases of Aërian Passages — Classification of them. — *Inflammation of Aërian Mucous Membrane* — Table.

Acute Mucous Catarrh — Morbid Anatomy. — *Functional Signs* according to situation : Coryza, Cynanche, Pharyngea and Tonsillaris, Laryngitis — Moderate form, acute form — Catarrh affecting Trachea and Bronchial Tubes. — *Local Signs* : Absence of Respiratory Murmur, Rhoncus Sibilans, Rhoncus Sonorus Gravis, Rhoncus Mucosus — Variations in these sounds — Value of local signs.

HAVING thus, gentlemen, completed the theory of the signs, we now proceed to the second section of this part of the course, or to the *diseases of the lungs*.

As I stated in the introductory lecture, I shall classify these diseases in an anatomical order ; and as the respiratory organs consist of a trachea and its ramifications ; of an accumulation of air-cells, constituting their proper substance or parenchyma ; and finally, of a fine serous investing membrane, called the pleura ; so we shall successively describe their affections under the heads of, 1st, diseases of the trachea and bronchial tubes ; 2d, diseases of the parenchymatous tissue ; and 3d, diseases of the pleura. We follow here the arrangement of Laënnec, which is simple and easily understood.

In speaking of each particular disease, we shall proceed invariably in the following order : — We commence with its morbid anatomy, constantly

illustrating that part by preparations and drawings: there is a great advantage in this mode of proceeding, for you will at once become acquainted with the lesion of structure which really constitutes the disease, and you will soon find that the symptoms will thereby be easily understood, and even frequently anticipated. I know that this is not the ordinary method of teaching medicine, but that the morbid anatomy, if described at all, is generally so, after the symptoms have been mentioned; but I have found from a tolerably long experience, both in my own studies, and in instructing others, that the plan I propose is most advantageous; indeed, it is impossible to understand the local signs of pulmonary and cardiac diseases without being previously acquainted with the organic lesions which occasion them.

After describing the morbid anatomy of the particular disease, we shall then proceed to the signs by which the alterations of structure are rendered evident to our senses; by showing, 1st, the *functional signs*, or the signs afforded by the more or less altered state of the functions which the lungs perform. 2d, The *local signs*, or those which arise from a local examination of the organ. 3d, The *general signs*, or the signs which arise from changes of structure or function of one or more of the rest of the organs of the body, in consequence of the disease of the lungs.

We shall then consider the *causes*, dividing them into two classes; the first relating to the *individual*, as age, sex, temperament, idiosyncrasy, &c.; and the second, causes *external* to the individual, and acting upon him, as cold, moisture,

miasmata, contagion, &c. I shall say but little upon what has been called the *proximate cause*, as that is a subject purely speculative, and we shall have but too little time as it is to describe well-authenticated facts, without entering into vague and useless hypotheses. We shall conclude the subject by pointing out the various indications of *treatment*.

DISEASES OF THE AËRIAN PASSAGES.

You are aware, gentlemen, that the aërian passages are lined by a mucous membrane, and it is particularly to the state of this membrane that we must direct almost all your attention: we shall first consider its inflammation; 2dly, the dilatation of the bronchial tubes; 3dly, hæmorrhage from the bronchial tubes; and finally, bronchial polypi.

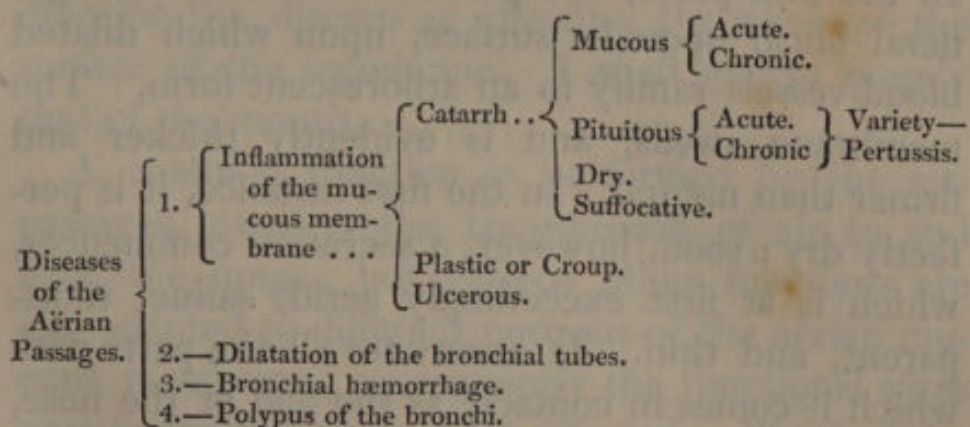
Inflammation of the Mucous Membrane of the Air-Passages.

Inflammation of the mucous membrane of the air-passages has been divided into species, from the nature of its results: thus, there may result a secretion, which does not concrete after its formation: to this the term *catarrh* is applied. There may result a secretion which instantly concretes as it exudes, and moulds itself to the form of the part affected: this is called by Laënnec *plastic inflammation*, or more commonly *croup*. Another consequence may be *ulceration* of the membrane.

Catarrhal inflammation, or that in which the

inflammation produces a secretion which does not concrete, is again subdivided, principally according to the colour of that secretion : thus, it may be yellow ; in that case it is called *mucous catarrh* ; or it may be transparent and viscous ; it is then known under the appellation of *pituitous catarrh* : each of these exists in the acute or chronic form. *Pertussis*, or whooping cough, may be considered as a variety of the latter species, at least as far as relates to the nature of the secretion. The secretion sometimes assumes a rounded and firm appearance, something like a pearl : this is named by Laënnec *dry catarrh*, not because there is no secretion, but because there is infinitely less than in the preceding species. The *suffocative catarrh* of authors, is not a distinct species, but only an excessive degree of the mucous or pituitous forms, often combined with œdema or emphysema of the lungs, and occasionally with peripneumonia.

I place before you this tabular view, which I hope will render the subject clearer.



Catarrh is the most common disease of this climate; perhaps there is not a single individual who has not been affected at one time or other with it;

and I may say still more, that perhaps not a year passes but every one is more or less troubled with this affection, and particularly in the dry form. There is scarcely an individual, though he may appear to be in a good state of health, in whom, if you apply the ear to the chest, you will not find some ronchus, some wheezing, especially in moist weather.

ACUTE MUCOUS CATARRH.

Definition. — Inflammation of the mucous membrane, having for its result a yellow secretion which does not concrete.

Morbid Anatomy. — It fortunately happens, that although this disease is so frequent, yet it is very rarely fatal, and we have therefore few opportunities of seeing the morbid condition of the membrane. However, we can readily form an idea of it from the state in which we perceive the Schneiderian membrane at the nostrils, when inflamed. In the first place, the part is red: there is a general blush upon its surface, upon which dilated blood-vessels ramify in an arborescent form. The membrane swells, and is evidently thicker and firmer than natural. In the first instance, it is perfectly dry; soon, however, a secretion commences, which is at first exceedingly acrid, saline, transparent, and thin. It excoriates all the parts with which it comes in contact, as the alæ of the nose, the lips, &c. After flowing for a certain time, varying from a few hours to a day or two, according to the intensity of the disease, it changes in its properties, loses its acrid nature, becomes more

bland, but still remains transparent. In an indefinite time, say two or three days, still farther changes take place: its bland character remains, but its colour is altered: it gradually assumes a greenish tint; it then passes to yellow, and finally becomes of a bright brimstone hue. The membrane also changes as the disease proceeds; for, as the bland fluid is formed, whether it be of a white or yellow kind, the membrane diminishes in thickness and firmness. The inflammation diminishes as the secretion increases. This is the general morbid condition of the aërian passages; particular modifications we shall speak of previous to describing the signs of each species.

Symptoms; functional. — You are of course aware, gentlemen, that the air-passages consist not only of the larynx, trachea, and bronchial tubes, but also of the cavities of the nose, the mouth, and pharynx: all these parts are lined by the same mucous membrane, modified, it is true, but subject to the same inflammation; our account of catarrh would, therefore, be incomplete, were we not to describe the disease as affecting the whole of the surface of the membrane. I shall except merely that of the mouth.

A common function is performed by the air-passages, namely, the transmission of air to and from the lungs; but various other functions are also adapted to different portions of the aërian mucous membrane, consequently the functional signs vary according to the part affected. We shall describe the symptoms in the following order: — 1st, of catarrh of the Schneiderian membrane; 2d, of

the pharynx ; 3d, of the larynx ; 4th, of the trachea and bronchial tubes.

Inflammation of the Schneiderian membrane, or coryza. — The difficulty of transmitting the air is in proportion to the swelling of the membrane of the nares : there is a general sense of fulness, uneasiness, oppression, and weight, referred to the nasal cavities, and more particularly to the bridge of the nose. If the inflammation, as is frequently the case, ascend into the frontal sinuses, the same sensations extend across the forehead, accompanied by more or less of cephalalgia ; the membrane lining the nasal ducts becomes swollen ; the affection extends to the lachrymal sac — the tears can no longer penetrate the puncta, and they flow over the cheek ; the conjunctiva often participates in the inflammation, and reddens. The sense of smell is of course disturbed, and the patient distinguishes odours with difficulty.

The membrane, as I have already described, is in the first instance dry, all secretion is suspended, and this dryness occasions an irritating, itching, and hot sensation, more particularly at the anterior opening of the nostrils. You will observe there, too, that the small hairs are covered by a fine white pulverulent deposit, from the absorption or evaporation of the thinner parts of the previously healthy secretion. Then follows the acrid fluid, which, flowing upon the upper lip and alæ nasi, excoriates and irritates them. It often passes into the posterior parts of the nares, distils, as it were, into the pharynx, upon the rima glottidis and epiglottis, and occasions a violent cough : — this fluid soon changes in its character, and becomes bland ; the

cough then diminishes, the irritation subsides, and the transparent secretion gradually assumes a yellow puriform appearance, and at last nothing remains but inspissated mucus.

Inflammation of the mucous membrane of the fauces, or cynanche pharyngea, and tonsillaris.—

Coryza is sometimes followed by, or co-exists with, inflammation of the mucous membrane lining the fauces; but that is by no means invariably the case, for these inflammations may happen perfectly independently of each other. If the pharynx be attacked, you will see that its surface is of a bluish red colour; and the young practitioner is often alarmed from the darkness of the tinge, thinking it indicative of approaching gangrene, but it arises from a greater quantity of veins existing in that part than in any other portion of the mucous surface, and the colour of these veins show through the membrane. The membrane is also swollen.

When, however, the anterior part of the fauces is affected, then the anatomical characters vary, in consequence of the amygdalæ, velum, and uvula participating in the disease; you will then see that the redness is brighter and more vivid; that all the parts are more swollen; the amygdalæ enlarge, approach, and touch each other, leaving a small triangular space below, having the upper and back part of the tongue for the base of the triangle, and a similar space above, covered by the swollen velum and uvula: abscesses of the amygdalæ frequently form; the membrane lining the Eustachian tube and the salivary glands often also inflame.

The functional signs have here a reference to three offices which the fauces have to perform:

1st, to the transmission of air ; but the swelling is rarely so great as to cause any serious impediment to its passage ; when there is any dyspnœa, it in all probability arises from the disease having extended into the rima glottidis, although I do not deny that the amygdalæ may so enlarge as to lie over and obstruct that orifice ; 2d, to the transmission of the food : and here the swelling and consequent pain occasion the greatest difficulty in swallowing, or *dysphagia* ; 3d, to the passage of air into the tympanum : this function is frequently impeded so that the patient becomes more or less deaf, and violent pains often shoot from the fauces in the direction of the Eustachian tube towards the ear ; the salivary glands occasionally inflame and swell also.

The succession of changes in the secretion is not so manifest in this case as in coryza : it is true that the inflamed surface is in the first instance dry, the acrid fluid then follows ; but when it assumes the bland character, it remains pituitous, and does not become so distinctly yellow. The quantity is often considerable, and obstructs the fauces from its viscid character.

Inflammation of the mucous membrane of the larynx, or laryngitis. — When inflammation attacks the mucous membrane of the larynx, it is called *laryngitis* ; the functional symptoms then vary considerably, according to the intensity of the disease : thus, it may appear in a moderate form, which is a very common affection, or an aggravated one, which is fortunately very rare.

Moderate form. — The functions of the larynx are to transmit the air and to form the primitive

note of the voice, so that the signs under this head refer only to these two objects. The transmission of the air is rarely obstructed, because the mucous membrane of the larynx, or rima glottidis, is but slightly affected, consequently there is no dyspnœa, or it is but very slight; but the voice alters from the commencement: it is hoarse, broken, sometimes almost lost, or the patient speaks only in a whisper; in fact, this is the common hoarseness, or cold, so constantly met with during rapid variations of temperature.

The mucous membrane is in the first instance dry: a sensation of irritation, heat, and tickling in the throat, is the consequence, and a violent cough follows, and continues until a sufficient quantity of mucus is torn from the trachea and bronchial tubes to moisten and lubricate the inflamed parts: the uneasy sensations are repeated, the cough is renewed, and often continues so long that venous congestions threaten, the face swells and becomes livid, the eyes are turgid, and the cerebral functions momentarily confused, so as to threaten apoplexy: the whole circumference of the chest, particularly at the attachments of the diaphragm, becomes tender, sore, and tired, from the reiterated coughing.

The acrid secretion soon however forms, and the cough, if possible, becomes still more aggravated, in consequence of its irritating the edges of the rima glottidis; as, however, it changes into the bland, transparent, and viscous fluid, and from thence into the yellow or mucous, all irritation gradually ceases, the expectoration becomes loose

and free, and the cough disappears, although the voice often remains hoarse for a considerable time.

It is generally observed that the cough is most troublesome at night, keeping the patient awake for hours; and you will always see it aggravated by suddenly changing the temperature, as in passing from the cold air into a warm room, or from a warm room into the cold air.

Severe Form, or acute Laryngitis. — But when the inflammation of this part of the mucous membrane is still greater, then it forms one of the most formidable and dangerous diseases to which man is subject.

You will recollect, gentlemen, that the danger of all the affections of the air-passages depends upon the degree of narrowness of their tube or tubes, and the consequent diminution of the quantity of air sent into the lungs.

The morbid anatomy of this affection is the same as in the preceding species; it differs only in the greater intensity of the inflammation. The mucous membrane lining the rima and larynx is considerably swollen and reddened; there is frequently serous effusion into its submucous cellular tissue, from whence it has been called also *œdema glottidis*: it is probable that even the cartilages participate in the inflammation. The same series of secretions occurs also here; and occasionally, although I think very rarely, abscess forms.

The epiglottis, in all the cases I have seen, is involved in the disease; its membrane reddens and swells; and the whole valve becomes erect, and is depressed with difficulty in the act of swallowing.

In these preparations, you see, gentlemen, very distinctly, the appearance which the rima, larynx, and epiglottis put on in this disease.

Signs. — The functional signs have a reference to the voice, to deglutition, to coughing, and to respiration.

The voice soon becomes hoarse, descends into a whisper, and is finally, in extreme cases, totally lost. The power of deglutition is often considerably impeded: this happens from the inflammation occasionally attacking the œsophageal face of the arytenoid cartilages, and also from the difficulty of depressing the erect and inflamed epiglottis. This latter circumstance is of great importance to attend to, as the history of the two cases from which these preparations were obtained will sufficiently exemplify. The one is the larynx of a student at St. Bartholomew's Hospital. You see its inflamed state, and particularly that the epiglottis is thickened, erect, and of diminished elasticity. You know that the function of the epiglottis is to act as a valve in closing the rima during the act of deglutition, so that the food shall not pass into the larynx; but, in the inflamed and inelastic state, this function can be but imperfectly performed. In this case an ordinary purgative draught was given to the patient, who, endeavouring to swallow it hastily, was suffocated by the effort. You see, gentlemen, that the epiglottis could hardly have acted as a valve here, and the fluid consequently entered into the narrow orifice of the larynx. The second case, which is of this preparation, was very similar to the first. You perceive here also the great swelling and redness of the dif-

ferent parts of the larynx, and of the epiglottis. This case, as you may suppose, presented all the symptoms of acute laryngitis. The patient had been most boldly treated by his medical attendant; but the symptoms became aggravated; and I determined, if a very extensive mercurial friction did not produce a good effect in a few hours, to propose the operation of bronchotomy. The wife of the patient was told of her husband's dangerous position: she pressed him to settle his affairs, and he became, for the first time, aware of his peril. He, by signs, insisted upon having some porter brought to him; he took a copious and hasty draught of it, threw himself violently upon his back, struggled, and expired.

The cough is often not so violent in the acute form of laryngitis as in the moderate; but the state of the respiration demands our utmost and most anxious attention.

As the disease progresses, the mucous membrane of the larynx becomes highly swollen; the orifice of the rima glottidis is narrowed to such a degree, that the purposes of life cannot be effected by the diminished column of air which passes through it: nature, however, makes a strong effort; she accelerates the movement; the respiratory muscles are thrown into rapid action, constituting dyspnoea from this cause. As the rima still becomes narrower, the difficulty of breathing increases; a violent burning pain, or suffocating sensation, is felt at the *pomum Adami*; the voice, which from the beginning was hoarse, is now lost; the air, in passing through the narrow orifice, produces a sound as if it passed through a brazen tube; the

face becomes livid, the eyes red; the patient is erect; he manifests the most unceasing anxiety and jactitation; points to his throat as the seat of all his distress; and dies, sometimes overpowered and exhausted from his exertions; at other times, especially if strong and robust, in a desperate and convulsive struggle, as if under the bow-string of the executioner.

Inflammation of the Mucous Membrane of the Bronchial Tubes. Bronchitis. — Having described these affections, we shall proceed to the disease as it affects the bronchial tubes.

When inflammation attacks the mucous membrane lining the trachea and bronchial tubes, the functional signs must have relation to the passage of the air only. There will be difficulty of breathing in this case, proportionate to the degree of swelling, and to the extent of the tubes affected. If only a single tube be inflamed, the dyspnœa will not be great; if it occupy the whole of the tubes on one side, the difficulty of breathing would be considerable; if it attack both lungs, it is often fatal. There is a feeling of tightness and oppression across the chest, and a burning sensation, often referred to the sternum. In the first instance the cough is dry, because the membrane is dry; then the secretion becomes acrid, and finally bland; it increases in quantity, and at last assumes the yellow colour, characterising the species; the cough now becomes looser, the expectoration free, and all the symptoms gradually subside.

Local Signs. — The local signs of inflammation of the aërian mucous membrane are sufficiently

evident, whilst the disease exists in the cavities of the nose or pharynx. The appearances described under the head of morbid anatomy constitute these signs: thus in coryza, the swelling of the membrane, its redness, firmness, and its series of secretions; in cynanche pharyngea, and tonsillaris, the swelling, the redness of the different parts, and the secretions also, are so many local signs; but, independently of these, the symptoms arising out of the lesions of their different functions would be sufficiently indicative of these affections. When the inflammation attacks the larynx, the altered condition of that organ cannot be seen, and local signs do not properly exist, and we are obliged to depend upon those arising from the derangements of its functions of forming the voice and transmitting the air.

But when the affection descends into the trachea and bronchial tubes, not only we cannot see the condition of the membrane, by which to obtain local signs; but as the functions of these tubes are now so simplified as to be mere conduits of air, so the only sign we can obtain arises from the lesion of that function or dyspnœa. It is true there is also cough and expectoration; they are, however, common to various other affections of the lungs, and are consequently not diagnostic.

Dyspnœa is common to every affection of the lungs and heart, when arrived at a certain intensity. By it alone, therefore, we cannot determine the nature of the specific lesion.

One of the most brilliant discoveries of Lëennec was that of a series of local signs, by which catarrhal inflammation of the bronchial tubes is rendered

almost as evident as any external disease of the body. We now proceed to describe them.

This inflammation has for its consequences a swelling of the mucous membrane of the bronchial tubes, and a series of secretions: the local signs, then, have a reference to these two conditions.

Absence of Respiratory Murmur. — When the mucous membrane is considerably swollen, it may completely obturate the tube affected: the consequence must be that the respiratory murmur cannot be heard in that portion of the lung which the tube supplies, since no air can pass the obstructed point; so that it frequently happens, especially in severe catarrhs, that the respiratory murmur is absent in various portions of the lungs; but as this absence of sound is common to several other affections of that organ, we call percussion to our aid; and by striking the chest, we find that the sound elicited is natural in catarrhs, whilst, in almost every other affection of the lungs, it is dull, when there is no respiratory murmur. The reason of this difference is, that in catarrh the cells are filled with air, which the obstruction confines and prevents the renewal of, and consequently occasions a good sound on percussion, although the murmur be lost; whilst, in other affections attended by absence of the respiratory murmur, the air-cells are impermeable, either from their consolidation or compression, and then the sound on percussion is invariably dull and fleshy.

Rhonchus Sibilans, or Sibilating Wheezing. — If the membrane be less swollen, and the bronchial tube not completely obturated, then the sibilating sound is heard. I refer you, for its characters, to

the lecture on Auscultation. As I there observed, it consists of a distinct whistle, sometimes a chirruping, or it is like the clicking of a small valve. With this sound, the respiratory murmur is always indistinct at the part affected, because of the diminished caliber of the tube.

Rhonchus Sonorus Gravis, or deep Sonorous Wheezing.—It has long appeared evident to me, that as the swelling of the membrane diminishes, this sound appears. It is loud, extremely distinct, like the rough scraping of the bass note of a violoncello by an inexperienced hand, or it is similar to the cooing of a dove; and its intensity is sufficient to cause a vibration upon the parietes of the chest, distinguishable by the hand. The respiratory murmur now becomes more distinct, evincing that the bronchial tubes are more patent; finally, the deep sonorous wheeze assumes a still deeper bass, merges into the respiratory murmur, mixes with it, and gives it a roughness which I have designated *rough respiration*.

Rhonchus Mucosus, or Mucous Rattle.—The mucous rattle, or wheeze, occurs when the secretion appears, and is loud and noisy in proportion to the quantity of fluid thrown out. This sound, when it is formed in the trachea, can be heard through the medium of the air alone; but the application of the stethoscope, or the ear, to the surface of the chest, is necessary when it is formed in the bronchial tubes.

It frequently happens that viscous and tenacious secretions will adhere with sufficient force to obstruct the tubes, and occasion absence of the respiratory murmur, sibilation, or the deep sonorous

wheezing; so that it would appear to be difficult to determine whether they depended upon the presence of secretions or a thickened state of the membranes: this difficulty is easily solved, by directing the patient to respire strongly, or to cough: then the air will be driven with sufficient force to remove the obstructing mucus, and the sounds will cease; the respiratory murmur will appear after a few efforts. If the sounds be occasioned by thickened membrane they will be permanent.

In examining these sounds, you must not expect to find them always precisely similar to the analogies I have presented to you: I have described only the exquisitely marked ones; for as the mucous membrane may be thickened in various degrees and in various parts—as the secretions vary in quantity and tenacity—so many modifications of these wheezings occur; but a very little practice will render them perfectly easy to distinguish.

It is hardly necessary to speak of the value of these local signs; by them you can decide at once whether the disease in question be catarrh or not; you can determine the extent of the affection; for sometimes the wheezing may be heard only in a single line, as if in the direction of a single bronchial tube; sometimes it is heard all over one lung, occasionally over both. By judging of the distance of the sound from the ear, you can tell whether the tube affected be in the centre of the organ or at its superficies; you become aware of the intensity of the inflammation by the nature of the sound, and of its variation in different parts, by

the variation of the wheezing. Thus there may be sibilation in one part, deep sonorous wheezing in another, mucous rattle in a third, or they may be intermingled. All this may appear to you now subtle and difficult; but there is not a more distinguishing organ than the ear, and I repeat my assurances that these difficulties exist only in the apprehension; even if they were greater, the subject is so important that you are bound to overcome them.

Nov: 19/40.

LECTURE VII.

DISEASES OF THE LUNGS.

CATARRH.

General Signs. — Causes. — Individual. — External. — Influenza. — A combined Disease. — Symptoms of. — Moderate Form. — Violent Form. — Supposed Causes. — Contagion.

Treatment. — Mild Form. — Acute Form. — Bleeding. — Blisters. — Expectorants. — Caution. — Cough. — Demulcents. — Treatment of Influenza. — Of acute Laryngitis. — Bleeding. — Blisters. — Mercury — Tracheotomy.

Chronic Mucous Catarrh. — Morbid Anatomy. — Secretion. — Local Signs. — Humid Asthma. — Functional and general Signs. — Suffocative Catarrh. — Disease simulating Phthisis.

Treatment. — First Indication. — To relieve inflammatory Congestion. — Second, To cause Expectoration. — Third, To give Tone.

GENERAL Signs. — We now proceed to consider the general signs of Catarrh.

At the period when the membrane commences to swell, and even sometimes before, the patient experiences a general lassitude and inaptitude to movement: he feels cold; a chilly air seems to run in an irregular manner between his skin and muscles, and this is more particularly felt in the back and loins; the sensation is at last so complete that it produces horripilation or rigor; the skin then contracts, its papillæ become elevated, forming what is called “*chaire de poule*,” or “goose skin;” the various functions of the body seem to have lost their energy, those of the brain evidently —

perception, judgment, and reasoning are confused. Sometimes there is a singular exception to this, for those faculties become exalted for a time; the pulse is rapid but weak, the urine small in quantity and limpid. These symptoms are soon followed by irregular flushes of heat, sometimes occurring at one part of the body, sometimes at another, alternating with the cold and intermingling with it, so that the patient feels frequently, in consequence of the rapidity of these changes, the two different sensations in the same place, and almost at the same instant; at last an universal glow pervades the frame; the pulse remains rapid, but becomes full, the urine is still small in quantity, but high coloured, the skin expands but remains dry; finally, a perspiration more or less copious comes on, the functions are restored, and debility alone remains. After this febrile attack there is then a remission of symptoms, to recur usually in the evening.

The other mucous surfaces of the body frequently sympathise with the bronchial mucous membrane; thus there is often slight diarrhœa, ardor urinæ, and nocturnal seminal emissions.

CAUSES.

Individual. — We now pass on to the causes; and first to those which relate to the individual. All ages, temperaments, and both sexes, are subject to catarrh; but you usually find that persons of dark complexions, dark hair, dark eyes, or of what is called the *bilious* temperament, are most subject to the mucous form, whilst the pituitous

commonly attacks those of an opposite or lymphatic habit. There are other causes relative to the individual, and to which catarrh has been attributed, but I doubt whether correctly. It has been said that the suppression of epistaxis, of hæmorrhoidal discharges, of the catamenia, and of eruptions, will produce this disease. This is a leaven of the old humoral pathology. I will not take it upon me to say that these are not occasionally causes; but I never saw a case of catarrh that could fairly be attributed to them.

There are causes which greatly predispose the individual to this affection, such as fatigue, hunger, excess of any kind, as drinking, venereal excesses, and low and humid habitations.

External. — The first and great external cause of catarrh is certainly cold, especially when combined with moisture. Cullen has exceedingly well laid down the circumstances under which cold acts. In the first place, it acts upon the body in proportion to its intensity. Secondly, according to its duration: if a person be exposed to cold for a long time, he will be more liable to its effects than if he be exposed only for a short period. Thirdly, according to the direction of the air on the body: it is well known that if it be directed in a current to a particular part, its prejudicial effect will be felt sooner than if the body generally be exposed to it. The most powerful circumstances, however, in inducing disease, are the variations from cold to warmth, and from warmth to cold.

There are other causes which may be said to be chemical, and which seem to act directly by irritation, viz. the inhalation of certain gases, as of

chlorine and of sulphureous acid gas. There is, however, this remarkable difference between catarrh thus induced, and that arising from cold, that the former is shorter in its duration.

Epidemic Catarrh, or Influenza.—Catarrh occasionally assumes an epidemic character, and spreads widely over various regions of the earth. It occurred in the days of Hippocrates; it has occurred also in modern times, and doubtless in the intermediate periods: there happened five instances of it in the sixteenth century, one in the seventeenth, nine in the eighteenth, and three in the present. This form of the disease has received a variety of names: in France it is called the *Grippe*, the *Follette*, *Baraquette*, *Petite Poste*; in Italy, the *Influenza*, which latter name we have adopted.

It is impossible to form an accurate judgment, from the works of the old authors, as to what really was the nature of the epidemic which they called catarrhal: that the symptoms of catarrh predominated there can be no doubt, but it is extremely probable that they were combined with those of peripneumonia, or inflammation of the substance of the lungs. In the epidemic of 1782, Dr. Smith describes, besides the cough and dyspnoea, the following symptoms. It commenced with a great languor and oppression of the precordia, sighing, sickness, and great headache. The pulse was quick and irregular, and there was often nocturnal delirium; the skin was moist, with a tendency to perspire; the tongue white or yellow, muscular pains, erysipelatous efflorescence, sometimes ending in gangrene and death. The greatest danger was in the first forty-eight hours, and it attacked particu-

larly those who were healthy and robust. As auscultation was not then known, and morbid anatomy was but little cultivated, it is impossible for us to ascertain whether the epidemic was simple catarrh, or a combination of it with pleurisy or peripneumonia; but I should think that in all probability it was the latter.

The epidemic of last year occurred in two forms. In the first, it prevailed in the form of common catarrh of various degrees of intensity: the second form was evidently a combination of catarrh and peripneumonia. I did not meet with a single severe case in which there was not crepitation in the inferior lobes of the lungs; the disease was, in fact, the peripneumonia notha of old authors: the *post-mortem* examinations proved it, for the bronchial tubes were filled with secretions, and the substance of the lungs was engorged with a sero-sanguinolent fluid; here and there hepatisation was apparent; but the disease generally destroyed the patient before extensive consolidation of the lungs took place: in old persons the engorging fluid was less red than in the young, the affection seeming to take an intermediate character between peripneumonia and œdema pulmonum.

The general symptoms of the *Influenza* differed from ordinary catarrh in the greater degree of violence with which they commenced, the febrile symptoms being commonly of a low or asthenic type, although they occasionally put on an exquisitely sthenic form: in the severe cases, the dyspnœa became extreme, the cough troublesome, and the expectoration difficult; frequently there were violent rheumatic pains, particularly in the

respiratory muscles: the local signs consisted of wheezing of all kinds, sibilating, sonorous, and mucous; and I invariably found crepitation in one or other lung, indicating effusion into the cells: congestion, more especially in the brain, soon came on, in the young, generally inducing delirium; in the old, a muttering coma, from which they could be aroused but with difficulty. These two last signs indicated a fatal termination.

What is the cause of this disease, and of its extensive prevalence? This is a question that has occupied the attention of medical philosophers from the period of Hippocrates down to the present time, but I cannot enter here into any elaborate details upon the subject. Hippocrates attributed it to Divine agency: others have supposed it to depend upon a certain condition of the electric state of the atmosphere; and others have attributed it to terrestrial emanations, particularly Sydenham. That certain diseases are caused by miasmata arising from the earth, there can be no doubt: we have the evidence of it in intermittent and remittent fevers; but these miasmata do not act far from the situation in which they are produced, whereas influenza prevailed over hill and dale, mountain and valley, over all countries, and did not seem to depend upon local causes. Epidemic catarrh sometimes took the course of the prevailing winds; but, like the cholera of India, it frequently travelled in the very teeth of them. In this country, the epidemic appeared in weather particularly favourable to the production of ordinary catarrh; but in other places it was most violent in fine weather. That there exists a cause is certain:

it probably arises from some modification of the atmosphere, but this is not yet proved. It is best therefore to avow, what all who have thought upon the subject must feel, that we are perfectly ignorant of its nature.

Another question that has arisen is, whether the disease can be propagated by contagion? We have no proofs that it can, though it was formerly supposed that it might be so extended. The following story is told by Dr. Hugh Smith regarding the epidemic of 1782:—A soldier went on a furlough to St. Alban's; the disease had not previously been felt there, but it occurred after his arrival. One instance, however, is not sufficient to prove a general rule, and I think we have no cases which decidedly show that the influenza was contagious.

Treatment. — We must now consider the treatment of catarrh. The disease in its mild form requires very little attention; it is usually sufficient to place the patient in bed in a warm room, and give demulcents, to determine to the surface, and in a few days the disease subsides. I generally give, for the purpose of inducing perspiration, the liq. amm. acetatis, combined with small doses of Dover's powder and camphor mixture. The patient usually soon recovers his strength, and any ordinary tonic, given after the febrile symptoms have subsided, may be then administered.

But if the disease be violent, if there be much dyspnœa, much oppression at the chest, and the cough is very irritating, then it may be necessary to diminish the powers of the system by venæsection, or by leeching. The quantity of blood abstracted must be proportionate to the strength of

the patient, and to the intensity of the disease: it is impossible to give any other general rule. Blisters are sometimes employed; but I should recommend you not to use them during the febrile state, for they produce considerable irritation, and add to the disturbance of the individual, without being of any benefit. But when the dyspnœa or oppression continues, or if the expectoration be difficult, then they are of great advantage. Another indication is to facilitate expectoration, and for this purpose ipecacuanha is with me a very favourite remedy. It may be given in doses of one grain every four hours. Tartar emetic has been exhibited with the same view, but I think ipecacuanha is better; it is not so liable to purge or depress. On the continent it is the custom to give emetics, and they are very useful, particularly to young children, who, although they expectorate, do not spit out the secretion, but swallow it, and the result is, that the stomach becomes disordered. In adults, squills and ammoniacum are frequently of great service; but you must beware of exhibiting them to children or persons of irritable fibre, or when the disease still exists in the acute form: they are best exhibited towards the termination of the catarrh, when it tends to become chronic, and when the secreted matters are viscous, tenacious, and, consequently, difficult of expulsion.

One of the most teasing symptoms is cough; it commonly disturbs the patient far more than any thing else. The cough, as I have already said, is first caused by the dryness of the passages, and then by the acrid secretion. To obviate this, you may give demulcents, as oily emulsions, barley-

water, linseed-tea, &c. : they seem to act mechanically, by forming an artificial mucosity, which lubricates the parts; sipping even water for a certain time will relieve the irritation, and free the patient for some hours. Sometimes the cough is so violent that the patient cannot obtain any rest, and opium then, especially after the febrile symptoms are over, is one of the most valuable remedies we possess: it acts like a charm, and gives time for a proper secretion to form.

As to the treatment of epidemic catarrh, or influenza, we have only the experience of the past year to guide us, and it was found that the moderate form required very little treatment at all; it was merely necessary to put the patient in bed, and give demulcents. In the acute, the treatment then became a matter of serious importance. Patients did not bear depletion generally, although there were exceptions to this rule, in the young and plethoric especially. Finding that there was always crepitation in the inferior lobes of the lungs, it appeared to me that mercury was one of the most proper remedies to be used, not only to subdue the inflammatory action, but to occasion absorption of the fluid effused into the air cells. I will mention one instance, strikingly illustrative of the advantageous effects of this mode of treatment. It happened to be my duty to admit the patients into this hospital, one week during the period when the epidemic was at its height: expecting a great many applications, the Committee placed at my disposal thirty-two beds, and these were all soon filled with individuals labouring under the severe forms of influenza—so severe, that I firmly believe, if these

poor people had been allowed to wander about the streets, or to remain at their own homes, with the insufficient attention they could there have obtained, they would for the most part have perished. If I recollect rightly, only one or two of these patients were bled, but they were all placed under the influence of mercury. This treatment commenced on Thursday, and by Saturday night all who were affected in the usual way by the remedy, safely and ultimately recovered, with the exception of two, and one of those had hypertrophy of the heart, with diseased aortic valves.

I should state that I never, if I could avoid it, carried on my plan to salivation, but merely rendered the mouth tender, and kept it so for a week or two. It was necessary that the action of the mercury should be prompt, and we found that the most quick and efficacious means of effecting it was the rubbing in the *linimentum hydrargyri*.

The common people have a mode of treating catarrh which is occasionally successful: it frequently destroys the disease at once; but if it does not produce that effect, it will do great mischief, and perhaps destroy the patient. It is by taking wines, spices, and things of that description. The good effect of these means is produced by inducing copious perspiration; but these remedies are pregnant with high danger, if the patient be young and plethoric, and of firm fibre, or if there be dyspnoea, heat and oppression of the chest, and hard dry cough. I may here remark, in passing, that there is scarcely an opinion entertained by the people at large as to the mode of treating disease, however erroneous it may be, which does not owe its origin

to some old and exploded theory. This is the mode proposed by Van Helmont, but has long been disused by medical men.

Treatment of Acute Laryngitis. — The principles upon which the treatment of acute laryngitis should be founded are similar to those of any other severe catarrh; the difference is only in the promptitude and activity with which the remedies should be administered. You will recollect, gentlemen, that the danger, and imminent indeed it is, depends upon the degree of swelling of the mucous membrane lining the rima glottidis; the more it is swollen the narrower the orifice becomes, and the smaller the column of air.

The first remedy to be used is general bleeding, and that should be carried to an extent proportionate to the intensity of the symptoms and the powers of the patient. If after bleeding, perhaps once or twice repeated, the symptoms are only slightly diminished, then leeches or cupping may prove of service; and here I may recommend you to follow the advice given by Dr. Farre, in his excellent lectures upon this subject, which is, not to apply leeches over the external surface of the larynx, but rather along the line of the clavicles of the neck, as they often induce an œdematous state of the skin or ecchymoses, which occasion a stiffness of the parts, and add to the suffocating feelings of the patient. Blisters may also be applied upon the chest, or nape of the neck, but not over the inflamed part, and for similar reasons to those above stated; indeed, there is another reason why blisters should not be applied upon the throat, which is, that if tracheotomy be necessary, you

would then have to cut through the inflamed skin, by which the operation would be rendered more difficult.

There is no remedy of more importance and utility in this case than mercury: it is useless to employ it in small doses, for its effects must be produced rapidly — often, if possible, within a few hours: if given internally to an adult, not less than three or four grains of calomel should be administered every two hours until the mouth be rendered tender: but I do not depend upon it alone; I always direct a considerable quantity, as one or two ounces of ung. hydrargyri fortius, or the linim. hydrargyri, to be rubbed in for an hour or two, and to be repeated in three or four hours, using it without limitation until the patient is relieved. You must not be afraid of the remedy; and you will often be rewarded by seeing a permanent relief afforded as soon as the mouth is affected.

But it sometimes happens that even mercury is insufficient, or that you may be called in so late that there is no time to employ it: you perceive your patient convulsed with distress — suffocating — struggling with death — and not a moment is to be lost; then, gentlemen, the only means you possess is to establish a communication between the trachea and the air, by forming an opening below the inflamed part: this is done by the operation of tracheotomy, for the description of which I refer you to your surgical works.

CHRONIC MUCOUS CATARRH.

Chronic mucous catarrh is the common infirmity of old age ; it not infrequently, however, occurs in children, particularly as a consequence of whooping cough. This disease may persist for many years.

Morbid Anatomy. — The mucous membrane is in this case usually of a pale violet colour, in patches : sometimes in old persons it has been seen very pale, or of a yellowish tint, mixed here and there with red. Dilatation of the bronchial tubes frequently accompanies chronic mucous catarrh.

The secretion is generally of a less viscous quality than in the acute form, but more opaque ; its colour is yellow, varying to grey or green ; it is sometimes clouded with a dark tint, supposed to arise from a mixture of black pulmonary matter ; it frequently happens that the yellow sputa are seen to float in a transparent viscous, or pituitous fluid : these sputa are commonly diffuent, but occasionally of an irregular round form ; the secretion is usually inodorous, sometimes it has the smell of pus, or the discharge from a blister, or even that of gangrene of the lung : the quantity of the expectoration varies of course according to the extent of the disease : there have been thrown up as much as two or three pints in twenty-four hours.

Local Signs. — The mucous rattle is one of the most common sounds heard in this disease, and to an extent proportionate to the tubes affected ; the deep sonorous wheeze, and sibilation even, may be distinguished when the mucus is tenacious ; but

these latter sounds are not permanent, for a strong act or two of respiration, or a cough, will remove the secretions, and occasion these noises to cease: there is rarely a total suspension of the respiratory murmur, as in the acute mucous catarrh; but sometimes the necessity for respiration is so increased, that the puerile respiratory murmur may be heard nearly all over the chest. It then constitutes one of the forms of the disease we shall hereafter describe under the name of *Humid Asthma*.

Functional and General Signs. — Laennec describes, with the most perfect accuracy, the symptoms and course of this affection, in the following manner: —

“ This disease is most commonly consequent upon severe acute mucous catarrh. The fever ceases without any diminution of the cough or expectoration, or changes into a slow fever hardly distinguishable, except in the evening. The patient recovers his strength and appetite, but generally remains paler, and becomes thinner: in a state of repose, he breathes easily; but during exercise, with difficulty: sometimes after having lasted for many months, or even a year or two, it gradually disappears, and leaves no trace, and this occurs particularly in young persons: more commonly the expectoration and cough diminish, and even disappear, in summer, although even then the patient is subject to catarrh in the dry and latent form: in winter it again becomes mucous and evident, and often its return is accompanied by fever, particularly if the expectoration be abundant: after many returns of this kind the affection remains permanent; and in most cases, although the patient be-

come weaker and thinner, the pulse and heat of the skin continue natural."

I have occasionally seen so large a quantity of secretion formed as to suddenly and completely obturate a large bronchial tube, so that the dyspnoea became instantly increased to such a degree as to threaten suffocation: in these cases relief was only obtained by a copious expectoration: this may be classed under one of the causes of humid asthma. If the secretion accumulates, and there be no power of expectoration, the disease is then called *Suffocative Catarrh*.

In some rare cases, hectic is established, the patient becomes exceedingly thin, and the disease terminates in death: indeed, so completely does chronic mucous catarrh put on then the appearance of tubercular phthisis, that none but stethoscopic means could possibly distinguish them. The following case will exemplify this fact:—

About seven or eight years since, Mr. Fuller, the surgeons' instrument-maker, living near this hospital (who permits me to use his name), was supposed to be in a state of hopeless consumption; I was requested to see him, and found he had been labouring for some months with severe cough and dyspnoea; the expectoration was puriform and copious, emaciation great, fever of a hectic character, having evening exacerbations, and terminating in night sweats, and the pulse always very rapid. The functional and general signs of tubercular phthisis were so very evident that I was hardly disposed to fatigue him by a local examination; however, upon doing so, I found no pectoriloquy, no cavernous cough or respiration, no local

evidences whatever of tubercles in the usual situations, below the lines of the clavicles, but the mucous rattle was universal; here, then, was quite sufficient, in the extent of the mucous catarrh, to account for the symptoms without supposing a tubercle to exist. Leeches were frequently applied to the chest, blisters still more so, so that at one time I might say he had on an epispastic waistcoat: nauseating expectorants were given, and in a few weeks he perfectly recovered. It is in doubtful cases, like this, where auscultation is indeed of high importance.

Treatment. — There are three indications to be observed in chronic mucous catarrh: —

1st. To relieve any inflammatory congestion.
2d. To free the expectoration. 3d. To give tone to the bronchial capillaries and to the general system.

First Indication. — To relieve inflammatory congestion. It frequently happens, especially during great atmospheric changes, that the acute form of mucous catarrh will be superadded to or grafted on the chronic, and the patient's distress of breathing will considerably increase; in this case, if he be young, the abstraction of blood by leeches, or cupping, is useful; and even in old persons, if the oppression be great, I often apply a few leeches upon the sternum with advantage; general bleeding is rarely borne, I will not say that you should never have recourse to it; but you certainly should not, except the inflammatory symptoms be urgent, and the patient possess sufficient power.

Second Indication. — To free the expectoration. One of the best means of fulfilling this indication is by an emetic: it is particularly useful where the

secretion is large and difficult of expulsion. The medicaments called expectorants are uncertain in their operation, but we have a considerable list of them in common use: thus, ipecacuanha and tartar emetic answer best in young persons, or when an inflammatory state exists; but the aged, or those of weak, lax, and lymphatic temperaments, are most relieved by the stimulating class, as squills, ammoniacum, copaiba, tolu, turpentine, myrrh, &c.: the sulphate of zinc has been exhibited with some advantage, but I think the most powerful of all is the sulphate of copper; I am in the habit of frequently using it where the secretion is copious and viscid, and in doses of a third or half a grain dissolved in water three or four times a day.

Inhalations of tar vapour, or of chlorine, prove frequently of advantage in chronic mucous catarrhs. Blisters are of great use, particularly if the oppression or dyspnœa be considerable.

The third Indication is to give tone to the bronchial mucous membrane and to the general system. This is fulfilled, as far as relates to the membrane, by many of the expectorants already mentioned, especially those of a stimulating kind, but the direct tonics should also be combined with them, as the quinine, cascarilla, gentian, acids, &c.

One of the best tonics is passive exercise, combined with change of air, when the weather permits, as sailing and riding. I have known many instances of persons recovering from chronic catarrhs after long journies, if these were not performed too hastily, so as to occasion fatigue.

I need not say that all the remote causes should

be avoided; above all, exposure to rapid atmospheric changes; indeed, you will find it necessary to confine persons afflicted with this disease in warm rooms during winter, for the purpose of obviating this cause.

LECTURE VIII.

SUMMARY.

DISEASES OF THE AERIAN PASSAGES.

PITUITOUS CATARRH.

Acute Pituitous Catarrh. — *Morbid Anatomy.* — *Local Signs.* — Respiratory Murmur. — Various Species of Rhoncus. — Sub-sibilant Respiration. — *Functional and General Signs.* — Cough. — Expectoration. — Dyspnœa. — *Causes.* — *Treatment.*

Chronic Pituitous Catarrh. — Consequence of the Mucous and dry Forms. — Cough. — Expectoration. — Its large Quantity. — Œdema of the Lungs. — Effects upon general Health. — Humid Asthma. — Suffocative Catarrh. — *Causes.* — *Treatment.*

Whooping Cough. — Synonima. — *Morbid Anatomy.* — *Functional Signs.* — Paroxysm of Coughing. — Recurrence. — Duration. — Dyspnœa. — Increased by Combination with Peripneumonia or Œdema Pulmonum. — Epidemic. — *Local Signs.* — Rhoncus. — Respiratory Murmur. — *General Signs.* — *Causes.* — *Treatment.*

Catarrhus Siccus. — *Morbid Anatomy.* — *Local Signs.* — Percussion. — Respiratory Murmur. — Rhoncus. — *Functional and general Signs.* — Latent. — Dyspnœa. — Dry Asthma. — Nervous, Gastric Coughs, &c. — Sudden Increase of Dyspnœa. — Effect of Sleep. — Emphysema Pulmonum. — *Causes.* — *Treatment.*

Hysterical Cough.

WE have occupied so much time in the description of the acute and chronic forms of mucous catarrh, that we shall have less occasion to dwell on the other species, as they all bear a great affinity to each other in their morbid anatomy, signs, and treatment.

You recollect, gentlemen, the difference between mucous and pituitous catarrhs. Mucous catarrh is an inflammation of the ærian mucous membrane,

having for its result a secretion of a *yellow* matter ; in pituitous catarrh the secretion is *transparent*, *colourless*, and *viscid*. Pituitous catarrh, like the mucous, is divided into acute and chronic varieties, and we shall commence with the acute form.

ACUTE PITUITOUS CATARRH.

Morbid Anatomy. — The aërian mucous membrane is moderately swollen, and frequently somewhat softened ; it is rarely extensively reddened, but generally spotted irregularly. You will also find, that, in almost all severe cases, the air-cells of the lungs are more or less infiltrated with serum.

The membrane is in the first instance dry ; soon, however, the same sort of acrid, thin, and colourless secretion takes place as I described in the acute mucous form, and the fluid then becomes bland, viscid, without colour, and transparent ; it has a certain degree of tenacity, so that it may be drawn out into long films. The expectorated matter, when collected in quantities, is spumous ; and when its surface is removed, the fluid beneath appears like a mixture of white of egg and water. The quantity varies, but it is generally considerable.

Local Signs. — The respiratory murmur is less audible during the accesses of cough than in the intervals between, but it is rarely completely suspended, as in acute mucous catarrh : — the sonorous wheezing, sibilation, and the mucous rattle, may all be heard upon an extent of the surface of the chest, proportionate to the bronchial tubes affected. The sibilation is often slight, and seems to be pro-

longed in the whole of the bronchi: Laënnec denominates this the *sub-sibilant respiration*. The respiratory murmur is often louder than natural during the intervals of coughing, so as to be nearly puerile: if the bronchial tubes be dilated, it becomes bronchial; and as the disease is frequently accompanied by serous effusion into the air-cells, the crepitating rhonchus is then distinctly audible, particularly at the parts of the chest corresponding to the lower and posterior portions of the lungs.

Functional and general Signs. — This disease is not uncommon in a moderate form, but there is then always a troublesome cough, occurring in distinct paroxysms; the expectoration is copious, frequently from one to two pints in twenty-four hours; the dyspnœa and oppression are almost always more severe than in the previously described species, and when this catarrh is violent it soon threatens suffocation: it commences with the symptoms of a common cold, and in a very few hours the cough becomes excessive and the expectoration enormous; so that as much as three or four pints have been thrown up in a few hours. The dyspnœa is then extreme, and the patient falls into a state of indescribable anxiety; congestions form, the face becomes livid, the cerebral functions confused, the pulse irregular, and the extremities cold. This affection occasionally attacks infants, and is easily mistaken for croup.

Causes. — The same as those of acute mucous catarrh; but as that species occurs most frequently in bilious temperaments, this happens by far most commonly in those of lymphatic constitutions.

Treatment. — The indications of treatment are the same as in acute mucous catarrh; the same indications and objections to bleeding occur also: emetics are of great use, especially in children, in consequence of the great quantity of viscid filmy secretion which obstructs the bronchial tubes, and which they generally swallow rather than eject by the mouth. Laennec used the tartar emetic in the same manner as I shall describe when we arrive at the treatment of peripneumonia, and with success: he recommends the belladonna and paregorics, to diminish the necessity for respiration. Blisters and mustard cataplasms may be used with great advantage.

CHRONIC PITUITOUS CATARRH.

Chronic pituitous catarrh, like the chronic mucous, is a frequent infirmity of old age, and especially in those of a lymphatic temperament, previously weakened by excesses, or by too sedentary a life: repeated attacks of acute mucous catarrh evidently predispose to this affection. It has been stated, too, that it is not uncommon in old gouty persons, in whom that disease has lost its regular form and some of its intensity.

Chronic pituitous catarrh is rarely a consequence of the acute species — it commonly follows a succession of attacks of the mucous, or dry. When the expectoration is well established, it becomes almost regularly intermittent; there being usually two attacks of cough and expectoration in twenty-four hours — the one on waking in the morning,

the other in the evening, and in some immediately after a meal. The quantity is always considerable. Laennec has seen as much as three pints in each access, the duration of which was not above an hour or two; and it is extraordinary how long patients will continue to expectorate so large a quantity: he speaks of an individual, above seventy years of age, who, for ten or twelve years, had brought up every day, in two paroxysms, about four pints of pituitous fluid. I had a patient at the Lung Infirmary, a young Irishman, who for many months expectorated daily from five to six pints of transparent viscous matter: he ultimately fell a victim to the disease. I suspect that, in these cases, the lungs are always greatly œdematous, for I never meet with them without discovering co-existing crepitation; and in the case I have just mentioned, the whole pulmonary tissue was gorged with colourless serosity.

It is singular that this quantity of expectoration is not incompatible with a certain degree of health, although the disease continue for years. It is true that, after a certain time, the patient becomes paler and thinner; but he does not fall into a state of marasmus: he becomes more lymphatic, and the blood, if drawn, shows less consistence; but as old age advances, the accesses of cough are more prolonged and have shorter intervals—the dyspnœa is constant, and he is now called an asthmatic; in fact, it is another form of *humid asthma*: finally œdema of the lungs supervenes, and the catarrh becomes *suffocative*. It occasionally happens that the disease is quicker in its progress, and that the patient falls sooner into a state of great

exhaustion and marasmus. It is fortunately uncommon to meet with chronic pituitous catarrh in its exquisite form, although, in a moderate degree, it is by no means infrequent.

Causes. — They are the same as those of chronic mucous catarrh, but, as I have observed before, the disease is most frequent in persons of lymphatic temperaments.

Treatment. — The same mode of treatment should be adopted as we have described for chronic mucous catarrh.

WHOOPIING COUGH.

We have now to consider a variety of pituitous catarrh, known under the name of *Pertussis* or *Whooping Cough*. I place it as a variety of this affection, because the secretion is of the same character; although there are essential differences, consisting in the spasmodic nature of the cough, in the spasm of the glottis, in its attacking children more particularly, in its contagious nature, and in its occurring but once during life.

Pertussis, or whooping-cough, has received a variety of names: it has been called *tussis convulsiva*, *tussis ferina*, *chin-cough*, and *kin-cough*.

Morbid Anatomy. — The condition of the mucous membrane has not been much observed; but there is no reason to suppose that it differs from its state in ordinary catarrhs: indeed, I have seen it swollen, and reddened in patches, and the bronchial tubes containing a considerable quantity of spumous secretion. When *pertussis* terminates fatally, it is usually upon the supervention of pe-

ripneumonia or œdema pulmonum: in the first case, the lung will be found to contain sanguinolent serum, and here and there a lobule hepatised; in the second, a large quantity of highly spumous and colourless serosity follows the scalpel upon the section of the pulmonary tissue.

Functional signs. — There is a peculiar cough in this disease, which, when once heard, can never be mistaken for any other: it occurs in paroxysms, and consists of a number of saccaded, sonorous, and precipitated expirations, lasting for some minutes, and leaving the patient hardly a moment to inspire; at last a deep inspiratory movement is effected, attended by a prolonged crowing noise, evidently depending upon a spasmodic contraction of the glottis: it is to this noise the term of whooping is applied; and you will observe, gentlemen, that it always occurs during inspiration. After the first whoop, another succession of similar expirations takes place, followed by another whoop; and the paroxysm will continue in this way for a considerable time, until the little sufferer stamps with impatience: his face swells and becomes livid, especially during the whooping; he grasps a chair or any other firm object for support, inclines his body forwards, and at last the fit terminates in a tolerably copious excretion of pituitous fluid, which hangs in films from his mouth: very often the access ends by vomiting. In almost all ordinary cases the patient will return immediately to his amusements, and be as playful and unconcerned as if nothing had troubled him. These paroxysms may occur many times during the day: they are generally observed to be most violent during the

evening, and least so at night; and, as the disease proceeds to a favourable termination, the fits of coughing are usually confined to the morning and evening, then to the evening alone, and at last they totally disappear. Pertussis is most uncertain in its duration; for sometimes it runs its course in two or three weeks, sometimes in seven or eight months.

The dyspnœa is always considerable during the paroxysm of coughing, and especially towards its termination, even to impending suffocation. In the intervals there is commonly no difficulty of breathing; and when dyspnœa occurs, you may almost be certain of the co-existence of peripneumonia or œdema of the lungs; in either case the danger is much increased. Two or three winters since, pertussis was extremely fatal in the eastern parts of London; and I then invariably found that its fatality arose from the above combinations.

Local signs. — In the intervals of coughing, the ordinary signs of catarrh present themselves; that is to say, the sound on percussion is good, and the respiratory murmur varies on different points of the chest: at one part it is lost; at another, slight; at a third, puerile; and these parts will alternate with each other in the variation, so that, where the sound is at one time inaudible, it will at another be puerile. The various kinds of wheezing are also heard in pertussis; and if peripneumonia, or œdema pulmonum be superadded, crepitation becomes always distinct at the inferior lobes of the lungs.

During the paroxysm of coughing, a singular phenomenon occurs, which never could have been

discovered but by auscultation. It might be supposed that the lungs would inflate during the strong inspiratory effort producing the whoop; but not only is the rima glottidis spasmodically contracted, but it would appear that the bronchial tubes are in the same state; for, during that period, and, indeed, the whole paroxysm, not the slightest respiratory murmur is to be heard; it only appears when the access has totally ceased.

General signs. — Fever rarely occurs, except with peripneumonic combination. If the disease be much prolonged, and the patient be of a weakly constitution, great debility takes place, with hydro-pic effusions. In many cases the general health is but little affected.

Causes — Individual. — Pertussis by far most commonly occurs in children; I have, however, seen three or four instances of it in adults: it attacks but once during life.

External. — The common causes of catarrh certainly predispose to whooping-cough, perhaps occasionally produce it; but it most frequently arises from contagion, although Laennec expresses some doubts on that point.

Treatment. — I commence this part of my subject by stating, that there are few diseases more intractable than pertussis. It seems to be an affection that will run a certain course in despite of any means which may be used. Many indications have been pointed out, and many remedies proposed: these remedies, so vaunted, may have been of advantage in some cases, and then it has been inferred they would answer in all; and here let me observe, gentlemen, that we are too much in

the habit of generalising in medicine from isolated facts: the "*Post hoc, ergo propter hoc*" is unfortunately also too common a sophism in medical reasonings. You cannot by possibility be too careful in accumulating a series of facts;—you cannot be too cautious in observing their regularity, and their invariability of succession, so as to determine their relation of cause and effect; and if, by reiterated and scrupulous examination, you are enabled to form a legitimate induction, so as to establish a general principle or quality pervading a whole class of phenomena, you will indeed have made a great advance in your profession.

So long as there is no dyspnœa or fever, I have but little apprehension of pertussis, but content myself by attending to the general health of the patient, and administering an occasional emetic, by which the viscid phlegm is removed, and, perhaps, the spasmodic cough somewhat relieved; but I watch closely for dyspnœa, fearing the peripneumonic combination. If that should unfortunately occur, you must be directed by the rules laid down for the treatment of inflammation of the lungs.

When the disease has lasted some weeks, and has become chronic, various means have been proposed,—as small doses of opium, or of conium or hyoscyamus, musk, acetate of lead, the recent powder of belladonna, in doses of one eighth to half a grain, or the extract of its root, from the eighth of a grain to a grain: the extract of the narcissus pseudo narcissus has been much extolled, in doses of half a grain to one or two grains every four or six hours: the hydrocyanic acid has been used with varied success: cantharides was formerly

a very favourite remedy, particularly when combined with the cinchona: ammonia, æther, and camphor, have all been successively the fashionable remedies.

Various external means have also been employed, as blisters, stimulating embrocations rubbed on the chest and along the spine; but I have so little faith in the use of these, that I shall not fatigue you with their detail, but refer you to the works on medicine for the different formulæ that have been and are still in use.

One of the best remedies is a change of air, although that does not always cure; at least its effects are so slow, that it is difficult to attribute the restoration to health to that cause. I had a remarkable proof of this in my own family. Two of my children were seized with pertussis in London; after a fortnight they were removed into Essex, where they remained two months longer with unabated symptoms; I then took them to Paris, and the disease did not finally disappear until they had been there four or five months.

CATARRHUS SICCUS, OR DRY CATARRH.

The generic character of catarrh being an inflammation of the ærian mucous membrane, having for its result a secretion, the expression of catarrhus siccus would seem to imply that it should not be included in that genus; but the term siccus is not to be understood in the positive sense, but in the comparative, since the disease consists also in an inflammation, having for its consequence a secre-

tion, although infinitely less in quantity than in the preceding species.

Morbid anatomy. — The mucous membrane in this affection is of an obscure red or violet colour; it is swollen, and the swelling more particularly takes place in the smaller bronchial tubes, which are sometimes almost entirely closed by it, although occasionally the membrane lining a larger branch will be thickened to the extent of a few lines, whilst in the continuation of that branch, or in its ramifications, it remains in its natural condition. The extent of the inflammation of the membrane is proportionate to the duration of the disease; yet the whole mucous surface has been thus affected in infancy. When the dry catarrh is universal, or even extensive, it always causes emphysema of the lungs.

The secretion in this disease is very different from that of the other species of catarrh: it consists of an extremely viscous matter, formed into globules, of the size of a pin's head, or larger; they are never mixed with air, are semi-transparent, and of a pearly aspect: this latter appearance is supposed to depend upon the mixture of a small quantity of the black pulmonary matter; a number of little dark spots are also occasionally seen in the globules. This affection is so common, that many persons, who make no complaint whatever, expectorate a small quantity every morning. These secretions have been named by Fourcroy, *bronchial mucus*; by Laennec, from their similarity to pearls, *pearly sputa*, or *sputa margaritacea*, to distinguish them from the mucous and pituitous fluids.

Local Signs.—The percussion of the chest affords a perfect sound, but the respiratory murmur is lost, or nearly so, at the points corresponding to the bronchial tubes affected. These points vary when the catarrh is general; for often the parts where, on a first examination, the murmur was inaudible, after a short time present it distinctly. These variations depend upon a greater degree of swelling of the membrane at one time than at another, or upon the varying quantities of pearly sputa obstructing the tubes at different periods.

If the engorgement of the smaller bronchial tubes be not carried to its highest degree, the respiratory murmur may still be heard, although very feebly; and at the same points there will be a slight sibilation or clicking, as if of the opening and shutting of a small valve; this last sound is least common, and can scarcely be distinguished, except upon deep inspirations or coughing: it depends upon the displacement of a pearly sputum by the air. It is probable that these noises would be louder were it not for the dilatation of the air-cells (*emphysema pulmonum*), which so frequently accompanies this disease.

Functional and general signs.—A moderate dry catarrh will often remain latent for years. Persons so affected perceive only that their breathing is not so free as that of others when they mount an elevation or run; but when the tubes become extensively affected, they breathe with difficulty, even in a state of repose, and particularly after a meal: some persons referring a sensation of obstruction to one side of the chest only. At a later period, the accesses of dyspnoea become so severe as to

merit the name of asthma; and it is one of the causes of the disease we shall hereafter describe under the name of *dry asthma*. Towards the end of these attacks, cough comes on, and the dyspnœa and oppression diminish; and in a short time a few pearly sputa are expectorated, often floating in a little pituitous fluid, by which the symptoms are still further diminished. In very slight cases, the sputa lose their globular form and density, become more abundant, and are slightly tinged by an admixture of yellow or whitish and opaque mucus; at other times they have the consistence and appearance of the vitreous humour of the eye: the *vitreous pituita* of the ancients.

A similar ejection of phlegm occurs habitually in many persons subject to slight degrees of dry catarrh; and if the secretion be not suspended, they are never subject to dyspnœa or asthma. Often the quantity is so small, that they do not observe themselves to expectorate; in others, there is really no cough or expectoration, or the cough is extremely slight and dry, and occurs but once or twice in the twenty-four hours; and then, especially if it has come on slowly, or has not been preceded by acute catarrh, it has been called a *nervous cough*, or it has been supposed to be sympathetic of some other affection, so that it has been denominated *gastric, hepatic, hysteric, &c.*, when, in fact, a local examination of the chest would soon enable us to detect its real cause.

When an acute catarrh is grafted upon the chronic dry form, then the dyspnœa suddenly augments, so as to constitute a fit of asthma; it is relieved by expectoration. Upon a recovery from this state,

the habitual dyspnœa is somewhat increased, because the disease has extended further in the course of the bronchial tubes.

It happens occasionally, in nervous persons, particularly in hysterical women, subject to dry catarrh, that a short asthmatic paroxysm will occur upon mental agitation. Thus, I had a patient, who, upon any stranger entering the room, commenced to breathe with great difficulty, and the sibilating wheezing was heard all over the parietes of the chest. After a few minutes, the dyspnœa subsided, and nearly all the sibilation with it.

During sleep, or upon the accidental occurrence of fever, the dyspnœa diminishes, and the instant of waking seems to the patient the only time he breathes freely. Finally, when the disease has lasted a considerable time, emphysema of the lungs and its signs supervene.

Causes. — This is a very frequent disease: persons of all ages and temperaments are subject to it; almost all the inhabitants of maritime and cold countries, or of humid valleys, are constantly attacked by it, although in various degrees; and Laennec asserts that half the inhabitants of the driest parts of France, possessing apparently excellent health, present traces under the stethoscope of some slight bronchial engorgement. From an official situation I hold, I have occasion to examine a number of persons in good health, and there are few indeed who do not present some slight wheezing at one point or other of the chest on a deep inspiration.

Gout, hypochondriasis, certain chronic cuta-

neous eruptions, are supposed to predispose to this disease.

Dry catarrh is often sympathetic of fever: you generally find the bronchial tubes more or less affected in that class of diseases.

Treatment. — The first indication we should have in view is to diminish the inflammatory action going on in the mucous membrane; but, as in the other forms of catarrh, patients rarely bear general bleeding. If, however, the dyspnœa be great, and the subject plethoric, then bleeding is necessary. I often apply leeches with advantage. Counter-irritants, as blisters, the tartar emetic ointment, Burgundy pitch plasters, and dry cupping, frequently produce a good effect, although not always a permanent one.

When the cough is violent and distressing, you will find opium produces the most beneficial results, given in doses of a grain, or a grain and a half, at bed-time. Laennec administered it in smaller doses, and repeated them frequently.

Our second indication is to diminish the viscosity of the tenacious pblegm which obstructs the small bronchial tubes. The authors of the humoral pathology imagined that certain medicaments had a solvent effect in these cases. Without reposing any confidence in their hypotheses, I can assert that many of the remedies proposed with that intention do much service. I use frequently the common yellow soap, in doses of from five to ten grains, three times a day. The carbonates of ammonia, soda, and potash, in doses of from ten to thirty-six grains a day, mixed in common drinks,

were frequently given by Laennec. The ipecacuanha has a very good effect, if long continued. A grain of the powder may be administered three or four times in the twenty-four hours. I have frequently used mercury in severe cases, and am satisfied that, if carefully managed, so as not to excite salivation, it often diminishes the bronchial tumefaction, and occasions a free and diffuent expectoration.

HYSTERICAL COUGH.

Although I have not classed this affection among the catarrhs, still I have placed the description of it here, as it assimilates more to those disorders than any other.

This disease invariably, as far as my experience extends, attacks young hysterical women; I have met with it thirteen or fourteen times, and never in the other sex. It usually occurs before the age of twenty. I have not observed a case beyond the age of twenty-five years. It can hardly be called a cough, it is rather a bark; it is a loud hoarse sound, produced by the act of expiration repeated many times in a minute, for hours, days, weeks; I have seen it continue, with but slight intermissions, for months, the patient having scarcely any relaxation from it but during sleep. The voice is almost always affected: it is generally reduced to a whisper, sometimes to perfect aphonia. A sense of soreness is occasionally complained of about the larynx; the breathing is not always difficult, nor is there always pain or oppression in the chest. The disease is rarely accompanied by expector-

ation, although in two instances I have seen it combined with pituitous catarrh, and then the quantity of transparent viscous fluid thrown up was considerable; but it was no doubt an accidental coincidence. Sometimes the patients are much distressed and weakened; sometimes they preserve their cheerfulness, appetite, and strength.

I know nothing of the morbid condition of parts in this disease, as I never saw a patient die of it. All I can say is, that there is evidently a spasmodic condition of the respiratory muscles, and of the glottis, depending, probably, upon some peculiar state of innervation.

Treatment. — This affection seems to be as intractable as pertussis, to which, indeed, it bears some analogy. I have tried bleeding, blistering, drastic purgatives, iron, and various antispasmodics, and I cannot say with the slightest beneficial effect. Change of air seems to do more for the patient than any thing; else although you must not consider it as a certain remedy. As the disease is somewhat rare, I will relate the following case:—

I was called to a young lady of about 20 years of age, of a sanguine lymphatic temperament, and who had been the subject of hysterical fits. I found she had been coughing unceasingly for five or six weeks, having no remission except during sleep. She had totally lost her voice. She did not appear distressed, but smiled, and looked as if she thought her state rather ridiculous than distressing. A variety of means had been used, but inefficaciously. I advised a change of air. She took a coasting voyage, and had hardly got to sea before the barking left her, and her voice returned.

She continued this voyage a week ; and, landing upon some part of the coast, her complaint returned. It diminished, but did not altogether leave her, upon getting to sea again ; but on her arrival home she became as bad as ever, and continued so, to the best of my recollection, about two months more, till one morning she suddenly exclaimed, " Mother, I can speak ! " From that moment her cough ceased, and her voice returned.

LECTURE IX.

DISEASES OF THE AERIAN PASSAGE.

PLASTIC INFLAMMATION, OR CROUP.

History. — *Morbid Anatomy.* — Mucous Surface. — False Membrane — Characters of — Situation of. — “Diphtherite.” — Separation of False Membrane. — Difference of Situation in Children and Adults. — *Functional Signs.* — Dyspnœa. — Sounds on Inspiration. — Coughing and Speaking. — Symptoms of Croup in Bronchial Tubes and Fauces. — *Local Signs.* — *General Signs.* — Fever. — *Causes.* — *Treatment.*

Dilatation of Bronchial Tubes. — Discovered by Laennec. — *Morbid Anatomy.* — Various Forms of the dilated Tubes. — Hypertrophy or Atrophy of their Parietes. — Situation of dilated Tubes. — *Causes.* — Chronic Mucous Catarrhs. — Tubercular Excavations. — *Local Signs.* — *Percussion.* — Auscultation. — Temporary Disappearance of Signs. — *Functional and General Signs.* — Dyspnœa. — Expectoration. — *Treatment.*

Bronchial Hæmorrhage. — *Morbid Anatomy.* — *Causes.* — *Symptoms.* — *Treatment.*

Bronchial Polypi.

So far, under the name of catarrh, we have spoken of the inflammations of the ærian mucous membrane, which have for their results the secretion of a fluid which remains in the liquid state. We now proceed to the description of another species of inflammation, which also has for its result a fluid secretion; but this secretion almost instantly concretes, and forms a false membrane, lining the affected parts of the air-tubes. This is called *Plastic Inflammation*, or, more commonly, *Croup*.

The nature of this disease seems to have been unknown to the ancients. In all probability, how-

ever, it existed in their times; but, as most of its signs are common to other pulmonary affections, especially catarrhs, and as morbid anatomy was then totally unknown, it is very possible that its special nature was either overlooked or misunderstood. The first author who mentions or throws any light on the subject, seems to have been Bailou, or, as he is often called, Ballonius,—the French Hippocrates: he described something similar to it in the year 1576. Tulpius, Morgagni, and others, had some notion of the disease, inasmuch as they mention excretions of *polypi*. Some thought the mucous membrane itself was thrown up, and others that the blood-vessels of the lungs were separated and expectorated. Ghisi, of Cremona, in 1740, first described the true character of the affection, and after him many authors took up the subject, more particularly Dr. Home, of Edinburgh; finally, Bretonneau, of Tours, has given the most complete account of Croup, under the name of "*Diphtherite*."

Morbid Anatomy.—Croup consists in an inflammation of the mucous membrane of the ærian passages, having for its consequence a secretion which concretes almost as soon as it forms.

When you elevate the concretion, or false membrane, you will perceive that the mucous surface beneath is reddened and swollen, but rarely to the degree we find in catarrhs; we must therefore conclude that the pseudo-membrane is not caused by a greater intensity, but that it rather depends upon some specific nature, of the inflammation. It has been supposed that catarrhs result from inflammation of the secreting follicles of the mucous membrane, and croup of the capillary arteries of its

tissue. This opinion is plausible and, probably, correct; but it has not yet been proved.

The false membrane lines and moulds itself to the part inflamed, and extends no farther: its thickness is from half a line to a line, and is generally greatest at its upper extremity; its colour is white, or whity-brown; it has the consistence of boiled white of egg, and its firmness is always greatest where it is thickest. As it descends into the trachea it becomes thinner and softer, terminating in mere mucus. The secretion constantly forming from the inflamed parts, separates the false membrane from the mucous surfaces, particularly at its inferior and soft extremity, and then, white shreds are often expectorated; the new secretion concreting in its turn.

The disease may attack any part of the aërian passages, as has been distinctly shown by Bretonneau; thus it may affect the cavities of the nose, the pharynx and mouth, the epiglottis, larynx, trachea, and the bronchial tubes; nay, the pseudomembranes have been observed in the œsophagus, stomach and intestines: with the exception of the nares, I have seen it in all these parts.

Croup attacks the larynx most frequently: Laennec observes that it rarely extends above the glottis; but, gentlemen, nearly every preparation before you, and there are six, presents the false membrane also on the inner surface of the epiglottis; there can be no question, however, but that the disease is usually confined to the larynx and epiglottis, and the adventitious formation then takes the characters I have already described.

But when the croupal membrane extends into

the bronchial tubes, it then becomes modified in its appearance: it generally is formed into a single cylinder, as Bretonneau has described, and occasionally into a series of tubes inclosed within each other in the closest contact, and forming altogether a solid body of a thick and tolerably firm structure, and of the exact form of the tube from whence it has been abstracted: upon making a transverse section of this polypiform substance, concentric laminae are very apparent. It adheres but slightly to the mucous surface, and may easily, after death, be drawn out entire. This structure was, I believe, first described by Dr. Withering. The specimen I now present to you shows a complete mould of a large bronchial tube, and its minutest ramifications. You perceive also the concentric laminae upon its section.

The croupal membrane has never been seen to consist of these distinct layers in the larynx and trachea, but in the bronchi only; the reason of this seems obvious. When the larynx and glottis are affected, the passage of the air is so obstructed that the disease soon terminates either fatally or otherwise, and before a second membrane has time to form and envelope the first. When also a cough takes place, the column of air ascending the trachea is of sufficient volume, and is projected with adequate force to tear away the loosened membrane, and occasion its expectoration; if, however, the formation takes place in a bronchial tube, even of large caliber, the danger is less imminent; the column of air passing through that tube during a cough is of less volume; and, finally, as the vessel fills with the concretion, no air passes at all, so that

there is sufficient time and repose for the pseudo-membranes successively to form and surround each other.

Bretonneau was the first to demonstrate that the state of the fauces, which is known under the names of angina gangrenosa, cynanche maligna, or gangrenous sore throat, depended, in the greater number of cases, upon plastic inflammation, and that the supposed gangrenous slough was nothing more than an adventitious membrane. He proved this, by showing that there was no loss of parts upon recovery, which there would have been, had there existed previous gangrene; by the disease descending into the larynx, and producing all the symptoms of croup; and, finally, by repeated dissections, showing the continuity of the membrane.

When the disease attacks the fauces, small yellowish-grey spots appear surrounded by the mucous membrane, highly and darkly reddened: these spots enlarge, coalesce, and form a continuous false membrane of the colour and consistence of the buffy coat of the blood, lining the throat, more or less, completely. I have seen it cover the posterior half of the tongue, so that its anterior part might be elevated by a probe in a membranous form: it rarely passes into the œsophagus, but frequently into the larynx, and suffocates the patient.

If this form of the disease terminates favourably, the false membrane then detaches itself: it is replaced by a thinner and less plastic exudation, altogether similar, to catarrhal mucus. Sometimes the membrane does not separate, but appears to be gradually absorbed, becomes then less opaque, and sufficiently transparent for the colour of the mucous

membrane to be seen through it; finally, it entirely disappears. In the worst cases, the pseudo-membrane is soft and friable, like soft cheese.

It may be observed, here, that the croupal membrane almost always originates in the larynx in children, and does not spread into the fauces, and is not accompanied by symptoms of gangrene; while, in adults, it commences in the fauces, and passes from thence into the larynx, and the symptoms then frequently are similar to those of gangrene.

The observations of Bretonneau would lead to the supposition that gangrene of the fauces never occurred; but there can be no doubt it occasionally happens, although much more rarely than the plastic inflammation: it is very probable that a combination of the two affections may occasionally take place; that is, a false membrane may form over a gangrenous surface.

Symptoms — Functional. — When croup attacks the larynx and trachea, the functional signs must have relation to the respiration and the voice.

The respiration is always impeded in proportion to the quantity of false membrane, by which the tube is rendered narrower, and consequently the column of air of less diameter. The edges of the circumference of the inferior extremity of the adventitious deposit being loose, mucous, and soft, frequently adhere to each other, so as to close up the tube, and require a strong effort of inspiration to overcome the temporary obstruction: I have seen a slight film of pseudo-membrane traversing the upper part of the larynx, suffocate an infant

almost instantly. The dyspnœa becomes always great when the concretion separates, and, if it be not quickly expectorated, often destroys the patient immediately.

The disease usually commences with the symptoms of common catarrh, soon to be followed by a violent cough, which rapidly assumes the croupal character: this cough is occasioned by the irritation of the false membrane, and may be considered as an effort of nature to expel it, which is occasionally effected in the form of small whitish shreds.

The acts of inspiration, and of coughing, produce peculiar sounds: it seems as if the noises were formed and resounded in a brazen tube: they have some analogy to the crowing of a cock. The voice, too, has a similar character: these sounds have been denominated, croupal inspiration, croupal cough, croupal voice.

If the disease attack the bronchial tubes alone, and not the trachea and larynx (which is, I believe, a very rare occurrence), the dyspnœa will be in proportion to the size and number of tubes affected; the croupal voice, inspiration, and cough, will no longer be present, and the expectoration of the false membrane is the only means of distinguishing the disease. I have seen two instances in adults, where, after violent coughing, pieces of false membrane of the form of the bronchial tubes were expectorated; being, no doubt, the polypi or bronchial vessels of the ancient authors.

When croup affects primarily the fauces, the act of deglutition becomes difficult, and there will not be present either dyspnœa, cough, or the croupal

sounds ; but when it progresses and descends into the rima glottidis and larynx, then all the before-described symptoms will be superadded.

Local Signs. — These signs have not been sufficiently studied when croup exists in the larynx, trachea, or bronchi. The appearances I have described, under the head of Morbid Anatomy, afford sufficient local indications when the disease attacks the fauces.

General Signs. — Croup is always accompanied by general disturbance of the functions of the body : in most cases the inflammatory sympathetic fever is acute and intense, and frequently combined with irregularities in the movements of the heart. In some instances, particularly in hospitals, or under the influence of certain epidemics, the general signs assume an asthenic character : thus, the pulse diminishes in frequency, and becomes exceedingly feeble ; the skin of a dirty or earthy aspect ; the breath foetid, even when no gangrenous points are seen in the throat ; and the prostration of the strength of the patient is extreme.

Causes — Individual. — Croup by far most commonly attacks infants ; but it sometimes occurs in adults, particularly when it commences in the fauces.

External. — It often appears as an epidemic, especially in places exposed to the north and north-east winds, particularly when they blow with violence, and for a long time. Laennec suspected, from seeing the plastic membrane forming in different and remote parts of the body at the same time, that the disease depended upon some peculiar state of the fluids rather than of the solids. The asthenic

form of croup occurs most frequently in hospitals. The question of its contagious nature is not so decided as to render it prudent to allow persons to expose themselves unnecessarily to the breath of croupal patients.

Diagnosis. — It is sometimes difficult to distinguish this disease from acute laryngitis, particularly in children; but croup rarely attacks adults, and children seldom labour under acute laryngitis.

You have no diagnostic mark even in the sounds of the cough or respiration; for sometimes they are but slightly formed, and frequently they are very like those produced in whooping-cough. The only pathognomonic sign is the expectoration of shreds of the false membrane.

Treatment. — Our indications of treatment are, to destroy the inflammatory action, and to cause the expulsion of the false membrane.

You will recollect, gentlemen, I have already observed that croup must not be regarded as an inflammation of a violent, but of a peculiar, nature; for the redness of the mucous membrane is usually less considerable than in ordinary catarrhs: you must consider, too, that the formation of the croupal membrane is probably an effort of nature to terminate the inflammation by such a secretion, as pus is the termination of that state when occurring in the subcutaneous cellular, and in other tissues; so that we have rather to contend here with effects than with the causes producing them. It is true that, although we may be certain that the false membrane exists, we cannot be sure that the inflammation producing it has subsided; consequently it will be prudent to bleed or apply

leeches in proportion to the strength of the sufferer; but these means must not be carried to any considerable extent, as experience has distinctly shewn. The usual antiphlogistic remedies may also be used.

Mercury yields to no other medicament in its powerful effect in subduing this disease: calomel should be given freely in doses of one, two, or three grains, every hour or two, until the mouth be affected, the breath foetid, or the patient relieved; I say until the patient be relieved, because you will sometimes see, in practice, that the disease will be subdued before the gums are rendered tender; and, under such circumstances, it is unnecessary to continue the remedy. If it should happen that the bowels are irritated by the calomel, then the hyd. c. cretâ may be substituted, or, what is far better, the free inunction of the unguentum hydrargyri.

Various derivatives have also been employed with the view of subduing inflammation,—as blisters, sinapisms, rubefacients of different kinds. All these act, probably, in the same way, by producing counter-irritation; although Laennec imagined that a cataplasm, sprinkled with muriatic acid, and applied to the throat, would tend specifically to separate the false membrane. That muriatic acid, applied to the surface of the false membrane in the fauces, will tend to destroy it, there can be no doubt; but that it will so act through the skin, is very problematical. Warm baths are very much used in this country, and often with advantage.

To effect the expulsion of the concretions, emetics have been given once or even twice a day for

some time, and often with complete success: hydrosulphuret of potash was formerly much used, from its supposed solvent quality; but its action, if any, is so slow, that it has fallen totally into disuse.

Topical remedies, of course, cannot be used when the disease exists in the larynx, trachea, or bronchi; but I can speak from experience that they are of great utility when it attacks the fauces; and I think its further progress is stopped by their prompt use. Various means of this kind have been adopted, — as alum, or the famous remedy of Van Swieten; which consisted of three parts of honey, and one of muriatic acid. You will find one of the best to be a solution of lunar caustic, in the proportion of six or eight grains to an ounce of distilled water: this may be applied by means of a little mop, made of a small piece of sponge or linen, fixed to the extremity of a piece of wood: use it two or three times a day, and, if possible, touch all the parts of the false membrane in sight freely.

If the danger be extreme, bronchotomy may be resorted to, by way of acting up to the maxim, that “*Anceps remedium melius quam nullum* :” but you must not promise yourselves much success from that operation; for it is impossible to determine previously, whether the false membrane has not descended below the point at which you would puncture the trachea: if this has been the case, then you would only add to the danger and distress of the patient. Bretonneau performed it with success on a child of four years of age, the daughter of the Comte de Puységur. Bronchotomy has been repeated in this country in croup, but with little success.

Ulcerous Inflammation of the Mucous Membrane.

— I shall defer the consideration of ulcerous inflammation until I describe Tubercular Phthisis, as it is an affection intimately connected with that subject.

DILATATION OF THE BRONCHIAL TUBES.

We now pass on to the consideration of the dilatation of the bronchial tubes.

This morbid condition of the bronchial tubes was not known until it was described by Laennec, although it occurs frequently. The reason it was not previously discovered is very evident. When we examine a lung, we rarely open the trachea, and trace its ramifications; but we generally cut the viscus across, and it is then impossible to distinguish whether a tube is dilated, or of the normal diameter. You should open the trachea, and follow its branches; and if, after a branch passes on to a certain distance, its diameter becomes greater, you may then consider it morbidly dilated.

Morbid Anatomy. — Dilatation of the bronchial tubes takes place in a variety of forms: it coexists almost invariably with chronic mucous catarrh. A bronchial tube, that in its natural state would scarcely admit a crow's quill, will increase in diameter to the size of the finger. A tube may continue to a certain extent of its natural size; it may then dilate, and afterwards reassume its proper dimensions: this is a common form to meet with. There may be a succession of dilatations and contractions along the same tube: sometimes the dilatation terminates in an irregular cul de sac, in which a number of

small bronchial tubes open; in other cases, the bronchial tubes lose their form, and present a cavity capable of receiving a cherry or almond stone. Often many contiguous branches are unequally dilated, particularly in the upper lobes of the lungs, and form, by their communications, sinuses full of puriform mucus, giving the appearance of a multilocular tubercular excavation. The extent of this disease varies considerably: it may occur in a single tube, in many, or even in them all.

The parieties of these dilated tubes also vary: they may be hypertrophied or atrophied. In the first case, the mucous membrane is most commonly from a third to a quarter of a line in thickness, softer than natural, and of a violet red colour; the submucous cellular tissue is white, very firm, and of a fibrous texture; the cartilaginous rings are occasionally seen to enter into the substance of the parietes. In the second case, the dilated bronchi are of extreme tenuity, so that no remains of their primitive organisation are apparent; they are then a little firmer than the healthy mucous membrane, red, and of a smooth internal surface: their tenuity becomes so great, that they may be compared to the pellicle of an onion. The whole of the bronchial tubes of a lung have never been seen dilated in this manner; and the largest Laennec saw would not have contained more than a nut, and it consisted of a number of bronchial tubes irregularly dilated, juxta-posed, and communicating with each other.

These dilatations almost always occur in the upper lobes of the lungs, and towards their anterior edge: ordinarily a few branches are only affected,

but sometimes all those of one lobe ; then the dilatations are greater in the small ramifications than in the branches from which they arise, and greater in the branches than in the trunks from which they originate. The pulmonary structure which exists between these dilatations is almost always condensed, and no longer permeable to the air.

Causes.—Dilatation of the bronchial tubes rarely occurs, except in individuals attacked by chronic mucous catarrhs. It depends, doubtless, upon the prolonged stay of sputa in the bronchi, by which these vessels become gradually dilated. The bronchial ramifications, which enter into a tubercular excavation, are also occasionally dilated, and remain so even when these excavations are converted into fistulæ, the tubes generally preserving their cylindrical form.

Symptoms — Local.—Laennec has given us the following description of the signs of this disease :—

When the bronchial tubes of the whole of a lung are dilated, the sound given by percussion is duller than in the natural state, because of the compression of the pulmonary tissue ; but this sign is usually less sensible, except other circumstances contribute to produce the same effect.

In the points where the dilatations are greatest, pectoriloquy may be heard more or less distinctly : it is accompanied by a mucous rattle, perfectly similar to the cavernous rhoncus of phthisical patients. Bronchial respiration is distinguishable on the same points which an inexperienced observer would easily confound with the puerile respiration, because of its intensity. This sound becomes cavernous on the points of the chest, correspond-

ing to the largest dilatations. The cough and mucous rattle have also the bronchial and cavernous characters in those dilatations which are nearest the surface of the lungs. The voice, respiration, and cough often, also, give the sensation of the "Souffle voilé;" that is to say, of a thin veil, or humid membrane, floating and preventing the column of air from penetrating into the ear at each vibration.

Sometimes all these phenomena disappear for a certain time, particularly at the lowest parts of the lungs, because of the accumulation of mucous sputa: they reappear only after a copious expectoration, or a change of position.

When the dilatations are slight, and nearly equal in a certain number of tubes, there will be diffused bronchophony instead of pectoriloquy. When the dilatations are extensive, there will be found on all the corresponding parts of the thoracic parietes, bronchophony and bronchial respiration, and on some points only, perfect pectoriloquy.

Functional and general Signs. — Even where the dilatations are most extensive, the symptoms of the disease rarely indicate its gravity: most commonly there is no fever, nor marasmus. If the patient has no laborious occupation, he rarely feels a diminution of power: dyspnœa only occurs on rapid movement.

The expectoration is not characteristic; its abundance is only remarkable in very extensive dilatations: it is always mucous, or puriform, usually inodorous, occasionally of the smell of pus, or even of a foetid wound. Sometimes the sputa augment with such rapidity, that you might suppose a vomica had ruptured.

It may be observed, that the local signs of dilatation of the bronchi are common to several other diseases, particularly tubercular phthisis, abscess from peripneumonic inflammation, and gangrenous excavations: but the accumulation of all the signs can leave no doubt in the mind of the practitioner. We shall, however, enter into greater details of the diagnosis, when we arrive at the description of those organic lesions of the pulmonary structure, which dilatations of the bronchi simulate.

Treatment. — As the disease is a consequence of, or is complicated with, chronic mucous catarrh, it is evident that the only means of contracting the dilated tubes is by diminishing the secretion from the mucous surfaces. The treatment, therefore, is precisely the same as that recommended for chronic mucous catarrh.

BRONCHIAL HÆMORRHAGE.

By bronchial hæmorrhage is meant an excretion of blood from the surface of the mucous membrane lining the trachea and bronchial tubes.

Morbid Anatomy. — There is generally found a certain quantity of coagulated blood in the bronchial tubes, putting on the appearance of small polypi, which are merely the fibrous portion of the blood, often deprived of its colouring matter. The membrane itself is redder and softer than in the healthy state.

Causes. — This affection often occurs in young plethoric persons. It may be induced by circumstances which throw the lungs into considerable

action, such as blowing wind instruments, hard exercise, and violent coughing. It will occasionally supervene upon the suppression of hæmorrhoids and the catamenia. The most common cause, however, is tubercular disease; and I look with more apprehension upon a small discharge of blood from the trachea than a large one, because the result is generally more dangerous. We sometimes find in epilepsy that a small quantity of blood is mixed with frothy saliva: its source is most probably also the bronchial mucous membrane.

Symptoms. — The hæmorrhage is usually slight, and spumous at first; but, towards the end of the attack, the blood is coagulated. When the quantity is great, it is commonly caused by that state of the lung, denominated by Laennec, Apoplexia Pulmonum.

It is highly important to determine, whether the blood flows from the mucous surface of the bronchial tubes, or from the parenchymatous substance of the lungs. The absence of all the signs of the latter disease principally determines the diagnosis: thus, in bronchial hæmorrhage, there is no absence of respiratory murmur, nor is there crepitation on any point of the chest. When the hæmorrhage is slight, there is scarcely any acceleration of the pulse, nor does it present the hæmorrhagic vibration. Dyspnœa, heat and oppression beneath the sternum, scarcely ever occur in bronchial hæmorrhage.

Treatment. — General bleeding should be employed, if the patient be plethoric: opening a vein in the foot, or the application of leeches to the inside of the thighs, is a common continental prac-

tice, if the hæmorrhage appears to depend upon a suppression of the catamenia.

Various astringents have been used, — as acids, alum, superacetate of lead, iced drinks, cold air in currents: the most absolute repose and silence should be enjoined, and absence from all stimulating liquids.

BRONCHIAL POLYPI.

Polypi of the mucous membrane of the bronchi are very rare: their structure is usually vesicular, and formed of a tissue analogous to mucous membrane, and enclosing small serous cysts. Laënnec once found a concretion of an inch and a half in length, and four or five lines in breadth, attached to the commencement of the left bronchial tube, and nearly obliterating it. Its tissue was compact, and similar to the polypiform concretions found in the heart and arteries; organisation had evidently commenced, for it was firmer and less humid than these formations: its internal colour was white, mixed with shades of yellow and red; a few small blood-vessels were seen ramifying within it. On the external surface a still greater number of vessels were observed, and the colour was there of a reddish violet, particularly at its largest extremity. Laënnec believes these concretions to be the fibrinous parts of the blood gradually becoming organised. We shall examine this opinion when speaking of the diseases of the heart.

LECTURE X.

DISEASES OF THE PARENCHYMATOUS TISSUE OF
THE LUNGS.

Division. — Table.

Peripneumonia. — Definition. — Morbid Anatomy. — Engorgement. — Hepatization. — Lobular Pneumonia. — Purulent Infiltration, gray Hepatization. — Vomica. — Gangrene — non-circumscribed — circumscribed. — Gangrenous Eschar. — Deliquescent Sphacelus. — Gangrenous Excavation. — State of Bronchi in Peripneumonia. — Situation. — Characters of Resolution. — Engorgement. — Hepatization. — Purulent Infiltration. — Duration of the different Stages.

HAVING thus, gentlemen, described the disease of the aërian passages, we now proceed to those of the parenchymatous substance of the lungs.

The diseases of the substance of the lungs may be subdivided into two sections: 1st, Primary diseases of the pulmonary tissue; 2d, Diseases of the substance of the lungs arising from adventitious deposits. I offer you the following table explanatory of the order we shall follow: —

I. Primary diseases of the pulmonary tissue —

Peripneumonia.

Œdema Pulmonum.

Apoplexia Pulmonum.

Emphysema Pulmonum.

Asthma.

II. Diseases of the substance of the lungs arising
from adventitious deposits. These deposits
are, —

Tubercles.

Ossific deposits.

Cysts.

Hydatids.

Medullary Sarcoma.

Melanosis.

I. *Primary Diseases of the Pulmonary Tissue.*

PERIPNEUMONIA. — By peripneumonia we mean an inflammation of the substance of the lungs. Although it is a disease of very common occurrence, yet its morbid anatomy was not well understood until described by Laënnec. We shall proceed precisely in the same order which we followed in the first section, and consequently at once describe the morbid anatomy of this affection.

Morbid Anatomy of Peripneumonia. — This subject may be considered under five different stages, or states, most of them having different signs, and requiring different modes of treatment. These states are named: 1. Engorgement. 2. Hepatization. 3. Purulent Infiltration. 4. Vomica; and, 5. Gangrene.

1st Stage — Engorgement. — The lung is now heavier than natural; its external colour is livid, or violet, and its firmness is increased: it still crepitates, though in a less degree than in the healthy organ; it feels more compact, and pits upon pressure like an œdematous limb. When a section is made, its surface is of a bright red colour, and a large quantity of sanguineous and spumous serum flows from the incision. The cellular or spongy texture of the lung is still perfectly apparent: here and there a few firm and compact

points may be seen, indicating approaching hepatization.

2d Stage — Hepatization. — The lung no longer crepitates upon pressure, because no air is contained within it; it has acquired the weight and firmness of the liver, and hence the term hepatization.

The colour of the external surface of the hepatised lung is often less livid than in the state of engorgement: internally it varies from violet to a deep blood-red tint: these colours are often so intermingled and shaded, that the cut surface presents the mottled aspect of certain marbles, or granites, and the similarity is rendered still greater by the inflamed lung being spotted irregularly by the black pulmonary matter, looking like small spots of mica disseminated within it. The branches of the bronchi and blood-vessels are seen ramifying over the surface of the section, and the interlobular cellular tissue becomes more distinct than in the natural state; it is whiter, and does not appear to participate in the inflammation.

There is no exudation from the section of a hepatised lung; but a thick and red-coloured fluid, in which can be frequently distinguished a matter still thicker, opaque, white, and puriform, may be scraped off by a scalpel.

If the hepatised lung be torn, or a thin slice of it be examined by a strong transmitted light, its texture appears granular, and the grains are small, red, round, or ovoid, and somewhat flattened: these are doubtless the air vesicles, rendered solid by the increased thickness of their parietes, and the infarction of their cavities.

When a lung is entirely hepatized, it appears,

on a first view, to be increased in volume, but it really is not so : this appearance arises from the solid organ containing no longer any air, and consequently not collapsing on the opening of the chest.

It sometimes happens, especially in children, that small patches of hepatisation occur here and there in the lungs, confined to a lobule, the surrounding lobules being perfectly healthy, or only slightly infiltrated. This state is called *Lobular Pneumonia*.

3d Stage — Purulent Infiltration. — The pulmonary tissue preserving still the same firmness and granular structure, as described in the second stage, becomes of a pale or light straw colour. At first, slight yellow spots of concrete pus are disseminated here and there, and render the shades upon the hepatised surface still more varied, irregular, and mixed. These points enlarge and unite until the whole becomes of a straw colour. A yellow, opaque, viscous, and puriform matter of a faint smell, may be scraped off, and the substance of the lung now becomes more soft and humid, and may easily be penetrated by the fingers. The granular structure finally disappears entirely.

In old persons, the lungs have always a dark aspect ; this arises from the quantity of black pulmonary matter contained within them : when, therefore, these organs are in a state of purulent infiltration in advanced life, this black colour, by its mixing with the yellow, gives a gray tint, very different in aspect from the same condition of lungs in young persons. This has been called *gray hepatization*. In adolescence and infancy,

the infiltrated concrete pus is of a fine whitish-yellow, and, as it softens, an unctuous matter may be scraped off, which has been mistaken for fat.

4th Stage — Vomica, or Abscess. — It is commonly supposed that abscess of the lung is of frequent occurrence, as a result of peripneumonia: nothing is, however, more rare. Laënnec, during twenty years, only met with five or six instances in many hundred autopsies. My friend, Mr. Langstaff, does not possess, in his splendid collection, a single specimen. I have met with none, after twelve years' habitude in post-mortem examinations. I here show you, however, a preparation of this disease, which I found in our museum, and which I believe has been there for many years. No doubt, tubercular excavations have been frequently mistaken for peripneumonic vomicae. I have seen what may be called pseudo-vomicae, formed by an inexpert anatomist, tearing away a lung adhering to the costal pleura, and plunging his fingers into the organ softened by purulent infiltration.

Laënnec describes a case in which he found an abscess situated at the middle and anterior part of the lung; its form was elongated and flattened: in proceeding from its centre, the liquid pus changed into a purulent detritus, then into a firmer tissue, still fully infiltrated with pus, and, finally, at half an inch farther, the lung was in the ordinary state of purulent infiltration. Andral mentions another instance, in which, towards the middle part of the inferior lobe, there was found a sort of "bouillie," containing, in its centre, true pus; the pulmonary

substance immediately around it was in a state of detritus, and beyond that, of purulent infiltration.

5th Stage — Gangrene. — Laënnec has described this disease in a special article : I think it best, however, to place it here, for the sake of arrangement, although it may be considered doubtful whether gangrene is ever a consequence of common inflammation of the lung. It is probable, but not certain, that it is essentially and primitively a gangrenous affection, as anthrax, malign pustule, &c.

There are two anatomical forms of this affection ; the one non-circumscribed, the other circumscribed.

a. Non-circumscribed Gangrene. — This is one of the rarest forms of organic diseases. Laënnec saw but two cases in twenty-four years. You have before you, gentlemen, a beautiful preparation of the lung in this state.

The pulmonary structure is more humid, and easier to tear, than in the natural state ; it is of the same density as in peripneumonic or cadaveric, or serous engorgements of its tissue. Its colour varies from shades of dirty white and slight green to a dark or almost blackish green, sometimes combined with a mixture of brown or dirty yellowish-brown : these various tints are often irregularly mixed. The lung, also, around the gangrenous part, is infiltrated with sanguineous serum, and is, in fact, in a state of peripneumonic *engorgement* ; the gangrenous part falls into a state of putrid deliquium. You perceive that a large portion of this lung hangs in filaments. A sanious fluid, of a greenish-black tint, oftentimes mixed with blood,

and possessing a most horrible fœtor, flows from the diseased mass.

The non-circumscribed gangrene occupies always the largest portion of a lobe, and frequently a whole lung. In some points the healthy pulmonary structure confounds itself insensibly with the gangrenous parts; in others, the lung is in a state of peripneumonic engorgement around the disease: rarely, and only in a few points, is it hepatised.

b. Circumscribed Gangrene. — This species differs from the preceding, by its occupying only a small part of a lung; by its slight tendency to invade the surrounding parts; and by its being a more chronic affection.

Circumscribed gangrene may be considered in relation to three states: — 1st, gangrenous eschar; 2d, deliquescent sphacelus; 3d, excavation resulting from the evacuation of the gangrenous matter.

1. *Gangrenous Eschar.* — A gangrenous eschar is irregular in its form and size; its colour is greenish-black; its texture more humid, compact, and hard than that of the healthy lung. It presents the appearance of an eschar formed upon the skin by the action of lunar caustic, and has a distinct gangrenous odour. The surrounding lung, to a certain extent, is in a state of engorgement or hepatization.

2. *Deliquescent Sphacelus.* — The eschar gradually becomes blacker, or brown, or yellow; it softens into a filamentous mass, and remains sunk in a depression formed by the destruction of the surrounding mortified parts.

More commonly the eschar softens into a kind of

putrid "bouillie," of a dirty green colour, sanguineous, and excessively fœtid. This matter bursts into the nearest bronchial tubes, is gradually evacuated, and leaves an ulcerous excavation.

3. *Gangrenous Excavation.* — The pulmonary tissue surrounding the excavation is infiltrated with a dark-coloured fœtid serosity ; after several days, points of hepatization appear disseminated within it. The colour of the engorged lung is reddish-black, and the organ where diseased contains but very little air.

When the eschar separates, the parietes of the cavity become lined with a false membrane of a gray or dirty yellow colour. This membrane is opaque, soft, and secretes a turbid pus, or black sanies, of a gangrenous smell. Sometimes the false membrane is produced even before the separation of the eschar, forming a line of demarcation between the dead and living parts.

Often the pseudo-membrane does not exist ; and then the fœtid pus is secreted from the parietes of the ulcer itself. These parietes are then firm, creak when cut with a scalpel, and are of a certain dryness. If incised, the surface is seen granular. This state of the lung rarely extends more than half an inch or an inch from the excavation, although it has been seen to occupy the whole lobe in which the cavity is situated. In other cases the parietes are soft, as if fungoid and putrilagenous : large blood-vessels are occasionally seen traversing the cavity, denuded and isolated, but intact ; at other times these vessels are partially destroyed, and their open mouths give occasion to a hæ-

morrhagy, which fills the excavation with clots of blood, and may produce a fatal hæmoptysis.

The decomposed gangrenous eschar occasionally opens into the pleura, and causes pleuritis, accompanied by pneumo-thorax: not infrequently, a series of gangrenous excavations exist, more or less communicating with each other, and often establishing a direct passage for the air from the trachea into the pleuritic cavity.

State of the Bronchi in Peripneumonia. — The mucous membrane of the bronchi is usually highly reddened in peripneumonia, although rarely swollen: when purulent infiltration supervenes, it is rendered sometimes pale, or of a more intense and violet red; and its tissue softens.

Situation of Peripneumonia. — Peripneumonia almost always commences at the lower lobes of the lungs, and has a strong tendency to invade the different portions of the organ from below upwards: of course the succession of morbid changes will therefore occur soonest in the inferior lobes; so that supposing a whole lung to be inflamed, and a perpendicular section to be made of it, from its apex to its base, the lower part will be found in a state of purulent infiltration, the middle in that of hepatization, and the superior engorged with bloody serum. You must always observe, however, that the lines of demarcation of these states are rarely perfectly marked and distinct, but that they run into each other by an insensible mixture or gradation.

Anatomical Characters of the Resolution of Peripneumonia: Engorgement. — The red serous fluid infiltrated into the tissue of the lung is ab-

sorbed, and the pulmonary structure becomes as dry as usual; a colourless serosity, however, frequently succeeds to the bloody serum, and remains for an uncertain time.

Hepatization.—The hardened parts first become pale, and pass from a red state to a gray or violet, and then to the natural colour; but they frequently remain somewhat redder after they become permeable to the air. While these changes of colour succeed each other, the lung loses its hardness, becomes more humid, and appears to contain more serosity than blood; which serosity becomes gradually more and more spumous.

The granular aspect of the hepatised lung gives way to the reappearance of the aërian vesicles. The diseased parts remain for some time more firm, elastic, and heavier than natural, no doubt from the hypertrophy of the parietes of the air-cells. The resolution does not proceed equally, for some points may be seen here and there hepatised in their centres, whilst their circumference is confounded by an insensible gradation of the inflammatory engorgement into the healthy structure.

Purulent Infiltration.—When the disease has arrived even at this stage, Laënnec affirms that resolution may take place without any disorganisation of the pulmonary substance. At the commencement of the resolution the yellow colour of the lung becomes paler and whiter; the pus is mixed with serosity, which contains a little air; the pus gradually diminishes, and puts on a grumous appearance; the air-cells become again apparent; the lung now feels firmer than in peripneumonic or

serous engorgement, and it crepitates slightly under the finger. The surface of a section is of a very pale dirty yellow, or green. If the resolution is very far advanced, this tint alone remains, and the pulmonary tissue is only slightly infiltrated with serosity, which finally is absorbed.

Duration of the different stages of Peripneumonia. — This disease is highly acute in its nature, rapid in its course, and requires the prompt use of remedial means; its duration is nevertheless variable. Laënnec has seen the state of engorgement last seven or eight days, and invading the whole of one lung, and part of another, before any portion was hepatized. This was the case in the influenza of 1803 and 1804, and I had repeated occasions to see the same in that of last year. In other instances, and particularly in weak and aged persons, or when combined with any other serious malady, the inflammation passes into the state of purulent infiltration in thirty-six, or even twenty-four hours.

Usually the disease runs the following course: the engorgement lasts from twelve hours to three days before the hepatization is complete; the hepatization lasts three days before points of purulent infiltration show themselves; and from that time until the pus is softened, it varies from two to six days.

This duration of the different stages of peripneumonia must be greatly changed by the mode of treatment, and the first and second stage thereby greatly prolonged. Thus I have seen several instances of the lung remaining hepatized for many weeks.

This, gentlemen, is the description given by Laënnec, of the various anatomical conditions of the lung in peripneumonia and gangrene of that organ. It is so perfectly accurate, that I have thought it right to give it almost in his very words.

LECTURE XI.

DISEASES OF THE PARENCHYMATOUS TISSUE OF THE LUNGS.

PERIPNEUMONIA — *continued.*

Local Signs. — *Engorgement.* — Crepitation — when absent — where heard. — *Percussion.* — *Hepatization.* — Respiratory Murmur. — Bronchophony. — Bronchial Respiration. — Rhoncus. — Blowing Respiration. — “*Souffle Voilé.*” — Puerile Respiration. — *Percussion.* — *Purulent Infiltration.* — Vomica. — *Gangrene.* — *Functional Signs.* — Pain. — Dyspnœa. — Cough. — Expectoration. — Pneumonic Sputa. — Gangrenous Sputa. — *General Signs.* — *Engorgement.* — Fever. — *Hepatization.* — Purulent Infiltration. — Vomica. — Gangrene.

Combination with other diseases. — *Causes.*

Treatment. — *Engorgement.* — Bleeding. — Tartar Emetic. — Mercury. — Blisters. — Evacuants. — Alkaline Plan. — Expectorants. — Regimen. — *Hepatization.* — Mercury. — Diet. — Repose. — Counter-irritants. — *Gangrene.*

Signs of Engorgement.

WE now proceed to consider the signs of peripneumonia, detailing them in relation to the order in which we have described the anatomical stages of the disease.

Local Signs. — 1st, *Engorgement* — *Crepitation.* — The lung, in the first stage of inflammation, is gorged with sanguineous serum. You recollect the proposition I advanced in treating of auscultation, — that whenever fluid, combined with air, existed in the tissue of a lung, it produced a crepitating sound. This sound is pathognomonic of

engorgement. It is analogous to the bursting of a number of small, equal-sized, and slightly humid bullæ in rapid succession: it is exceedingly distinct if the engorgement be near the surface of the lung, but becomes much less so if it be an inch or two from it; if farther still, the crepitus is no longer heard: so that it sometimes occurs, when the inflammation commences in the centre of the organ, that no local signs whatever are appreciable, although the functional and general symptoms of peripneumonia be present. It, however, almost invariably happens that the engorgement arrives ultimately at the pulmonary surface, often in a few hours, or, at most, in a day or two; and then the crepitation, which was masked by the sound of the respiratory murmur, becomes perfectly distinct, and the disease is no longer latent or obscure.

The crepitation is heard first in the lower lobes of the lungs, because the engorgement begins there. As hepatization commences, the bullæ seem more humid, are less equal in size, and succeed each other with less rapidity. The respiratory murmur gradually diminishes, till it finally ceases with the crepitation, as the organ solidifies.

Percussion. — If the engorgement occupy but a small portion of a lobe, then there is no difference in the sounds on percussion: indeed, if even the engorgement be considerable, it requires an experienced hand and ear to detect a difference, because the inflamed lung still contains a considerable quantity of air. As the liver often mounts high in the chest on the right side, no inference ought to be drawn from eliciting a dull sound from the inferior

ribs of that side, as it may depend upon the presence of that viscus, as I have before explained.

2d Stage — Hepatization. — Neither the natural respiratory murmur nor crepitation can now be heard, because no air exists in the vesicles to produce the first sound, nor is there a mixture of air and serous fluid in them to produce the second. Occasionally *bronchophony* takes place, particularly when the inflammation attacks those parts of the lungs where the bronchial tubes are the largest, as at their roots or upper lobes: this phenomenon is very evident when the disease appears at the surface of the organ, and is indistinct when in the centre.

When, combined with hepatization, a thin layer of fluid is interposed between the pleuræ, the bronchophony becomes louder, in consequence of its combination with *œgophony*. These sounds are always most distinct at those points of the chest which correspond to the roots of the lungs, because there the bronchial tubes are largest; but as these tubes become of less diameter in the inferior lobes, there the bronchophony is less audible.

The bronchial respiration and cough always accompany bronchophony; sometimes these sounds are heard when the latter is not. It appears that the two first signs may be distinguished when the hepatization is deep-seated, but the union of the whole only when it is superficial.

The solid lung renders the wheezing incident to accidental coexisting catarrhs much more distinct.

When the hepatization surrounds a large bronchial tube near the surface of the organ, particularly at its roots or superior lobes, then the bronchophony simulates pectoriloquy, and is often also accom-

panied by the sensation of blowing into the ear, described in the lecture upon auscultation. If a slight portion of non-hepatised lung be placed between the parietes of the chest and the resounding bronchial tube, then the "souffle voilé" is heard.

As the inflammation extends, the crepitation extends also. Thus the signs of engorgement will occur in the first instance in the lower lobe, to give way to those of hepatisation; then they will be heard surmounting the parts hepatised, to give way also to those of consolidation; and so on successively, until the whole of the lung becomes solidified.

In consequence of the power of respiration being lost in the inflamed lung, the necessity for respiration increases in the other, so that in the latter the respiratory murmur becomes extremely loud, constituting the *puerile* respiration. This condition of the respiratory murmur in the healthy lung is always distinct in proportion to the extent and completeness of the hepatisation.

The sound is dull upon percussion, except the disease attacks the centre of the lung, and then sufficient air will be interposed between the solid parts of the organ and the parietes of the chest to produce a good sound. Percussion on the lower part of the right side of the chest cannot be depended upon, from the presence of the liver, as I have before indicated. If, however, you have been enabled to follow the disease from the state of engorgement, then you can unhesitatingly say, if the sound be subsequently dull, that it depends upon hepatization, and not upon the presence of the liver.

3d Stage — Purulent Infiltration. — There are no local signs of this state differing from those of hepatization. The same physical state exists here, as far as relates to the solidification of the lung, and consequently the same physical signs present themselves.

4th Stage — Vomica. — But when the concrete pus softens, and is evacuated; at the point of the chest corresponding to the vomica a noisy mucous rattle occurs, which is gradually transformed upon inspiration and coughing into a cavernous sound. Pectoriloquy also appears. The sensation of blowing to and from the ear occurs, if the abscess be near the parietes of the chest, and the “souffle voilé” becomes distinct, if the walls of the cavity be thin and soft, and not adherent to the costal pleura.

5th Stage — Gangrene. — I have met with fourteen or fifteen instances of gangrene of the lung, and in every case crepitation was discoverable; doubtless, because of the accompanying engorgement of the lung. When the organ commences to break down and form an excavation, then the mucous rattle is very audible: it gives way at last to complete pectoriloquy, with cavernous rhonchus and cough. If the excavation opens at once into the bronchi and pleura, then the metallic tinkling, or the amphoric resonance, are produced.

Local Signs of the Resolution of Peripneumonia.

1st Stage — Engorgement. — You will recollect, gentlemen, that when the resolution of the engorged lung occurs, that the red-coloured serum gives

place to a colourless fluid, infiltrating the air-cells; consequently, although the inflammation has diminished, or even disappeared, yet the crepitation will remain for some time; it finally, however, becomes less distinct, until at last it is totally lost, leaving the sound of the natural respiratory murmur.

2d Stage — Hepatization. — When resolution takes place from hepatization, you will remember also that the solidified lung returns into a state of serous engorgement; the crepitation will therefore reappear, constituting the "*rhonchus crepitans redux*" of Laënnec. This sign, accompanied by the re-appearance of the respiratory murmur, and the absence of the dull sound on percussion, is pathognomonic of the resolution of hepatization.

3d Stage — Purulent Infiltration. — Laennec asserts that the rhonchus crepitans equally announces the resolution of the disease even in this state, but that it is ordinarily preceded by mucous or sub-mucous rattle, indicating the softening of a part of the pus. He says, also, that the respiratory murmur always appears more tardily.

When peripneumonia has attacked a large portion of the lung, the resolution occurs first in those parts which were the last affected. The resolution has sometimes, although, I believe, very rarely, taken place in the inverse order.

4th Stage — Vomica. — We know but too little of this stage to be able to give the exact signs of its resolution. There can be but little doubt that when it does happen, all the signs previously described must gradually disappear, and give way to the natural respiratory murmur.

5th Stage — Gangrene. — Resolution may take place even in this stage : the serum engorging the lung totally disappears, and with it its sign of crepitation. I have found pectoriloquy disappear also — no doubt from the union of the sides of the gangrenous excavation. A young sailor, in in the wards of this hospital, was the subject of this formidable disease : we were enabled to trace all the signs, almost from the commencement, to excavation of the pulmonary substance. In four or five months he was discharged. In a year afterwards he returned from a voyage, and presented himself at the hospital in robust health. The previously existing pectoriloquy had completely disappeared.

Functional Signs of Peripneumonia.

Pain. — This is a very uncertain symptom : sometimes it occurs, sometimes it does not ; when present, it usually is deeply seated, and of considerable extent ; occasionally it is found only in a fixed point, and if it be acute, it generally arises from pleuritic combination.

Dyspnœa. — This symptom varies according to the quantity of lung affected : if the inflammation be slight, the number of respirations are but slightly increased ; if it be very extensive, I have seen, especially in children, the number augment to sixty or seventy in a minute, and the distress and danger of suffocation then become extreme.

Cough is often present, although occasionally it is so slight that the patient does not notice it.

Expectoration.—When expectoration takes place, — for it is not of constant occurrence, — it becomes so characteristic, that Laënnec believed that from it alone the disease might be detected. The sputa, which he calls *glutinous* or *pneumonic*, collected in a flat open vessel, as a plate, form a mass so tenacious and viscous, that if the vessel be reversed when full, the expectorated fluid separates, or is detached from it, with difficulty: it trembles, something like a jelly, upon agitation. The colour of the sputa varies — it may be red, or rusty, sea green, orange, saffron, yellow, or of a dull green: these colours are often intermixed. The expectoration “en masse” has something of the transparency of horn, or of white of egg coloured; it contains bullæ of air of unequal size, and in large quantities. The state of expectoration seems to take place only in the first stage of peripneumonia or engorgement.

But these characters are not always so distinct, for sometimes the sputa are less viscous, less coloured, and less spumous; at others, a small quantity of fawn-coloured glutinous matter may be seen mixed in a considerable mass of mucous or pituitous fluid: occasionally these glutinous sputa occur only in the beginning of the disease, and then but in small quantity.

During hepatization, the sputa vary in appearance: they are composed of viscous pituita, or white or semi-opaque yellow mucus: they are still more mucous in character when purulent infiltration supervenes; sometimes mixed with yellowish striæ, indicating, probably, the presence of pus:

rarely, indeed, is the whole of the matter expectorated puriform.

The expectoration in gangrene of the lung is so characteristic, that when once met with, it will be impossible to mistake it; for the odour is so fœtid and peculiar, that it is almost insupportable. I have known the breath of a patient infect the air of the whole house, so that it was hardly bearable by its inmates. The colour of the gangrenous sputa varies exceedingly: it is sometimes of a bright yellow, or green, brown, or of a milky white. I have seen, in two instances, a yellowish-green substance, mixed in streaks or large spots, with a matter as black as ink. The consistence of the expectoration is that of pus, although sometimes it is much thinner.

General Signs of Peripneumonia.

1st Stage — Engorgement. — It is to this state of the lung that the term inflammatory ought, I think, really to be restricted, for hepatization, purulent infiltration, and vomica are merely consequences of engorgement, as much as abscess is a consequence or termination of subcutaneous cellular inflammation. The general as well as local signs of engorgement are also very different from those of the other stages of peripneumonia.

Thus, this stage is ushered in by an access of acute inflammatory fever, usually intense, except the peripneumonia be slight and partial. The face becomes coloured of a vivid red in the first instance: if the disease be severe, the redness becomes mixed with a livid, slaty, or bluish tint, consequent upon

venous congestion. The pulse is rapid, full, and hard; the blood usually presents a thick buffy coat, and the crassamentum assumes a cup-like form. As the inflammation extends, various congestions occur, but principally towards the cerebrum, so that delirium is often induced in young persons, coma in old, and convulsions frequently in children. The tongue is often intensely red. The urine is high-coloured, and small in quantity; and, towards the termination of the febrile symptoms, diarrhœa occasionally supervenes.

This symptomatic fever ceases upon the diminution of the inflammation of the lungs. If, however, the febrile state depends upon other coexisting causes, or if it be of the character called essential, then it often remains with unabated force after the peripneumonia has diminished or even ceased.

2d — *Hepaticization*. — Usually, the inflammatory fever now disappears, and dyspnœa alone remains, although commonly with diminished intensity: the difficulty of breathing also soon ceases, as the resolution of the hepaticized lung takes place. But it will occasionally happen, gentlemen, that you may meet with cases supposed to be consumptive, but in which the symptoms depend upon the long duration of the hepaticization: thus there is dyspnœa, cough, expectoration, emaciation, an habitually rapid pulse, a slight fever in the evening, and even occasionally night sweats, although rarely so profuse as in phthisis. Examine your patient, and you will now find the value of auscultation, by discovering that there are no local signs of tubercular disease, but that one or other of the lungs is hepa-

tised, perhaps to half or more of its mass. This state is often *curable*, and I have no doubt that many patients have been considered to have recovered from pulmonary phthisis, when they have really only been affected with hepatization of the lungs.

3d and 4th Stage — Purulent Infiltration and Vomica. — The general symptoms of these states have not been sufficiently observed, but most probably they are similar to those of chronic hepatization.

5th Stage — Gangrene. — The general signs of gangrene vary; for they may be of exactly opposed characters: thus, when the disease attacks the young and plethoric, it may be accompanied by all the symptoms of inflammatory fever; if the weak or aged, it is commonly combined with a general asthenic condition of the system, indicated by extreme prostration of strength, and great anxiety. I have frequently seen the dyspnoea very slight, and bearing no proportion to the extent of the local, or the intensity of the general signs.

Combination of Peripneumonia with other Diseases. — Peripneumonia may be combined with pleurisy, with hæmoptysis, and with œdema pulmonum; all of which complications we shall speak of when we arrive at the consideration of these diseases.

The sero-sanguineous congestion of the lungs, which occurs so commonly in the agony of death, is often transformed into peripneumonia, if that agony be long.

Peripneumonia and catarrh are not infrequently

combined; this combination often prevailed in the influenza of last year, and in that of 1803. This is the *peripneumonia notha* of the old authors.

Tubercular disease is occasionally accompanied by inflammation of the lungs. This will be particularly indicated by the supervention of any sudden and unusual febrile attack upon the ordinary symptoms of phthisis; it is accompanied also by as sudden an increase of the dyspnœa.

There are few fevers in which peripneumonia may not occur during their course, as in measles, particularly towards the disappearance of the eruption. It exists often in the course of confluent small-pox, or erysipelatous fevers. I have seen peripneumonia come on during the progress of continued fevers, either of the synochoid or typhoid forms; and such complications are by no means infrequent.

Inflammation of the lungs occasionally takes place during the progress of pertussis: two years ago this complication appeared epidemically.

Causes of Peripneumonia.

Individual. — Although the disease is always more intense in young persons of sanguine temperaments, yet it occurs much more commonly in old people, in whom, according to Laënnec, it runs sooner into the suppurative stage.

Peripneumonia very frequently attacks infants; it usually exists then in the lobular form, and the disease is generally fatal to them in the stage of engorgement.

External. — The external causes are the same as those of other inflammations, — as cold, sudden changes of temperature, &c. The bite of the rattlesnake is said to induce inflammation of the lungs. The disease is most common in winter.

The causes of gangrene of the lung are at present hidden in the most complete obscurity. I believe I can say to a certainty that it is not a contagious disease.

Treatment of Peripneumonia.

I shall speak of the various remedies that have been employed in what I conceive to be the order of their importance.

1st Stage — Engorgement. — Bleeding. — There is scarcely any remedy so powerful in its effect upon peripneumonia as bleeding. It is in the stage of engorgement, particularly when accompanied by general febrile excitement, that blood should be abstracted promptly and freely; and you will find, even as it flows, that the dyspnœa will diminish, and the patient express himself relieved. It is the practice in this country to bleed the patient in an upright posture, and to make a large orifice in the vein, so that fainting may be speedily induced. Experience has shewn that a greater impression is made upon inflammatory diseases by this method, and that a cure can be thus effected by a less loss of the vital fluid than if a larger quantity be abstracted in a small and slow-flowing stream. It often happens after a single bleeding, that the dyspnœa recurs with febrile reaction, and a full pulse; it will then be necessary to repeat the venesection,

perhaps even three or four times, before the inflammation be subdued. It is impossible to give any other general rule as to the extent you may bleed, but that you must be guided by the intensity of the symptoms, and by the strength of the individual.

Leeches and cupping may frequently be used with advantage, when the weakness of the system contra-indicates a general bleeding.

Tartar emetic. — Antimony, in its various forms of preparation, has been long used in medicine, and as an emetic, particularly, in peripneumonia. But it was reserved for Rasori to point out the special advantage of the tartar emetic in inflammation of the lungs. I shall not enter here into the theory of *contro stimulus*, by which he attempts to explain its action, but proceed at once to the description of the mode in which you should use it, premising by saying that there is no medicament so decisive in its action in combatting this disease.

Laënnec, immediately after bleeding his patient, gave a grain of tartar emetic in two ounces and a half of a slight and cold infusion of orange flowers, sweetened with half an ounce of syrup of mallows, or orange flowers. He repeated this dose every two hours, until the patient had taken six grains, and then suspended the use of the remedy for seven or eight hours, if the symptoms were not urgent, or there was a disposition to sleep. If the symptoms were distressing, and the head became affected, he repeated the medicine in the same way, until an amelioration took place, sometimes, in severe cases, gradually increasing the dose to as much as two grains and a half.

Many patients bear the tartar emetic without its producing either vomiting or purging: most, however, vomit two or three times, and are purged during the first day, but commonly afterwards they *tolerate* the remedy perfectly. If they do not then, the addition of a little syrup of poppies, or a few drops of the tinct. opii, with the first few doses, occasions it to be borne easily.

If the first dose produce vomiting, the subsequent doses will doubtless often produce the same by mere association of ideas, and the patient will have considerable repugnance to taking his medicine. It is best to obviate this: I, therefore, after freely bleeding, commence with one third of a grain of tartar emetic in half a wine glass-full of water, adding a few drops of tinct. opii, or syrup of poppies. I give two doses at the interval of one hour from each other; I then increase the quantity by another third of a grain for the next two succeeding hours, omitting the opiate if no sickness be induced, and so on successively, by the addition of a third of a grain every two hours, until I arrive at two grains. I have not exceeded that quantity, although I have continued it for many days without producing any injurious effect.

You will often find, gentlemen, in following this plan, the disease will be considerably relieved in three or four hours, sometimes, however, not for twenty-four or thirty-six hours. According to my experience, the tartar emetic always acts best when it produces no effect, except upon the inflammation itself; that is, when it occasions neither vomiting, purging, or depression of the powers of the system. It has been supposed by many, but erroneously,

that it cannot subdue the disease, except by previously producing these effects.

There is, perhaps, no stage of peripneumonia in which the tartar emetic may not be employed with some advantage; but it is certainly of most use during the engorgement of the lung. You will be called, gentlemen, to cases in which bleeding, perhaps, has been already carried to its fullest practicable extent, or where, in old and cachectic persons, venesection is no longer admissible; then you will find the tartar emetic, carefully administered, produce frequently the most astonishing curative effects. I have frequently left patients under the impression that their recovery was almost impossible, and yet have found that the continuous use of this medicament has snatched them from the jaws of death.

Laënnec recommends that the tartar emetic should be continued until all signs of crepitation be merged into the natural respiratory murmur. This is not my practice: I am guided by the cessation of dyspnœa alone; for, as he has himself observed, a non-inflammatory serous infiltration of the pulmonary tissue will supervene, and give rise to crepitation, after the inflammatory engorgement has totally disappeared.

Mercury. — Before I had occasion to see the admirable effects of tartar emetic in the first stage of peripneumonia, I freely employed mercury after bleeding: this remedy sometimes produced a good effect, but it very frequently did not: it, however, is of great use when the lungs become hepatised, as I shall presently describe. In comparing the effects of tartar emetic and mercury upon acute

inflammations, I have no hesitation in saying that the first seems to act most energetically upon those of the parenchymatous tissues of the organs, the second upon those of the serous membranes.

Blisters. — Counter-irritants ought not to be applied in the first stage of the disease, especially if there be much febrile irritation; they only tend to increase the distress, and produce rarely any effect in diminishing the dyspnœa: when, however, the heat of the skin diminishes, the pulse becomes less frequent and full, and a sense of infarction or oppression exists in the chest, with difficult expectoration, then blisters, or other counter-irritants, are of great utility.

Evacuants. — It is useful to keep the bowels gently open, but free purging is rarely of service. Emetics were formerly employed, and, it is said, with much success; but, most probably, that success must have arisen in cases where the peripneumonia was combined with derangements of the digestive organs.

Alkaline Plan. — Under the supposition that inflammation depended upon a certain plasticity and thickness of the blood, alkalies were proposed for the purpose of attenuating that fluid: thus the subcarbonates of potash, soda, and ammonia, medicinal soaps, the purgative neutral salts, were administered; but these remedies, as well as the plastic theory, have become obsolete: the polygala virginiae has also been highly vaunted; but I have never seen it produce any good effect.

Expectorants. — The ipecacuanha is an useful expectorant in all the stages of peripneumonia, especially in children: the squills or ammoniacum

should not be used, except in old or cachectic persons, and then only towards the resolution of the disease.

Regimen. — The regimen during the state of engorgement must be strictly antiphlogistic ; no stimulants should be given, but the patient should have a free supply of cool drinks. The room ought to be kept cool ; for if its temperature be high, it will certainly tend to aggravate the disease. I need not say that the most perfect repose should be enjoined, and that the patient should be permitted to speak as little as possible.

Hepatization of the Lung. — Hepatization of the lungs occasionally becomes a chronic affection, lasts for many weeks, and produces a train of symptoms frequently simulating phthisis. You will recollect, gentlemen, that in this case the air-cells are filled with a solid matter, and our first indication is to occasion its absorption. I have tried the tartar emetic, iodine, and mercury, with this view, but I have found the last incomparably the best remedy, if properly administered.

As the constitution is usually much impaired in this state of the lung, when the disease has become chronic, and as there is frequently a strong tendency to gastric and intestinal irritation, you will find the blue pill in five grain doses, combined with a third of a grain of opium, three times a day, one of the best means of affecting the system : if, however, there be purging, then rubbing in the mercurial ointment is still better. These means should be carried on until the mouth be rendered slightly tender, and it should be kept so for two or three weeks : the remedy should be then discontinued for a week or two, otherwise it will irritate

and exhaust the patient; you will then recommence it, and this alternating plan must be carried on until the resolution of the lung be complete.

The patient during this time should have a moderately nutritious diet: thus I allow a little meat once a day, and a free use of milk; but no other drink stronger than water. You should watch closely the state of the circulation, general appearance, and dyspnœa, so that if the pulse becomes full, and the difficulty of breathing increases, a few ounces of blood may be abstracted, and the diet diminished, endeavouring thereby to establish an equilibrium between the quantity of blood sent into the lung, and the quantity of air the cells contain.

The next circumstance you should attend to, is to keep your patient in the most perfect state of repose, both in mind and body; allow no exercise, otherwise the blood will be driven into the diseased lung with too great rapidity: indeed, I always endeavour to make the patient understand the nature of his case, the objects I have in view, and, consequently the positive necessity of absolute repose, and you will generally find he will readily submit.

Counter-Irritants. — I usually also keep a blister open from time to time on the surface of the chest of the side affected.

Gangrene of the lungs requires two modes of treatment, varying according to the general state of the system. In some cases, although the least frequent, it is accompanied with inflammatory fever, and then you must treat it on the antiphlogistic plan. But when you meet it combined with great asthenia, then the affection must be treated upon the tonic and stimulating methods, as by wine and bark, &c.

LECTURE XII.

DISEASES OF THE PARENCHYMATOUS STRUCTURE
OF THE LUNGS.

EMPHYSEMA PULMONUM.

Definition. — Species. — 1. *Vesicular Emphysema*. — 2. *Interlobular Emphysema*.

Vesicular Emphysema. — Morbid Anatomy. — Vesicles, Rupture of them. — Projection. — Form. — Pediculated. — Orifices. — Irregular Vesicles. — Rupture of Internal Substance of Lung. — Clots of Blood. — State of Bronchial Tubes. — Modes of preparing Specimens. — Extent of the Disease. — Disease described by several Authors. — Causes. — Dry Catarrh. — Forcible Inspirations. — Excess of Puerile Respiration. — Case.

Signs. — *Functional and General*. — Dyspnœa, peculiar Character of. — Dry Asthma. — Colour of the Skin, &c. — Sleep, tendency to. — Cough. — Expectoration. — Hypertrophy and Dilatation of the Heart. *Local Signs*. — Form of the Chest. — Percussion. — Respiratory Murmur. — Sibilation, dry Crepitation. — Progress of Disease. — Treatment.

Interlobular Emphysema. — An acute Disease.

Morbid Anatomy. — Causes. — Violent Efforts : spontaneous Exhalations of Air. — *Local Signs*. — Dry Crepitation. — “Frottement ascendant et descendant.” — Percussion. — *Functional Signs*. — Sudden Dyspnœa, &c. — Treatment.

WE define emphysema pulmonum to be a dilatation of the air-cells of the lungs, or an infiltration of air into the cellular substance which separates the lobules of that organ from each other. The first species may be called *vesicular emphysema*, the second *interlobular emphysema*.

VESICULAR EMPHYSEMA.

Morbid anatomy. — The size of the aërian vesicles increases from the slightest addition to their volume, to that of a grain of millet-seed — to the kernel of a cherry — to a bean ; or they may enlarge, in extreme cases, to a capacity sufficient to hold a pint of fluid. In the latter cases this great development is due to a rupture of a vast number of cells into each other ; for you may see the *débris* of their partitions upon the inside of the transparent parietes of the unilocular cavity formed by them.

The smaller dilated cells do not project beyond the surface of the lungs, the larger ones usually do ; the latter presenting the vesicular appearance of blisters. These large projecting cells are often globular, and sometimes pediculated ; but the pedicles are only formed by strangulations of the cells, where the vesicles emerge from the pulmonary substance ; and in cutting into them, and passing a probe through the pedicle, as large a cavity will be found embedded in the substance of the organ as there is projecting beyond it. At the bottom of the dilated cells you perceive several dark orifices, which are the openings of communication with the surrounding air vesicles. It is clear that these dilations do not depend upon extravasations of air, because of the continuation of their cavities into the substance of the lung, and because they cannot be pushed by the finger from point to point under the pleura. It, however, does occasionally happen that, in consequence of the rupture of the cells, the air passes into the interlobular cellular tissue,

and beneath the pleura, forming irregular vesicles of various sizes, which may be displaced by the finger; but they have no depressions in the substance of the lungs corresponding with them, like the previously described species.

Although it be rare for the air to permeate the interlobular cellular tissue, or the cellular substance surrounding the bronchial tubes and great blood-vessels, yet such may be the distention of the central aërian cells, that their bursting may occasion a rupture of the pulmonary tissue; then the part of the lung corresponding to the rupture will be seen largely swollen; and on cutting into it to a greater or less depth, a cavity will be found from whence air will escape, this cavity often containing a little blood, generally coagulated, but always small in quantity compared to the space in which it is contained.

The bronchial tubes, particularly the smaller ones, communicating with the dilated cells, are occasionally, although not frequently, dilated also.

There are two modes of preparing specimens of emphysema pulmonum; the one by forming a dry, the other a wet preparation. To make a dry preparation, the lung should be well distended by insufflation, and the bronchial tube, through which you have distended, should then be firmly tied, and the specimen be dried pretty quickly. You will soon find that your preparation will shrivel and contract, from the escape of air through some imperceptible orifice; it becomes, therefore, necessary to renew the insufflation during the drying several times. When quite dry, it should be varnished, and sections made by a sharp razor or scalpel, and the dilated cells will thereby be very well shown.

The other mode is effected by immersing the emphysematous lung in spirits of wine. A preparation of this sort requires a large weight to keep it down in the bottle, in consequence of the buoyancy of the organ from the increased quantity of air it contains.

Vesicular emphysema may affect a part of a lung, the whole of a lung, or both lungs. When the disease is extensive, the moment you elevate the sternum a part of the organ escapes from the chest, as if there was not sufficient room for it, and projects externally, instead of subsiding as it usually does. The lungs now feel to the hands like a down pillow; they give the impression of greater firmness than natural; the crepitation, upon pressure, is of a different character; and if an incision be made, and the air pressed out, a sound is produced like air passing slowly through a bellows. These phenomena probably arise from the communication between the air-cells and the bronchial tubes being less free, or from a diminished flexibility of the parietes of the vesicles, or, perhaps, from a combination of these two causes.

An emphysematous lung swims on water almost like a bladder distended with air. The pulmonary tissue is usually drier in an emphysematous than a healthy lung; exceptions to this are rare.

When one lung only is affected, it is much more voluminous than the other; in that case, it may press the mediastinum and the heart from their natural positions, and even enlarge the side of the chest corresponding to the diseased organ.

Emphysema pulmonum had been indistinctly seen by several authors, as Bonetus, Morgagni,

Van Swieten, and Storck. Sir John Floyer, in the appendix to his Treatise on Asthma, gives an account of the disease in a broken-winded mare. Ruysch and Vansalva had seen dilated air-cells; and Baillie has given a good description of them. Laënnec's account of the disease, which I have almost given literally, is, however, by far the most minute and correct.

Causes of vesicular emphysema. — Laënnec, in the year 1823, gave, in his lectures at the "Collège de France," the following explanation of the physical causes of this disease:—

Vesicular emphysema almost always develops itself in consequence of intense, extensive, or long-continued dry catarrh. Almost all asthmatics from that form of catarrh present, upon post-mortem examination, a dilatation, more or less marked, of the air-cells. I never saw any patient affected by dyspnœa, from any cause, of a few years' standing, without detecting this disease in various degrees. Laënnec observes, that in dry catarrh the small bronchial tubes are often completely obstructed, either by pearly sputa, or by the swelling of their mucous membrane: and, as the inspiratory muscles are strong and numerous, their action is sufficient to overcome this obstruction, so that the air can pass with more or less freedom into the cells, where it becomes, as it were, imprisoned, in consequence of the expiratory power being insufficient fully to force it back again through the obstruction. A long succession of inspiratory acts will, under these circumstances, at last permanently dilate the air-vessels to an extent proportionate to the degree and duration of the obstructing cause. We must

also recollect that the atmospheric air is introduced into the lungs at a temperature much lower than that of those organs, so that it dilates in proportion to the increased heat, and, consequently, must tend to dilate the cells in which that air is contained. It follows, from what has been said, that dry catarrh ought mechanically to produce vesicular emphysema, as much as chronic mucous catarrh leads to dilatation of the bronchial tubes.

There are some other causes which may produce this disease: thus players upon wind instruments are often obliged to retain the air forcibly, and for a long time, in the air-cells, and thereby occasion their dilatation. Certain other violent respiratory acts may also be considered as causes; but these I shall particularly refer to in speaking of interlobular emphysema.

Any cause, also, which prevents the free emergence of the air in expiration may produce vesicular emphysema: thus tumours developed in the bronchial or mediastinal glands, or aortic aneurisms compressing the bronchi; tumours of a certain size formed in the lung itself—as cysts, or masses of tubercles—will partially produce this effect.

There is another cause, which Laënnec has not noticed, which I have seen acting in two or three instances. In consequence of extensive disease in one lung, the puerile respiration sometimes becomes so extreme in the other, as to occasion the air-cells to dilate. The following remarkable case exemplifies this fact.

I was called to a child of five or six years of age, labouring under very severe dyspnœa of a few days' standing, in whom the face had become livid, the

pulse lost, and the extremities cold. Everything indicated approaching death; indeed, the child expired a few hours after I saw it. Such was the distress of the little sufferer, that I would not add to it by making a useless local examination of its chest. Upon a post-mortem investigation, we found that percussion afforded a very dull and fleshy sound on the left side, whilst the right was exceedingly sonorous. On opening the pectoral cavity, the left lung was found to be hepatized to nearly the whole of its extent,—a circumstance already sufficiently rare in so young a child. The right lung was depressed in the chest, and of nearly one third its original volume, and covered by large vesicles, presenting the appearance of a number of elevated blisters upon the surface of the organ. The space in the chest unoccupied by the lung was filled with air, part of which had escaped with a hissing noise, upon the first incision made through the ribs of the right side. The lung itself, when taken out, presented a most beautiful specimen of vesicular emphysema.

No doubt, in this case, the hepatization of the left lung had increased the necessity for respiration in the right,—a necessity already great from the age of the child. Its natural puerile respiration became so intensely developed, the energy of the function so extraordinarily great, as to largely dilate the air-cells of the healthy lung. Some vesicle must have ruptured, perhaps by a very minute orifice, so that a certain quantity of air passed in every inspiration into the cavity of the pleura, and, by its gradual accumulation, pressed upon the lung, and diminished it to the volume I have mentioned.

We had, therefore, in this case, vesicular emphysema of one lung, in consequence of hepatization of the other, and pneumo-thorax as the result of the emphysema.

SYMPTOMS OF VESICULAR EMPHYSEMA.

Functional and General Signs.—Dyspnœa invariably occurs when the disease has become somewhat considerable: the reason is obvious. The expiratory act cannot expel a sufficient quantity of air at each movement, so that too great a proportion remains in the cells, deprived of its oxygen, and preventing by its presence a complete renewal during inspiration. The mode of respiration is peculiar; in extreme cases, you will see the patient expand his chest in every direction, as if he were endeavouring, by a strong effort, to sweep out and displace the vitiated air contained in his lungs.

Various causes may augment the dyspnœa; as flatulence of the stomach or intestines, preventing the free descent of the diaphragm — emotions of the mind — exercises of the body, as running, hard working, &c., by which the blood is sent with too great rapidity to the lungs.

The dyspnœa finally becomes constant; but it will sometimes be considerably aggravated by a fresh inflammation attacking the already-obstructed bronchial tubes, or by a spasmodic action of them. When this occurs, then it constitutes a form of *dry asthma*, which we shall hereafter describe. After the asthmatic paroxysm is over, the ordinary state of dyspnœa returns.

As the blood does not flow freely through the

lungs, venous congestions have a tendency to form; thus, at last, the skin presents an earthy tint, often mixed with a dull violet or lead colour. The veins on the surface frequently enlarge, the lips appear thick and swollen, and of a leaden hue; and, according to my observations, emphysematous patients are more disposed to sleep, particularly after a meal, than others; probably from a greater disposition to temporary cerebral venous congestion.

There is generally habitual cough — often, however, infrequent, slight, and dry. If there be expectoration, it is usually viscous and transparent: the cough is rarely violent, or followed by mucous sputa.

The constant obstruction to the free passage of blood through the lung ultimately occasions hypertrophy and dilatation of the heart.

Local Signs.—When emphysema attacks one lung only, or when it exists much more considerably on one side than the other, then the diseased side is evidently the most voluminous. If both sides are equally affected, the chest presents at once a globular and a cylindrical form; being swollen before and behind, instead of being somewhat depressed in those situations.

By *percussion*, a much louder sound than natural is elicited, in consequence of the great accumulation of air in the lungs.

The respiratory murmur is very imperfectly heard in an emphysematous lung; now and then a sibilating sound, or a sound like the clicking of a small valve, may be distinguished, indicating the presence of pearly sputa.

But when the disease is intense, then there is a

sign which is pathognomonic : it consists in a species of dry crepitating sound, before described under the name of rhonchus crepitans with large bullæ ; it is a sound heard when the patient coughs or inspires strongly, like air blown into half-dried cellular tissue. It differs from the ordinary crepitation of peripneumonia, or œdema of the lungs, by its carrying with it the sensation of dryness, and by the greater size and inequality of the bullæ which compose it.

This phenomenon is rare, and of short duration : it is heard only for a few moments, and to a slight extent. It is much more common in interlobular emphysema : some patients feel a crackling at the point affected at the moment the crepitation is heard ; sometimes, though rarely, the vibration of the sound is communicated to the hand pressing the parietes of the chest.

Progress of the Disease.—This organic alteration of the cells most commonly arises from the grafting of acute dry catarrh upon the chronic form ; and dry catarrh being, of all others, that which is most accompanied by tumefaction of the bronchial mucous membrane, it may readily be conceived how an obstruction of the smaller tubes may produce a dilatation of the ærian vesicles.

The frequent returns of acute dry and latent catarrhs often induce an asthmatic paroxysm ; but these are not the sole causes of this affection, as we shall hereafter see.

If, upon chronic dry catarrh, the acute form supervenes, the dyspnœa becomes almost suffocating. This oppression diminishes if fever coexists, or a pituitous or mucous expectoration appears. The

pearly sputa become less tenacious, and are carried from the bronchial tubes by the more liquid secretion which accompanies the recent catarrh. But if the fresh accession of catarrh lead to no ultimate relief of the chronic disease, then the attack will be prolonged; it finally gradually diminishes, and often leaves the patient more oppressed than before. Violent attacks of dry asthma occur at long intervals during the first few years; but if the emphysema has been of long duration, as old age approaches, the accesses approximate more closely, and become of greater intensity; each of them increases the extent of the dilatation of the air-cells, which, in these cases, sometimes rupture, and produce the interlobular form of the disease.

Treatment.—We have but little to say upon this subject. The indications here are to subdue the dry catarrh, which is the mechanical cause of the emphysema, and then to diminish the volume of the enlarged vesicles. To fulfil the first indication, the same treatment must be adopted that I have recommended for dry catarrh, which it is needless here to recapitulate; I refer you to the lecture upon that subject. I know of no possible means of fulfilling the second indication, that is, to diminish the volume of the emphysematous air-cells, for they have been subjected to an organic change, and the present state of science affords no means by which that change can be obviated.

INTERLOBULAR EMPHYSEMA.

Vesicular emphysema is generally a chronic affection; interlobular emphysema is a sudden and acute disease, the result frequently of accident.

Morbid Anatomy.—Dense as the cellular tissue is which separates the pulmonary lobules, yet it occasionally becomes infiltrated with air. These slight partitions now increase in breadth, from a line to five or six lines, or even an inch, forming, particularly towards the edges of the lungs, transparent bands, plunging more or less deeply into their substance. These bands are broadest at the surface of the organ, and become gradually narrower as they descend towards the centre. They sometimes run parallel; at others, traverse each other, so as to isolate one or many lobules, according to the extent of their intersections. Small grains, as it were, of air run frequently in the cellular tissue surrounding the blood-vessels and bronchial tubes, often also extravasating beneath the pleura; and when the air penetrates to the root of the lungs, it passes from thence into the mediastinum, and so to the neck, and the cellular tissue of the rest of the body.

It might be supposed that this disease could only arise from the rupture of a number of cells, and that the air passed from thence into the surrounding cellular tissue. It may be so: but the broken cells have not yet been detected, nor are the vesicles contained in the isolated lobules even dilated.

Sometimes the mass of air is so broad in the interlobular cellular tissue, that it is difficult to suppose that it has not infiltrated into the whole of a lobule, and, by insinuation between the cells, compressed them, and occasioned them thereby to be reduced to their mere cellular rudiment: yet no positive evidence exists of this circumstance.

Causes of Interlobular Emphysema.—Any cause by which the air is retained a long time, and with violent effort, in the lungs, may induce this disease — as the resolute attempt to lift too great a weight, the efforts of accouchement, of defecation, &c. Can air be generated spontaneously in the interlobular cellular tissue? It is possible; for we see exhalations of gaseous fluids occur in cavities where no communications exist with the external air, as in the peritoneum, pericardium, and within the synovial membranes.

It would appear surprising, considering the extreme dyspnœa which occasionally takes place from vesicular emphysema, that the dilated cells do not rupture, and produce the interlobular species: this, however, has never yet been seen. It is true that sometimes a little air will escape under the pleura; and I have seen an instance of the cells rupturing through the pleuritic covering of the lung, inducing pneumo-thorax; but I believe no instance is known of vesicular emphysema producing interlobular. This, in all probability, is due to the increased firmness of the cells when permanently dilated, and to the extreme density of the interlobular cellular substance.

Local Signs of Interlobular Emphysema.—The dry crepitation with large bullæ is the pathognomonic sign of this disease. The “frottement ascendant et descendant” are also signs: the first occurs at the moment of inspiration; and at this time, too, the crepitation may be distinguished: the second accompanies expiration. It is sometimes heard once, and at one moment only; at others, two or three times successively; occasionally only when

the expiration is completed, and it then appears as if something descended into its usual situation. Usually the rubbing seems to be against the costal pleura; now and then against the diaphragm, or deeply against the mediastinum, or among the pulmonary lobules. A crackling at the affected part is often felt by the patient.

These sounds often communicate their vibrations to the hand. The crepitation may even be produced occasionally by pressing the finger against an intercostal space, corresponding to the part of the lung affected.

By percussion you may always elicit a loud sound, except these be any coexistent pulmonary engorgement at the emphysematous part.

If the emphysema attack the neck, then, by pressing the part, crepitation is produced, and the diagnosis becomes still more complete.

Functional Signs. — Sudden dyspnœa after the occurrence of any of the above-mentioned causes. Infants are most commonly attacked with this disease: thus, it may occur in them as a consequence of croup, or of severe catarrh. It may occur also in adults, although not so frequently, under the same circumstances.

Treatment. — Very little can be done in this disease, and fortunately it is an affection which has rarely any serious results. When the air infiltrates into the subcutaneous cellular tissue, a few punctures with the lancet will be ordinarily sufficient to allow of its escape. When the disease is confined to the lung, in most cases the air appears to be absorbed, and the interlobular cellular septa return gradually to their natural size.

LECTURE XIII.

DISEASES OF THE PARENCHYMATOUS STRUCTURE
OF THE LUNGS.

ŒDEMA PULMONUM.

Definition.—*Morbid Anatomy*.—Serum, its Character.—Texture of Lung.—Disease, recent or chronic.—Gravitation of Fluids in the Lung.—*Functional Signs*.—Dyspnœa.—Cough, Expectoration.—Combinations with Peripneumonia, or Mucous Catarrh.—General Œdema.—*Local Signs*.—Percussion.—Respiratory Murmur.—Crepitation.—Difference between Signs of Peripneumonic Engorgement and Œdema of the Lung.—*Causes*.—Generally Symptomatic.—Consequence of various Affections—especially of Diseases of the Heart.—Theory of Dropsy, from Obstruction of the Circulation.—*Treatment*.

Apoplexia Pulmonum, or Hæmoptysis.—*Morbid Anatomy*.—Firmness of Lung.—Extent of Disease.—Circumscription.—Surrounding Cells, Rosy Serum.—Uniformity of Colour, neighbouring Veins.—Granulations.—Lung torn by the Effusion.—Situation of Apoplexy.—Difference from Cadaveric Engorgement.—Resolution of the Disease.—*Functional and General Signs*.—Dyspnœa, Pains, Heat, Cough, Pulse, Absence of Fever.—Expectorations of Blood.—Colour—Quantity.—*Local Signs*.—Caution.—Percussion, Auscultation.—*Causes*.—Stimulants.—Violent Efforts.—Vicarious, Tubercles.—Temperature.—Obstructions at aortic or mitral Orifices.—*Treatment*.

WE now arrive at the consideration of Œdema Pulmonum.

By œdema pulmonum we mean an infiltration of serum into the pulmonary texture. This disease was first noticed by Albertini, and was afterwards pretty fully described by Barrère, physician to the

Military Hospital at Perpignan, in his "Observations Anatomiques," published in 1753.

This affection has often been confounded with hydro-thorax ; but by the latter term we mean an effusion of serum into the bag of the pleura, while this is an effusion of serum into the parenchymatous substance of the lung. Both may arise from the same cause; but this is the anatomical difference between them.

Morbid Anatomy. — In cutting into an œdematous lung, serum immediately flows from the section, the quantity varying very considerably, so that only a small portion may escape, or there may be a pint, or a quart, or even two quarts. This serum is usually colourless and transparent, but sometimes of a slight fawn colour ; and, according to the duration of the disease, it is more or less spumous : thus, if the disease be recent, the secretion is always mixed with a considerable quantity of air ; if chronic, it contains scarcely any. The weight of the lung is increased, and it does not subside upon opening the chest. The lung still crepitates in the hand upon compression, because there is still a certain quantity of atmospheric air present. It also pits upon pressure, like an anasarcaous limb. You will sometimes find that the blood-vessels which supply the organ are almost ex-sanguineous ; they are colourless, and nearly empty, and the lung itself is generally of a pale grey or yellowish tint.

The spongy texture of the viscus is never altered in this disease ; it may always be seen after a certain quantity of serum has flowed from the incisions ; but when the lung is entire, it is difficult to distinguish the air-cells, in consequence of the fluid which

fills them diminishing their transparency. The increased secretion is evidently contained in the interlobular cellular tissue, as well as in the air-vessels.

If the œdema be recent, it is always, as I have already stated, very spumous, which, I apprehend, arises from the fluid hardly having had time to discharge or dislodge the atmospheric air contained in the cells. If the œdema be of long duration, you will find that the serum flowing from the section of the lung is not spumous; and I presume that this arises from its having had sufficient time to displace the air. A short period before death it would appear that a certain quantity of serum is deposited in the lung, except in cases of great marasmus; so that you will frequently find, on making a post-mortem examination, œdema of the lung, combined with sanguineous cadaveric deposit, and these deposits are always found in a position relative to that in which the body has been placed. Thus, if it be placed on the back, then they occur at the posterior part of the lungs; if on the face, at the anterior part; and if on the side, laterally; showing evidently that the position of these fluids depends merely upon their gravitation.

Symptoms. — Functional Signs. — Dyspnœa is a symptom of this disease, and, as a general rule, its intensity is proportionate to the quantity of fluid contained in the air-cells. A slight cough often also occurs, followed by the expectoration of an aqueous fluid, more or less in quantity; sometimes there is no excretion whatever: but if the œdema be combined, as it often is, with pituitous catarrh, the expectoration increases considerably,

and changes also in its appearance. It then consists of a colourless pituitous fluid, very similar to the white of an egg, mixed with an equal quantity of water : it contains also bubbles of air. If inflammation of the lungs supervene, a few sputa may be seen of a slight fawn, green or rusty colour, but still transparent; and sometimes, from the coexistence of mucous catarrh, there will float upon the transparent pituitous secretion a certain quantity of yellow mucus, often assuming an irregular round form. General serous infiltration of the body frequently accompanies œdema pulmonum.

Local Signs. — Percussion rarely affords distinct signs of œdema pulmonum, inasmuch as the disease attacks usually both lungs, and, consequently, there is no difference in the sounds which may be elicited from the opposite sides of the chest. In most cases the sound thus produced is tolerably good, because there is yet a considerable quantity of air contained in the air-cells; but in chronic cases, when the serum has displaced the air in the vesicles, the sound is duller on percussion.

Respiratory Murmur, &c. — You may readily suppose, that the respiratory murmur becomes indistinct, as the air cannot freely enter into the lungs. You will recollect that whenever air and serous fluid coexist in the ærian cells, the “rhonchus crepitans” is produced. It is therefore very distinct in this disease : the bullæ convey to the ear a sensation of humidity, and are larger than in peripneumonic engorgement.

As the local signs of œdema pulmonum and peripneumonia in its first stage, are the same, it becomes difficult occasionally to determine which

disease it is; but inflammatory engorgement is almost always attended by fever, œdema rarely; engorgement is an acute disease; œdema most frequently a chronic one: the latter is commonly a symptomatic affection; the former an idiopathic. Yet it must be admitted that sometimes the diagnosis is attended with considerable difficulty, and requires great caution.

When the lung is also emphysematous, or when a severe dry catarrh supervenes, the diagnosis becomes still more difficult, as the respiration is too slight to produce crepitation. By desiring the patient to inspire deeply, or to cough, you will often, nevertheless, hear the rhonchus crepitans.

Causes. — Œdema pulmonum is very rarely indeed an idiopathic affection; it is almost always consequent upon some other disorder. First, it may arise where there is a general serous diathesis, in which the whole body is in a state of dropsy. Secondly, it may be consequent to certain fevers, more particularly of the eruptive class, and especially scarlatina. Thirdly, it frequently co-exists with measles and its catarrh. Fourthly, you recollect I mentioned that in the resolution of peripneumonia, in the first stage, the red colour of the serum becomes absorbed, and the fluid which remains is colourless; the portion of the lung affected is then in a state of œdema. Fifthly, it accompanies chronic catarrh, more particularly the chronic pituitous form, and especially pertussis. Sixthly, and lastly, it arises from diseases of the heart.

These are nearly all the circumstances in which we find œdema pulmonum. Of all these causes, diseases of the heart are the most frequent, and

the œdema then may be considered as a dangerous symptom. I shall here endeavour to explain the general theory of dropsy arising from obstruction to the current of the circulation.

It must be well known to you that the cellular tissue of the whole body, and the free surfaces of the serous membranes, are constantly bedewed with a secretion by means of which the organs move easily upon each other: this secretion is deposited by the arterial capillaries; and, as the function is constantly acting, the fluid would accumulate, were it not for the absorption which is as constantly performing also: so that in the healthy state there is a perfect equilibrium between the quantity of serum secreted, and the quantity absorbed. That the function of secretion of the serum is effected by the arterial capillaries, there can be no doubt. It is not doubted either, that the vessels, called absorbents, absorb; but it has been supposed that the venous capillaries perform the latter function also. To determine this point, Magendie made several experiments, and the one I am about to detail seemed confirmative of the latter opinion. The hinder limb of a tolerably large dog was amputated high up, and its separation rendered complete, with the exception of the femoral artery and vein, so that the limb hung suspended to the body by these vessels, the loose cellular substance around them being carefully dissected off. The upas, a most potent poison, was then inserted under the skin of the amputated leg, and in a very short time its baneful effects became apparent in the body of the animal. Now the poison could only have entered into the general system by the femoral vein; for it would be incon-

sistent to suppose it could have ascended in the artery, since it would have gone against the current of the circulation. It was now objected that, although the artery and vein might be clearly dissected of its surrounding loose cellular tissues, yet a sufficient number of absorbents might be left in the parietes of the vessels to admit the passage of the subtle poison. To answer this objection, Magendie, with great tact, after cutting off portions of the vein and artery, contrived so to introduce corresponding pieces of quills in their places that the blood flowed through them instead of the living vessels; yet the upas produced its effects in the same manner. This experiment seemed to be conclusive of the fact of the absorbing power of the veins.

Lippi, of Florence, however, showed that the lymphatics not only had a termination in the thoracic duct, but, as they followed the course of the veins, and were in close contact with them, they opened by minute orifices of communication into them, so that mercury injected into the lymphatics might be made to pass into these vessels; and I present you a preparation, in which mercury injected into the lacteal absorbents, passed into the mesenteric veins; so that it is quite possible, in Magendie's experiment, that the poison first flowed into the absorbents, and from thence, by the minute orifices of communication, into the veins of the amputated portion of the limb, and that, consequently, the blood circulating through the isolated vein or the intermediate quill, was mixed with the poison before it had arrived at them. It is true that this demonstration does not disprove the ab-

sorbing functions of the veins, but it invalidates the experiments of Magendie, by showing that they may be explained in another manner.

Under the supposition that either one or the other set of vessels absorb, or that they both do (for there is no inconsistency in the latter hypothesis), the following general proposition is not the less true: That any cause obstructing for some time the free current of the blood in the veins, will occasion an accumulation of serum in the parts from whence that blood is immediately derived. Thus it is a fact well known, that when the impregnated uterus enlarges to a certain extent, it often presses against the iliac vein or veins, and produces an enormous swelling of the corresponding limb or limbs: in this case the veins of the extremity affected become too full, and they are often seen blue, meandering and varicose in consequence: no serum can now be absorbed, for there is no room for additional fluid in the veins, admitting that these vessels alone absorb. Admit, again, that Lippi's demonstration be fully proved, as I think it is, that the veins receive the serum by the minute orifices I have spoken of, still there is no room in the already filled vessels to receive the lymph, and the lymphatics must become gorged also, and their function suspended. Finally, supposing that the lymphatic vessels alone absorb, we know that the parietes of the veins are so interlaced and interwoven by the absorbents, particularly the iliacs, that it is impossible to press upon the one system without pressing the other. I repeat again, that pressure upon the veins occasions dropsical effusion in the parts supplying them with

blood, or, in other words, destroys the equilibrium between secretion and absorption in those parts.

Let us examine now how certain diseases of the heart produce dropsical effusions. You will soon perceive here that it is from obstruction to the venous circulation, although a small part of the arterial is involved in it also.

The aortic orifice is often obstructed, either by ossific or cartilaginous deposits in its valves, or by congenital malformation. Let us suppose the caliber of the aorta at its origin to be diminished one half, as the blood arrives at that orifice from the lungs in its ordinary quantity, and only half of the column can pass through it, it follows that that fluid must accumulate in the left ventricle, the parietes of which often increase in muscular thickness, and acts most energetically to overcome the resistance before it, or it dilates, or both these circumstances occur at once, the left ventricle being then said to be in a state of hypertrophy and dilatation. As the left auricle is a continuation of the ventricular cavity, it also becomes gorged with blood, and its parietes become thickened or dilated; the four pulmonary veins are filled also to their capillary extremities, and so far the arterial system is alone affected; but now the pulmonary arterial capillary vessels meet with their obstruction, in consequence of the fulness of the pulmonary veins; the pulmonary artery becomes gorged with blood; and as the right ventricle and auricle are continuations of the canal, they soon arrive at the same state of plethora. Thus in the whole round of the smaller or pulmonary circulation, a series of obstructions exist, the one consequent upon the other, and the result

is, that the veins cannot take up the serum in the air-cells, or in the pleuritic cavity, nor can the lymphatics, from the general plenitude of both sets of vessels; and œdema pulmonum, or hydro-thorax, or both, may be the consequence.

We will now trace the manner in which dropsy forms in the general cellular tissue. If the right side of the heart become loaded with blood, from the obstruction at the aortic orifice, the blood from the venæ cavæ meets with its obstruction in the habitually overfilled cavities of the right auricle and ventricle. The superior cava swells; its branches, to their capillary extremities, become congested; the sinuses of the brain become turgid; the face and lips assume a leaden hue, and swell, and this swelling depends upon an accumulation of serum in the cellular tissue of the parts, arising from the obstructed state of the superior cava.

If the fulness of the superior cava produce these effects, where the propulsion onwards of the blood not only arises from the *vis-à-tergo*, but is also accelerated by the force of gravitation, a similar series of results must more certainly occur in the system of the lower cava, where that fluid has to mount by the mere vital force: abdominal dropsy, and general cellular serous effusion of the inferior extremities, then take place.

Treatment. — The treatment of œdema must depend upon the disease with which it is complicated. Thus, if it arise from a general serous diathesis, in all probability there is a disease of the heart, and this is the organ to which your attention must then be directed. If an organic lesion exist, palliative measures only can be adopted, the disease

itself being generally incurable. In such circumstances, diuretics, mercury, and the occasional administration of elaterium, are useful. When it occurs at the termination of fever, except dyspnœa take place, there is but little danger, and it will generally give way to tonics, purgatives, and particularly to mercury. If it accompany measles and catarrh, the treatment will then merge into that adapted for these complaints. If it be the result of peripneumonia, it requires little or no treatment; if it be conjoined with pertussis, the treatment must be the same as described for that affection.

APOPLEXIA PULMONUM; OR, HÆMOPTYSIS.

We now pass on to the affection called by Laënnec *Apoplexia Pulmonum*, or that state of the lung which is the most frequent cause of hæmoptysis, or spitting of blood.

It is a singular fact that, although this disease has been known from remote antiquity, although it is somewhat a common affection, yet its morbid anatomy has not been understood until lately: it has generally been supposed that hæmoptysis arises from the bursting of large blood-vessels; but that is an erroneous opinion: it seems rather to depend upon an exudation of blood from the capillary vessels of the lungs.

Morbid Anatomy.—Laënnec has given us the following description of the morbid anatomy of this disease:—

The lung becomes as firm as in ordinary hepatization—I should say even much firmer. The disease is always partial, and rarely affects a large portion of the organ: its extent varies from a pin's

point to three or four cubic inches: the firmness is most frequently exactly circumscribed, and the engorgement is as great at the circumference as at the centre: there is no gradual diminution of density, as in hepatisation, but a distinct line of demarcation separates the healthy from the diseased parts: the surrounding cells are usually healthy and crepitant; they are often even paler than natural: sometimes they present a rosy, or even red colour, depending upon the infiltration of a certain quantity of coloured serum.

The colour of the diseased part is of a deep black red, similar to that of a clot of venous blood, and differing from that of hepatization inasmuch as the former is homogeneous, and permits us no longer to distinguish the bronchial tubes, the blood-vessels, or interlobular cellular substance; for all are involved in a deep red colour. The neighbouring veins are often filled with concrete and half-dried blood. The lung, if torn, presents a granular appearance like hepatization, but the granules are larger.

The pulmonary tissue is less humid than in hepatization: you may scrape from the surface of a section a small quantity of black and half coagulated blood; sometimes the centre of the apoplexy is softened, and filled with a clot of pure blood. It occasionally, though rarely, happens, that the substance of the lung may be torn by the sanguineous effusion; and Corvisart mentions an instance in which the pleura was lacerated, and the blood escaped into and filled its cavity.

Several apoplectic engorgements may occur in the same lung: often both lungs are simultaneously

affected; and although they may be found in all parts of the organ, yet they are most frequently situated in the centre of the inferior lobes.

It is very easy to distinguish this disease from the ordinary cadaveric engorgement. In the latter case a red and spumous serum flows from the section, which fluid gravitates towards the lower part of the lung. The œdematous effusion is not circumscribed; there remains always some degree of crepitation; no granules can be seen, but the areolar texture of the organ is to a certain degree apparent, and the lung may be rendered flaccid by repeated washings in water. A pulmonary apoplexy is always exactly circumscribed, very firm, of a blackish-red colour, granulated, and scarcely humid, becomes but little paler by frequent ablutions, and then does not lose its firmness.

Resolution may even take place in this morbid condition of the lung, for many persons recover. In the few cases which Laënnec had seen, it appeared that the engorgement passed successively from a black to a pale red colour, and that it gradually lost its granulated texture and density.

Signs — Functional and General.—The patient complains of dyspnœa, of oppression in the chest, and frequently of pains and heat along the course of the sternum. There is a frequent, short, and troublesome cough, depending partly upon the blood irritating the larynx as a foreign body, partly from the presence of that fluid in the trachea and bronchial tubes. The pulse is full, often hard, and presents a peculiar vibratory feel, and quick movement, as if quicksilver were circulating in the arteries, constituting what is called the “hæmor-

rhagic pulse," and is often accompanied by the "bruit de soufflet" of the heart and arteries. There is rarely fever, and the skin is of its natural temperature, or nearly so.

But the great and distinguishing symptom of apoplexia pulmonum is the expectoration of blood. It is usually copious, and returns frequently for some hours, or even days, accompanied by a distressing cough, oppression in the chest, and great anxiety: the face is at first intensely red, but afterwards becomes pale; the extremities are cold. When the spitting of blood is abundant, the cough is often slight, and it would appear by the movements of the diaphragm to be ejected by vomiting; a circumstance which, no doubt, frequently takes place from some of the blood having been previously swallowed: this is further proved by the alvine evacuations being almost always of a very dark black red colour, as we find during an attack of hæmatemesis.

The colour of the blood expectorated is bright red, or black; it is spumous, and often coagulated; the quantity varies exceedingly; it is often enormous: Laënnec speaks of an instance in which ten pounds were expectorated in forty-eight hours. I have seen two or three cases of similar quantities. These quantities are not, however, certain proofs of the magnitude of the apoplectic engorgement, as the bronchial mucous surfaces sometimes, though very rarely, give origin to great hæmoptyses, and large apoplexies do not always give rise to great hæmorrhages; nay, it sometimes happens that an engorgement of one or two inches may exist without producing any expectoration of blood.

Local Signs. — From having seen in two or three instances the expectoration of blood increased by an injudicious mode of examining the chest, particularly by obliging the patient to inspire deeply for the purpose of hearing the sounds more distinctly, I rarely now make any local observations, except I can do so without occasioning the patient the slightest fatigue or change of position. A local examination is also less necessary, because the general and functional signs are upon the whole sufficiently evident to indicate, if not the seat, at least the nature of the affection, and the treatment required. But these signs are as follow : —

Percussion indicates but little, inasmuch as the apoplexy is usually but of slight extent, and is so placed towards the centre and base of the lung, that no difference of sound can be appreciated : if, however, the engorgement be considerable, and approach the surface, then percussion would elicit a dull sound from the part of the chest corresponding to the part of the lung affected.

Auscultation affords two signs : the first is absence of the respiratory murmur, proportioned to the extent of the apoplexy ; the second is a “rhonchus crepitans,” surrounding the part where the murmur is inaudible, indicating the presence of a slight quantity of rosy serum around the engorgement : this crepitation is heard at the beginning of the disease, and afterwards ceases. When these signs occur, it is evident that the hæmorrhagy has its source in the substance of the lung, and not in the bronchial tubes ; but as, in bronchial hæmorrhagy, a mucous rattle, formed of bullæ with apparently thin parietes, and seeming to burst from

distention, is audible, particularly at the parts of the chest corresponding to the roots of the lungs, this species of rhonchus is doubtless produced by the presence of blood in the large bronchial tubes.

Causes. — The causes of apoplexia pulmonum are the same as those of bronchial hæmorrhagy; as general plethora, the abuse of alcoholic fluids, singing, playing on wind instruments, violent exercise, blows upon the chest; in fact, any cause tending to accelerate the circulation.

A pulmonary hæmorrhagy may be vicarious of some other habitual discharge of blood; thus the suppression of the hæmorrhoidal flux has been said to produce hæmoptoe. The cessation of the catamenia at the critical period of female life is certainly an occasional cause of this disease, and a surprising quantity of blood is sometimes lost from the lungs at this period, and yet the patients usually recover. I have also seen hæmoptoe coexist with amenorrhœa.

Laënnec observes that spitting of blood often occurs during the primary eruption of tubercles: it appears to me that that sign is most common at the time of their softening. The quantity is usually then inconsiderable; generally merely a few spots or streaks mixed with the sputa. It might be supposed that the destruction of portions of the lung consequent upon the softening of tubercles, and the formation of excavations, would produce considerable hæmorrhagy: I have seen such cases, but they are very rare; for, as we shall hereafter find, the blood-vessels, as they approach tubercular cavities, become obliterated.

Temperature has evidently an effect in the production of this affection; thus the sudden or long-

continued impression of cold acts as a cause. Laënnec conceived it possible, from seeing such immense quantities of blood occasionally ejected, that there must occur a sudden dilatation of that fluid. It is certain, when the atmospheric pressure upon the body is diminished, as in ascending high elevations, dilatation takes place, as the surface of the body then becomes turgid, and considerably reddened by the fulness of the capillaries, and effusions of blood from the nose, mouth, and bronchial surfaces, often happen. You will observe, also, that when the heat is great, and of long continuance, hæmoptysis is most common, as in the months of July and August.

Obstructions at the aortic or mitral orifices, by inducing sanguineous engorgement in the pulmonary circulation, may produce apoplexia pulmonum. These are, indeed, among the most common causes; but we shall advert to them again when we arrive at the description of disease of the heart.

A sudden and fatal hæmoptoe is sometimes caused by the rupture of an aneurism into the trachea or its divisions.

Treatment. — As, in the ordinary forms of hæmoptysis, the pulse is strong, full, and jerking, so it becomes necessary to diminish the force of the circulation by abstraction of blood. Venesection is the best means of effecting this, and the quantity to be drawn should be proportionate to the force of the individual, and to the fulness, tension, and jerking of the artery. I never consider the patient safe until this violent action of the pulse is subdued, or at least very much diminished. A large bleeding effected at once is better than a number of small

bleedings. Cupping, or leeches, in some cases, may be employed, when the patient's powers do not admit of general depletion.

The next point to be attended to, is to insist upon the most positive repose, both of limbs and voice: do not allow your patient to speak, put your questions in such a manner that he may answer you by gestures alone, recollecting that to speak it is necessary to throw the lungs into increased movement, and they should be as quiescent as possible.

You should allow a free current of cold air to pass over your patient; let him be but very lightly covered; wash the chest and neck frequently with cold water, or vinegar and water, or, what is better still, apply, from time to time, a large bladder containing a quantity of pounded ice to the surface. Let the thirst be satisfied with cold fluids, avoiding, of course, all stimulants.

After having bled your patient, and placed him under the circumstances I have detailed, then you will find the administration of a drastic purgative to be useful: it was the practice of Sydenham, and it tends considerably to diminish the force of the circulation.

I confess I have not much confidence in other remedies in this disease; you will find that amongst them the digitalis is the best; alum, the acids, and the superacetate of lead, are commonly given, and you may use them according to the formula you will find in medical works. Dr. Rush, of Philadelphia, recommended large doses of common salt.

When the force of the pulse is diminished, and the violence of the disease is abated, counter-irri-

tants applied to the chest, as dry cupping, stimulant embrocations, or blisters, are most useful means of relieving any remaining oppression or dyspnœa.

You will occasionally find, in persons of debilitated constitutions, the hæmorrhagy to be of a passive kind, that is, the quantity of blood expectorated is, upon the average, small, the pulse weak, although frequent, with no jerking or fulness. In such cases, general bleeding cannot be borne; you must depend upon repose, upon the application of external stimulants, and upon the use of the acids and astringents I have already mentioned.

A teacher, gentlemen, is obliged to select for his subjects marked cases, so as to give good exemplary ideas. In describing this disease, I have taken two extreme instances; the one accompanied by increased tone of the system, the other by diminished power; but there are also all shades of intermediate differences, requiring modifications of treatment, which neither my time nor your patience will permit me to detail; but the knowledge of which is to be acquired only by a sedulous attention to practical, or, as it is called, clinical medicine.

LECTURE XIV.

DISEASES OF THE PARENCHYMATOUS STRUCTURE
OF THE LUNGS.

ASTHMA.

Definition. — Vulgar Acceptation, Table. — Causes of continued Difficulty of Breathing. — Chronic, Mucous, Pituitous and Dry Catarrhs. — Causes of Asthma. — Organic. — Chronic Mucous, Pituitous, and Dry Catarrhs. — Paroxysm of Humid Asthma. — Nervous Asthma, from increased Necessity for Respiration, or with peurile Respiration. — Respiratory Murmur in Infancy, adult and old Age, in different Persons, in Sleep, in the different Parts of the same Lung. — Paroxysm of Asthma from increased Necessity for Respiration. — *Spasmodic Asthma*. — Definition of Spasm — Tonic — Clonic. — Muscular Fibres of Bronchial Tubes. — Proofs of Spasm in Whooping-cough, sobbing, gaping, anhelation from running, during Spasmodic Asthma. — Paroxysm of Spasmodic Asthma. — Relaxation of Bronchial Muscular Fibres. — Active or passive Movements of the Lungs. — Proof of active Expansion in voluntary Movements of Chest, not increasing Intensity of Respiratory Murmur, in Puerile Respiration. — *Asthma from Combination of Organic and Nervous Causes*. — Table. — *Remote Causes* of Asthma. — *Treatment* of the various Species.

WE now, proceed, gentlemen, to the consideration of one of the most complicated affections of the pulmonary system — I mean Asthma.

Asthma is a disorder which, until lately, has been but little understood; firstly, because the organic changes of the different tissues composing the cardiac, and especially the pulmonary structures, have been but little known; secondly, because the signs derived from auscultation have been but recently

discovered, and are not yet sufficiently appreciated; and lastly, because we have long been in the habit of considering signs as diseases themselves, and not as their representatives, — a gross error indeed, since the same sign may be common to a variety of lesions, each of a very different nature, and each requiring a very different mode of treatment. To give you proofs of this, the older nosologists created a class of diseases under the name of *dolores*, or *pains*. Now, pain is not in itself a disease; it is a symptom which becomes a sign of a certain condition of parts, which condition is the disease. *Tussis*, or *cough*, has been established as a genus, but it is a sign only of a variety of lesions of the pulmonary structure, which lesions constitute the diseases, and not the signs. Cullen formed a genus under the appellation of *dyspnœa*, or difficulty of breathing; but this again is but the denomination of a sign — a sign, too, representing every change of the organs contained in the chest, when arrived at a certain intensity. The same objection may be applied to the term *asthma*, or difficulty of breathing, occurring at intervals without fever, for this state is only a sign of certain abnormal conditions of the pulmonary or cardiac organs, which conditions ought to be considered as the diseases, which we shall soon see are very various, and require different modes of treatment, although their effect may be the same in producing the common sign.

Definition of Asthma. — Asthma is defined to be a difficulty of breathing, occurring at intervals, without fever. I object not to the term, or the definition, so long as we recollect their true value;

namely, that asthma is a mere symptom, or sign, of various changes producing the common effect of intermittent dyspnœa.

According, however, to the vulgar acceptation, the word asthma is applied to every continued difficulty of breathing assuming a chronic form. To understand this subject, it will therefore be necessary to examine, firstly, the various causes which induce continued and chronic difficulty of breathing; and, secondly, those which produce an intermittent difficulty. In the hopes of elucidating this very complicated subject, I offer the following tabular view, and beg you will constantly refer to it as I proceed:—

Chronic Difficulty of Breathing.

Species First, *Continued*—Dyspnœa of Cullen.

Causes, are Catarrhs { Mucous.
Pituitous, with Œdema Pulmonum.
Dry, with emphysema and Œdema Pulmonum.

Species Second, *Intermittent*, or occurring at intervals, without fever—
Asthma of Cullen, &c.

Causes { 1. Organic — { Mucous.
Catarrhs { Pituitous, with Œdema Pulmonum.
Dry, with Emphysema and Œdema Pulmonum.
2. Nervous . . { Asthma, with Puerile Respiration.
Asthma, spasmodic.
3. Complication of Organic and Nervous Causes.

You perceive, gentlemen, that I head this table with the phrase “chronic difficulty of breathing;” an expression which requires no definition. I divide this into two classes; 1st, that which is *continued*, having no intermission, and existing with greater or less intensity, and which answers in general to the *dyspnœa* of Cullen; 2d, that which is *intermittent*, or occurring at intervals, which corresponds to the *asthma* of the same author. I then

proceed to examine the causes, or real diseases, which effect these aberrations in the functions of respiration.

Causes of continued Difficulty of Breathing. — These causes may very easily be understood: indeed, I have already described them, and shall therefore be very brief in their detail, since you can refer to what I have already said relating to them: they consist of the various forms of chronic catarrh, of emphysema, and œdema pulmonum.

Chronic Mucous Catarrh consists in chronic inflammation of the mucous membrane lining the ærian passages, and having for its result a yellow secretion. This disease is usually trifling at first, but, by the reiteration of its external causes, may extend from point to point of the mucous lining, until the whole becomes at last affected. In proportion to the extent of the disease, the patient is troubled with dyspnœa: at first it may be slight, requiring, perhaps, unusual movement to excite it, but gradually the difficulty becomes more and more permanent, till it terminates, as old age advances, in suffocative catarrh.

Chronic Pituitous Catarrh. — The same observations precisely apply to this disease as the preceding, the principal difference consisting only in the nature of the secretion, which in this case is clear and colourless. The two forms of catarrh are not infrequently combined. *Edema* of the lungs often coexists with chronic mucous and pituitous catarrhs, especially the latter; and in proportion to the quantity of serum infiltrating the organ, so must the difficulty of breathing be increased,

since that fluid occupies the space which should be filled by air.

Chronic Dry Catarrh. — You will recollect that this affection is so named, not because there is no secretion, but because there is infinitely less than in the two preceding forms of catarrh. The mucous membrane is here habitually reddened and swollen, and the disease has a very strong tendency to become chronic, and extend gradually through the whole of the bronchial tubes. Constant dyspnœa is always the result, its degree being proportionate to the number of tubes affected. *Vesicular emphysema* is a frequent consequence of this form of catarrh, and, when present, adds to the difficulty of respiration, by preventing the free entrance and exit of the atmospheric air to and from the lungs. *Œdema* is also occasionally superadded.

Chronic hepatization and gangrene of the lungs, the presence of tubercles, or other adventitious deposits, chronic effusions into the pleuritic cavities, might be enumerated as causes of continued chronic dyspnœa; but these diseases are of slight duration compared to catarrhs, since the latter may last for many years, and the former run their course much more rapidly.

Such are the causes of continued chronic difficulty of breathing; we will now investigate those which produce the intermittent form.

Asthma, or Intermittent Dyspnœa.

The causes of asthma, or intermittent dyspnœa, may be referred to three heads: the first are

called *organic*, the second *nervous*, the third consists of a *complication* of both.

1. *Organic Causes*. — These causes are precisely the same as of continued chronic dyspnoea, which are, chronic mucous, pituitous, and dry catarrhs, with their combinations of emphysema and œdema pulmonum.

Chronic Mucous Catarrh. — It not unfrequently happens that, when this disease has lasted for a considerable time, especially in aged persons, a fresh catarrh is grafted upon the bronchial mucous membrane, and a great increase of mucus suddenly takes place, so great and so sudden, that some of the larger bronchial tubes become filled by it, so as totally to prevent the passage of air down them: the result is, that the habitual dyspnoea becomes suddenly increased, the patient breathes with great difficulty, and he is only relieved by a large expectoration of mucus, after which he returns to his usual state. This fit is often repeated. In consequence of the copious expectoration which terminates the paroxysm, this form has been called *humid asthma*.

Chronic Pituitous Catarrh. — The same observations exactly apply to this catarrh, the difference being only in the colour of the secretion, and in its being accompanied more frequently by œdema of the lung, rendering the paroxysm more severe. It is also a case of *humid asthma*.

Chronic Dry Catarrh is also an organic cause of asthma: it occurs from the sudden engrafting of a fresh catarrh. I have so fully already explained the symptoms of this disease, that I need only refer you to it. In consequence of the slight quantity

of expectoration terminating the paroxysm, it may be considered as an instance of *dry asthma*.

Paroxysm of Humid Asthma.—Although I have previously described the symptoms of catarrh, yet it may be necessary to throw into one point of view those by which increased temporary difficulty of breathing is accompanied.

Humid Asthma more commonly attacks men than women, more frequently old persons than young. It is generally preceded by nausea and unpleasant eructations; by sensations of flatulent distention of the stomach and bowels; by loss of appetite, and the general symptoms of dyspepsia; and often by drowsiness, — this state continuing two, three, or four days before the attack. At last, towards the evening, the patient complains of tightness, or a sense of stricture or straitness about the chest, particularly in the direction of the sternum; the dyspnœa gradually increases, and becomes very great; there is often considerable wheezing, and desire for fresh air. The cough is usually slight at first, and the expectoration is but trifling. The paroxysm thus formed now continues for some hours, generally until the approach of morning. Finally, the cough increases, and the patient becomes relieved by a copious expectoration of mucous or pituitous matter, by which the bronchial tubes become emptied: the paroxysm then ceases, and the sufferer returns to his usual state of habitual but slighter dyspnœa.

If the chest be examined during the fit, it will be found that a good sound may be elicited by percussion. The respiratory murmur is often lost in many points of the lungs, in consequence of the

obturation of the bronchial tubes ; in others it becomes puerile here and there. All the different species of wheezings may be heard in the same patient. Thus, in the beginning of the paroxysm, you will find the sibilating or sonorous sounds ; but towards its termination they become intermixed with the mucous rattle. When œdema of the lungs occurs, it is indicated by the rhonchus crepitans.

A paroxysm of humid asthma is frequently renewed in three, four, or five days, especially in damp or cold weather : occasionally it does not return for many months. In aged persons it sometimes happens that the power of expectorating is lost, or so extremely diminished, that the secretion accumulates. The fit in that case will continue for many days ; the dyspnœa increases ; the face becomes bloated and livid ; coma, from which the patient is with difficulty roused, supervenes ; the mucus still continues to increase ; the catarrh becomes suffocative ; and the scene closes in death.

2. *Nervous Causes.* — It sometimes occurs, that individuals are attacked with severe and sudden dyspnœa, without any apparent organic cause whatever : we then attribute it to some peculiar condition of the nervous system. Pure nervous asthmas are somewhat rare, for, as we shall hereafter see, they are most commonly combined with organic lesions ; but I have seen them in their purest forms, in which it was impossible to discover any of the species of catarrh, emphysema, or œdema.

Laënnec has divided asthma from nervous causes into two varieties. 1. Asthma from increased

necessity for respiration, or with puerile respiration.

2. Spasmodic asthma.

1. *Asthma from increased Necessity for Respiration, or with Puerile Respiration.*—The expression “increased necessity for respiration,” requires explanation, or it will be impossible to understand what is meant by this form of asthma.

The elucidation of the following proposition will demonstrate what is meant by the term.

The loudness of the respiratory murmur is always proportionate to the necessity for respiration, so that it becomes a measure of that necessity.

Thus, if you examine the respiratory murmur of an infant (if the breathing be going on calmly and without interruption), you will find it very distinct and loud; every air-cell seems to expand with vigour and force. The necessity for respiration in a child is great, because all its organic functions are in high activity; it requires to be fed frequently; its digestion is extremely active; the chyle is carried with a more rapid current into the blood; the heart is more active and frequent in its movements; the blood requires a greater proportionate quantity of air, and the function of respiration is carried on with increased force; or, in other words, the necessity for respiration is great, and the loudness of the sound of the respiratory murmur is at once the proof and the measure of it.

Examine the chest of a youth: the sound of the respiratory murmur, although perfectly audible, is less so than in the infant; because, at the adolescent period, the extreme energy of the functions of nutrition and sanguification are diminished, and the blood requires proportionately less air. The

necessity for respiration is now diminished, as well as the loudness of the sound of the murmur.

Apply the stethoscope to the chest of an old person, and, in many, the respiratory murmur is scarcely to be heard at all: it is always less loud, because all the vital functions become less energetic; the digestion is slower, the circulation less frequent, and less air is needed: the necessity for the respiratory act is consequently diminished, and the sound of the murmur is almost lost.

I may add, here, that we see in the organisation of the bones and cartilages of the chest, at different ages, a beautiful adaptation to the necessity for respiration. In infancy, the chest admits of free dilatation in all its diameters, in consequence of its flexibility; as age advances, its mobility becomes less; the octogenarian scarcely moves his ribs at all, but breathes by the diaphragm alone.

The necessity for respiration not only varies at different periods of life, but it varies also in different individuals. Thus you will find that the respiration of persons of certain temperaments, as of hypochondriacs or hysterical women, produces a much louder sound than that of individuals of an opposite or phlegmatic disposition. The function of respiration is commonly more active in women than in men.

It is a curious fact, that the necessity for respiration is commonly less during sleep than in the waking state. It is for this reason you will find that patients who are labouring under considerable dyspnœa during the day, when awake, will at night, whilst sleeping, respire with comparative ease and freedom.

But not only may the necessity vary according to age, sex, temperament, and the states of sleeping and waking, but it may vary in the different parts of the same lung. If the lung of one side be hepatised, or compressed by fluid in the pleuritic cavity, the function of the opposite lung will become doubly energetic to make up for the deficiency, and the murmur of the healthy organ will become as loud as that of a child; from whence it is called *puerile respiration*. Nay, if a portion of the lung of one side alone be affected, the healthy part of it will act with increased force, and give the puerile sound.

If I have been sufficiently clear in explaining the doctrine of *increased necessity for respiration*, you will have no difficulty in understanding what is meant by asthma from this cause. Thus, you may be called to a patient labouring under great and sudden dyspnœa, who is endeavouring to expand his chest in every direction, grasping surrounding objects for the purpose of obtaining fixed points from whence he may effect that expansion; whose face is turgid, livid, or slate-coloured, from venous congestion: you examine the local signs, expecting to find an absence of the respiratory murmur, or at least the presence of catarrhal signs; but you find, to your surprise, the lungs acting with great force, and the respiratory murmur extremely loud at every point. The paroxysm may last for many hours, but it is not terminated by expectoration, nor does the patient relapse into continued dyspnœa afterwards, as in asthma from organic causes. This is another form of *dry asthma*.

This disease in the pure state is rare. In ten

years, I have seen but three cases ; all of them were in females, and none were fatal. The paroxysms were frequently repeated.

2. *Spasmodic Asthma*. — In the infancy of morbid anatomy, every sudden dyspnœa unconnected with inflammation was attributed to spasm : since that science has made such great progress, the existence of spasm has been denied, and all sudden difficulties of respiration have been supposed to depend upon organic lesions.

I will not deny — nay, I am fully disposed to believe — that organic changes must be the immediate causes of all symptoms ; for it is difficult to conceive how a function can be altered, without supposing some material change to be effected in the organ performing that function, or in the vessels and nerves supplying it. It is very true that the scalpel of the anatomist, or the test of the chemist, cannot always shew the nature of the lesion or change ; but may not this arise from the imperfection of our means ; or may not these alterations be so fugacious or microscopic as to elude our research ? I would freely admit, therefore, in the cases of spasmodic asthma, the probability of the nervous system being temporarily affected organically, although there be no other proof than the aberration of function ; I assert only, that spasmodic asthma may occur without the evidence of any lesion of the lungs.

But spasms of the bronchial tubes and air-vessels has been affirmed by some, and denied by others, to exist. Let us examine the question.

Spasm is defined to be an involuntary contraction of a contractile organ. It has been divided into

two species : — 1. *Tonic*, or continued contraction, as in tetanus ; 2. *Clonic*, or a sudden contraction, followed by as sudden a relaxation — as in chorea, convulsio, hysteria, &c.

If, as is supposed, spasm can only affect muscular fibre, it will be necessary to search, whether such fibre can be found in the bronchial tubes. Reissessen has demonstrated their existence in bronchial vessels of a very small caliber, and Laënnec says he has traced them to tubes of one line in diameter. The former author infers their presence in the air-vessels themselves, but such is their minuteness, that we can have no ocular proofs of this, although the facts I am about to detail would lead us to coincide in that opinion.

Admitting, then, gentlemen, the existence of muscular fibres in the bronchial tubes, we must admit also that they are subject to the same pathological laws as the muscular fibres of the rest of the body ; that is, they may be affected by spasm and relaxation.

We shall first examine, whether there are any stethoscopic proofs of spasm of the bronchial tubes, and then describe spasmodic asthma ; and, finally, proceed to offer you evidences of muscular relaxation, by which asthma from increased necessity of respiration will be still further elucidated.

Stethoscopic Proofs of Spasm of the Bronchial Tubes. — In describing whooping-cough, I mentioned that, after the violent and repeated respirations were made, a deep and convulsive inspiration followed (producing the whooping sound). It might be supposed that the air entered the lungs during this apparent inspiration, but it does not ; for, during

this period, the respiratory murmur is inaudible; the dilatation of the cells, therefore, does not take place. Why is this? It can only arise from occlusion of the tubes or air-cells; and what can close them but a spasmodic constriction? It cannot be from any organic cause, for that would be permanent; but the respiratory murmur immediately returns upon the cessation of the paroxysm.

Sobbing consists in a rapid succession of short inspirations and expirations; *gaping*, in a very deep inspiration, followed by a shorter expiration. In both these cases you will often hear no respiratory murmur. What prevents the air entering into the cells in these cases? There certainly must be some obstruction, which is explicable by a temporary spasmodic state alone.

If you examine persons in a state of anhelation from running, you will usually find that their respiratory murmur becomes gradually evident as the dyspnœa diminishes, but it is not heard at all during its intensity. Here, then, is another instance in which the temporary occlusion of the cells is only attributable to spasmodic constriction.

No respiratory murmur can be heard, or, at least, but very little, during an attack of spasmodic asthma. Desire a patient, during a paroxysm, to read as loud and as long as he can in one breath; you will then find the puerile respiration form in one or more points of the lungs. Why was the murmur not heard at first? Because the obstructing spasm prevented the admission of the air. Why was the puerile respiration heard during the reading? Because you threw the patient into still greater dyspnœa, and thereby occasioned so great a necessity

for respiration as to overcome the spasm : it was a greater force overpowering a less.

If, then, these facts be true (and I have often enough proved them to satisfy myself that they are so), we shall not be accused of an insufficient enumeration of causes in attributing them to spasm alone; for they occur during the absence of organic lesions, and the disappearance of the respiratory murmur is so sudden, that I think it impossible they can arise from any other source.

Supposing, then, gentlemen, that spasm of the muscular fibres of the bronchial tubes may occur, you may readily conceive it to be of the tonic form, or persistent for some hours, and that the patient, during that time, would be thrown into great distress from difficulty of breathing. This state constitutes what is called *spasmodic asthma*.

Symptoms of Spasmodic Asthma.—The functional and general symptoms of this affection are precisely the same as those of asthma from increased necessity for respiration : the dyspnœa is equally sudden, equally great; the face becomes swollen and livid, and the brain the seat of sanguineous congestions; there is the same desire for fresh air. The fit will sometimes last for hours; sometimes the patient, as I have seen, expires in a moment, in a state of asphyxia. The pathognomonic sign arises from the absence of the respiratory murmur almost all over the chest. The fits do not terminate by any expectoration, nor does the dyspnœa return between them. This is another form of *dry asthma*.

Having thus, gentlemen, proved to you that spasm may affect the ærian passages, I now proceed to examine the subject of *relaxation* of the

muscular fibres of these tubes. To prove a relaxation of muscular fibre after its contraction were unnecessary, it is self-evident; but a question has arisen, particularly relative to the hollow muscles, whether it is always a passive movement, or whether the opposite state to contraction is not effected by an active force — a strong expansion, rather than a mere relaxation.

Upon opening the chest of a large living animal, and embracing its heart with your hands, you will instantly, I think, satisfy yourselves that the dilatation of the ventricles is effected by a most active expansion of their parietes; for you will find it extremely difficult by pressure to prevent it. It may be said that this expansive force arises from the impulse given by the blood entering these cavities; but separate the heart entirely from the body, so that no blood can pass into it, still the expansion will continue for a time, so as to afford resistance to pressure. It appears to me an abuse of terms to say that that is a passive relaxation which a compressing force cannot prevent.

If one hollow muscle can dilate with an active force, it is fair to infer that another may do so. Do the muscular fibres of the bronchial tubes and of the air-cells (if they exist in the latter) dilate by an active or passive movement? Are the movements of the lungs mere consequences of the movements of the chest; or do they contract and dilate by a power inherent in themselves?

There can be no doubt that the movements of the chest and diaphragm strongly influence the capacity of the lungs; but I think there are quite sufficient proofs that the pulmonary organs are not

passive in these changes, and are not to be considered as inert.

To obtain a direct proof of the active resiliency of the lungs, I have four or five times examined their movements by opening the chests of living animals. I have twice thought only that I had seen the independent movements of these organs; but I confess myself not satisfied with these experiments. But the moment we expose the pleuritic surfaces of the lungs to the atmospheric air, we submit them to a pressure which may prevent their expansion: the animal, too, is in a struggling and expiring state. We should not forget the advice given by Laënnec, that, before we draw our conclusions from experiments on living animals, we should always defalcate the anomalies arising from the agonies of death.

But we have sufficient proofs from auscultation of the active expansion of the lungs, without resorting to experiments, which, however necessary for the advancement of physiological science, are always performed with reluctance. Thus the most active voluntary movements of the chest can never so dilate the air-cells as to induce puerile respiration; shewing clearly that that condition does not depend upon external movements, but upon the active expansion of the lungs themselves. Again, if any portion of a lung be impermeable to air, puerile respiration will arise in some other portion, even when the movements of the chest are not perceptibly increased.

I may state, here, that asthma from increased necessity for respiration consists in too active an expansion of the air-cells,—a state exactly opposed

to asthma from spasm, but both evidently arising from some peculiar condition of the nervous system, of the nature of which we are at present totally ignorant.

Asthma, from Combinations of Organic and Nervous Causes.

It is somewhat rare to meet with purely nervous asthmas; but those from combined causes are very frequent. It is impossible for me to enter into the details of these combinations, as it would lead me far beyond the limits which a course of lectures like this permits; but if you have well understood what I have said, you will have no difficulty, by a little reflection, in anticipating these complications, since you are now acquainted with all the elements from which they are formed. Thus, —

Asthma with puerile respiration may be combined with all the forms of catarrh, with emphysema and œdema of the lungs.

Spasmodic Asthma may have precisely the same complications: so that if you would extend the tabular view I have already given, it might be formed in the following manner: —

Asthmas from combinations of organic and nervous causes.	{	Asthma from increased necessity for respiration, combined with . .	{	Chronic mucous catarrh	} œdema
				Chronic pituitous catarrh	
				Dry catarrh.—Emphysema pulmonum.	
	{	Spasmodic asthma, combined with	{	Chronic mucous catarrh	} œdema
				Chronic pituitous catarrh	
				Dry catarrh.—Emphysema pulmonum.	

The pathognomonic signs of these varieties will, of course, depend upon the nature of the complication. In asthma from increased necessity for respira-

ation, the puerile respiratory murmur will be the essential sign; if it be combined with chronic mucous, or pituitous catarrh, the local signs of these affections will be present also — as the sibilating sound, the deep sonorous wheeze, or the mucous rattle. The yellow or white expectoration will determine which species of catarrh it is. If œdema coexist, then its sign of rhonchus crepitans will be distinctly heard; and these would be forms of humid asthma. When dry catarrh is the complication, then sibilating or sonorous wheezing will be present; and the expectoration at the termination of the fit consists of a few pearly sputa, mixed with a small quantity of diffluent pituitous matter. This constitutes a form of *dry asthma*.

Spasmodic Asthmas are still more commonly complicated with organic changes than the preceding. In these cases, superadded to the almost general absence of the respiratory murmur, the various species of rhonchus may be heard at different parts of the chest; and the appearance of the expectoration will show the character of the complicating catarrh.

Percussion elicits a good sound in all the forms of asthma, simple or combined, since in asthma, from increased necessity for respiration, there is an excessive quantity of air in the lung; and in spasmodic asthma some air is always confined below the obstructing cause. An emphysematous complication renders the sound upon percussion much louder.

Relative to the signs, I should also state, that there are other essential differences between the simple nervous, and the organic and compli-

cated asthmas. In the simple nervous forms, no dyspncea or local signs of organic pulmonary lesion remain after the paroxysm is over; whilst in the organic and combined, the patient is always affected with more or less difficulty of breathing; and there constantly exists afterwards some local sign indicating a material change in the bronchial or parenchymatous structure of the lungs.

After repeated attacks of nervous asthma, some organic pulmonary or cardiac lesion, or both, most frequently supervenes. Dry catarrh is the most common consequence, although the other forms of catarrh are not infrequent. If the asthma has been of long duration, emphysema of the lungs is an inevitable result. The cardiac lesions we shall advert to when we arrive at the description of diseases of the heart.

Remote Causes of Asthma.

The remote causes of organic asthmas are precisely those of catarrhs. I refer you to that subject.

The remote causes of nervous asthmas are evidently those which are considered to act upon the nervous system. The disease attacks peculiarly persons of a nervous or irritable temperament. The principal causes are as follow: — Emotions of the mind, venereal excesses, the presence of a strong light, or the absence of light. A paroxysm of asthma has often been induced by certain odours — as of the tuberose, the heliotrope, of masses of apples, the smoke arising from the snuff of a candle. If a body be moved before the face of some persons, they will immediately feel a sense of closeness

and suffocation. A very opposite cause will bring on the attack occasionally. Laënnec mentions the case of a gentleman who, in riding on horseback, arrived at an extensive plain ; as he proceeded upon it, gradually a sense of suffocation came on ; he returned, and the sensation went off. He again attempted to proceed on his journey, and the dyspnœa again returned. He finally was obliged to give up all thoughts of proceeding onwards. A young friend of mine cannot touch ipecacuanha without being thrown into a violent paroxysm of asthma. Certain associations often produce the fit. Dr. Bree, in his excellent work on this disease, states, that after having suffered a severe asthmatic attack on a certain spot, he could never pass it without experiencing its renewal. I have met with similar instances. I have known strong green tea to produce the disease violently. I knew, also, two clergymen, on whom the paroxysm was arrested by active mental exertion ; for in ascending the pulpit, the dyspnœa gradually disappeared, and they improved in voice as they warmed with their subject.

Treatment. — Continued Chronic Difficulty of Breathing.

I need not allude to the treatment of continued chronic dyspnœa, since I have already described it in speaking of the various diseases which produce that effect.

Asthma, or Intermittent Difficulty of Breathing.

1. *Asthma from Organic Causes.* — The treatment in these cases must be directed to the paroxysms, and to the intervals between them.

Chronic Mucous, or Pituitous Catarrhs. — As the paroxysm of asthma from these causes depends upon a sudden accumulation of secretions in the bronchial tubes and air-cells, our indication is to promote as free an expulsion of them as possible. Emetics, by the succussion they afford to the lungs, will often effect this object. Ammoniacum, squills, antimony, and ipecacuanha, are also used as expectorants, and frequently with success. Inhalations of the vapour of water, or of vinegar and water, often succeed in loosening the viscid phlegm, and occasion its easy expectoration. Bleeding is rarely of advantage in these cases; for it is our object to preserve the strength of the patient as much as possible: if he be strong and plethoric, it may be used with some advantage; but the application of leeches, or the scarificator, are safer remedies. Counter-irritants may always be applied, — as blisters, or, what are more immediate in their effects, mustard cataplasms, or stimulant liniments.

I would strongly recommend you to avoid the use of opium, particularly in old persons, for there is generally a great tendency to coma already; and if the patient sleep profoundly, the accumulation of mucous or pituitous matter increases; he loses the power of expelling so great a quantity, and dies of suffocative catarrh. I have seen instances of this.

Dry Catarrh. — Asthma from this cause requires a different treatment from the preceding species. This disease consists of a sudden ingrafting of a fresh inflammation upon the chronic state, by which the mucous membrane becomes additionally swollen, and more completely obstructs the bronchial tubes. General bleeding may now be used with less caution; still it is rarely necessary: the application of leeches, or the cupping-glasses, are usually sufficient: the use of ipecacuanha and tartar emetic, in nauseating doses, frequently dissipates the paroxysm.

The treatment of the intervals between the paroxysms in the organic forms of asthma is precisely the same as I have described for the various species of catarrh, œdema, or emphysema of the lungs.

2. *Asthma from Nervous Causes.* — *Asthma from increased Necessity for Respiration* is so infrequent an affection, as to have afforded but little experience of the mode in which it should be treated; but as we find that the necessity for respiration is less during sleep, it is fair to infer that narcotics might be used with advantage. I have administered opiates in all the cases I have seen, and, I think, with good effect. Various other means have been used with this view, as the belladonna, stramonium, aconite, colchicum, tobacco, conium maculatum, hyoscyamus, nux vomica, boletus suaveolens, narcissus pseudo-narcissus; also certain irritants, as the tincture of cantharides, Fowler's solution of arsenic, arsenic in vapour, sulphate of zinc, and muriate of barytes. Laënnec, after having given this list, gives the preference to narcotics, and then to the distilled laurel-water, or hydrocyanic acid.

Asthma from Spasm. — Most of the above remedies answer as well for this species as the preceding. To the above list may be added, the foetid gums, æther, musk, castor, camphor, myrrh, ammoniacum. I have tried the lobelia inflata pretty frequently, but I cannot say I have met with all the advantages which have been attributed to its use. Very strong and clear-made coffee often produces an excellent effect. The inhalation of oxygen gas, recommended by Fourcroy and Beddoes, has fallen into total desuetude: I have no experience of its value.

Electricity and galvanism have not produced the effects anticipated from them. Mineral magnetism has also been proposed. I have lately, as you are aware, been making some experiments with it upon the neuralgiæ, and with a certain degree of success, but not sufficient to warrant as yet a safe conclusion.

Combined Forms of Asthma. — The treatment of these cases must consist of a combination of that of the organic and nervous forms of asthma.

After all the remedies I have mentioned, it must be admitted, that their effects are extremely uncertain upon the paroxysm of any form of asthma: one medicament seems to suit one person, another a second, and none a third. You may form this conclusion, — that whenever you find a great number of means proposed for the cure of any disease, it is an affection of an intractable nature.

Although therapeutics offer but little certain in the treatment of the asthmatic paroxysms, yet much may be done to prevent them by a judicious treatment of the intervals between. Thus you will find asthmatics very subject to dyspepsia: you should

obviate that affection by a close attention to the stomach and bowels, which may be done by the occasional administration of the warmer purgatives combined with antacids, as the rhubarb and magnesia; and these may be followed by tonics, as gentian, cinchona, and, above all, the carbonate of iron.

The diet should be light, yet nutritious; the patient should clothe himself according to the season, and let his own feelings of comfort be the guide to the quantity of vestments. He should not expose himself to sudden changes of temperature: a change of air is often highly advantageous; but you must often be directed in this by the experience of the invalids themselves; for some feel better in the country, others in town. Some, too, breathe most freely at a high elevation, and others on a low plain.

Habitude, no doubt, has a considerable effect in producing the paroxysm; in that case, a total change of position and employment should be recommended, where practicable.

Oct. 18/40

LECTURE XV.

DISEASES OF THE PARENCHYMATOUS STRUCTURE
OF THE LUNGS.

ADVENTITIOUS DEPOSITS.

TUBERCLE.

Adventitious Deposits. — Definition. — Inorganic, Organic. — *Tubercular Deposit.* — Table. — *Miliary Tubercle* — First Period — Second Period. — *Tubercular Granulations.* — *Encysted Tubercles.* — *Infiltration* — Gray — Gelatine, form softening of Tubercular Matter. — Chemical Analysis. — *Tubercular Excavation* — Columns of Bayle — multilocular, unilocular Excavation. — Effects upon large Blood-vessels and Bronchial Tubes. — *Tubercular Fistula* — Lining Membranes. — *Tubercular Cicatrix.* — Situation of Tubercles — Succession of Crops. — Recapitulation. — Coexisting Tubercles in other Organs. — Laënnec's Table — Louis's Table. — Mortality in Children from Tubercular Disease — Table by Lombard de Genève. — Comparative Mortality in Adults from Tubercular Disease — Table by Bayle. — Duration of Phthisis — Table by Bayle. — Mortality according to Seasons — Table by Bayle. — Lesions consequent upon Tubercular Disease — Table by Louis.

I now call your attention to that class of diseases of the lungs which are consequent upon adventitious deposits in their structure.

Definition. — By adventitious deposits we mean matters placed in the substance of an organ, foreign to its normal structure, depending upon an aberration of secretion or nutrition, and having for their consequences the atrophy or destruction of the organ, to a degree proportionate to their quantity and nature.

Inorganic and Organic. — There are two classes

of adventitious deposits. The first we shall call inorganic, because no vessel has ever been seen to exist within them. They appear to grow by external accretion. The most important of these is *Tubercle*, the immediate cause of phthisis pulmonalis. The second are organic; so called because they are more or less vascular. They grow from within, and occasionally attain a considerable volume. The organic adventitious deposits have been considered by Cruveilhier as parasitical growths, connected with the living animal by a system of vessels plunging into their mass, and increasing it by the deposit of similar matter: these are of rare occurrence in the lungs: I shall dwell, therefore, but little upon them, but devote almost the whole of our attention to the important subject of tubercular disease.

TUBERCULAR DEPOSIT.

Tubercular matter has for its result the accumulation and succession of symptoms known under the appellation of *phthisis pulmonalis*; and observe, gentlemen, that I restrict that term to tubercular disease alone.

Tubercular matter may be defined to be an inorganic substance, varying in form; so that it may be isolated, or infiltrated, in irregular masses in the substance of the lungs. It is usually at first gray, and semi-transparent, becoming afterwards yellow and opaque, being then called *crude*. It finally softens, and is expectorated, leaving an *excavation* in the lungs, which excavation may be lined with a false membrane. The cavity is then denominated a

tubercular *fistula*. The parietes of the fistula may approximate, unite, and form a *tubercular cicatrix*.

The following is a tabular view of the various forms which tubercular matter presents at its commencement:—

Isolated Tubercular Matter.

1. Miliary.
2. Granulations.
3. Encysted.

Infiltrated Tubercular Matter.

Gray.

Gelatiniform.

In whatever form the tubercular matter appears at the period of its primitive deposit, it subsequently becomes yellow and opaque, or, as it is called, *crude*; after which the mode of destruction of all the forms is the same, and the effect upon the lungs identical; that is to say, in its softening, formation of excavation, fistula, and cicatrix.

I shall first describe the various forms of tubercular matter in the lungs to the period of *crudity*, and then the common effects upon the pulmonary structure.

Miliary Tubercles. — *Period 1.* — The first form to be considered is the miliary tubercle: its size is generally small, something like that of a millet seed: it appears to be round, at least to the unassisted eye; but, on examining it with a powerful glass, it is found to be somewhat angular: its colour is gray, and it is transparent; its firmness is nearly equal to that of cartilage; it adheres with considerable force to the pulmonary structure, so

much so, that, on tearing away a tubercle, you detach also a portion of the pulmonary substance. The position of these tubercles is most commonly in the cellular texture separating the cells from each other; but they may also exist in any other part of the tissue of the lungs, as in the cells themselves. As to quantity, there may be very few, — four, five, or six, — or there may be thousands, for they are sometimes perfectly incalculable: I have seen the whole lungs completely gorged with them.

Period 2. — In the second period, the tubercle becomes enlarged by accretion to its external surface: it cannot grow from within, since it is inorganic, at least, no injection can be made to penetrate it: another proof is, that, however large they may become, the original form is still preserved. When there are few, the isolated tubercle may acquire a very considerable size, even to that of an almond. The colour now changes, and a small yellow spot is seen towards the centre; and this tint gradually pervades the whole of the tubercle to its circumference. In this stage it is called the crude yellow tubercle. These tubercles, in consequence of their increased growth, approximate to each other. Fresh ones form also in the intermediate pulmonary tissue, so that a large yellow amorphous, or irregular mass, of cartilaginous density, is the result. If you examine closely into a section of this mass, you will find that the tubercles are not so impacted as to prevent their original rounded form being here and there observable.

Tubercular Granulations. — They are very small, and generally perfectly uniform in their volume; their form is round or ovoid, their colour gray and

transparent, and they exist in innumerable quantities in the otherwise healthy lungs: these tubercles generally continue isolated, and rarely form in groups; when they do, a section of the mass shows them still separated from each other, by a tissue perfectly or slightly infiltrated with serum.

The centre of the granular, like the miliary tubercle, becomes yellow and opaque, which appearances gradually spread to its circumference, and then it is called *Crude*.

A dark spot is to be seen in the centre of either the miliary or granular tubercle, which disappears as the adventitious deposit enlarges. This coloration probably depends upon the presence of a particle of black pulmonary matter. They may also become coloured by the accidental presence of another disease; thus icterus will give them a yellow tinge, particularly on their surface; and in gangrene of the lungs they become black.

It sometimes occurs that, in consequence of the immense accumulation of tubercles, death may result before the process of softening takes place: these cases are very rare; I have seen but two or three.

Encysted Tubercles. — The next species, the encysted tubercles, is so rare, that I have never seen a single instance of it; and Laënnec, in the space of twenty-four years, only met with three or four: he describes the cyst to be of the consistence of cartilage; that internally it is rugous, although the rugæ are smooth and polished. Its external surface is attached firmly to the pulmonary texture, and the tubercular matter is so loosely adherent to the interior as to be very easily separated from it.

Gray Tubercular Infiltration.—This infiltration is frequently formed around tubercular excavations: it exists, though very rarely, independently of isolated tubercles: it forms large masses of a somewhat transparent grayish appearance. Under these circumstances the pulmonary tissue is of a cartilaginous density, humid, and perfectly impermeable to air: if, then, sections of it be made, their surfaces are smooth and polished, of a homogeneous texture, and the pulmonary cells are no longer distinguishable: small yellow and opaque spots appear disseminated in the mass; they gradually enlarge and unite, until the whole is converted into a body of *crude tubercular infiltration*.

Gelatiniform Tubercular Infiltration.—Between miliary tubercles, there is often found a matter of the consistence of jelly, colourless, or slightly sanguineous; the natural appearance of pulmonary structure is there utterly lost; no air-cells can then be seen; gradually this matter becomes more consistent, and is transformed into the species just described; yellow spots are frequently seen in the most liquid and transparent part of the deposit, showing the commencement of its conversion into crude tubercular infiltration. This formation is, probably, only a modification or germ of the preceding species.

The transformation of these two forms of tubercular infiltration into the crude state is sometimes so rapid as to present no traces of their primitive appearances: they then are seen in irregular masses of a palish-yellow colour, of an angular, and never of the rounded form of ordinary tubercles. The tubercular matter is here evidently infiltrated

into the pulmonary tissue, totally destroying it, so that no remains of its texture are visible; whilst the miliary tubercle is plunged into the substance of the lungs, compressing but not uniting with it molecule by molecule. These masses often occupy a considerable portion of the organ, but never project beyond its surface.

Softening of the Tubercular Matter. — In whatever form the tubercular matter be first deposited, when it has arrived at the crude state, it softens and liquefies: this softening commences at the centre of each tubercle, or tubercular mass, or, if the mass be considerable, simultaneously at many points. Some authors assert that this process begins at the external surface: that it may occasionally do so, I do not deny; but, gentlemen, examine for yourselves, and you will find, on making a section of crude isolated tubercles, that the greater number of them are perfectly soft within, whilst their external parts are still in a state of considerable firmness. The softening proceeds until, at last, it affects the whole tubercle or mass of tubercles.

The softened matter may appear in two forms; sometimes it is like a thick pus, inodorous, and of a yellower colour than the crude tubercle; sometimes it is of the consistence of soft and friable cheese: this latter state occurs principally in scrofulous individuals, in whom also the matter is occasionally like fragments of curdy substance floating in whey.

According to Thenard's analysis, tubercular matter consists of —

Animal matter, principally fibrine	}	98.15
and gelatine - - -		
Muriate of soda, phosphate of lime,	}	1.85
carbonate of lime - -		
Oxide of iron, a few traces.		

Tubercular Excavation. — When the tubercular matter is completely softened, it bursts into the nearest bronchial tubes, and is gradually expectorated, leaving an excavation in the lung: the orifice of communication being narrower than the excavation into which it opens, becomes, as well as the cavity itself, fistulous.

It is rare to find one excavation only; it is generally surrounded by miliary tubercles, or masses of tubercular infiltration, which gradually soften in their turn, and burst into the primitive cavity, often forming irregular sinuses, communicating more or less freely with each other, and occasionally extending to the very extremity of the lung.

It is in this way that bodies, presenting somewhat the appearance of the *carinæ columnæ* of the ventricles of the heart, are seen to traverse these excavations: they are usually thinner at their centres than at their parietal extremities, and were thought by Bayle to be blood-vessels: they are, however, merely portions of lung in a state of crude infiltration. Often, too, masses of lung of amorphous forms are attached to the parietes of the cavity, hanging loosely within it: these are also pieces of lung in a similar state. It has happened, though rarely, that a small portion of these masses has broken off, and remained in the excavation as a foreign body.

The columns or masses I have described form, in their interstices, by their confused arrangement, a series of irregular sinuses rather than cavities; or you may consider the whole a general excavation, intersected by a number of incomplete partitions: it then may be called a *multilocular tubercular excavation*. As these infiltrated masses successively break down, the cavity becomes more open; and when their destruction is complete, it is called a *unilocular tubercular excavation*.

The tubercular matter appears to throw aside the large blood-vessels, for they are often seen spreading along the parietes of the excavations: these vessels are frequently flattened, though but rarely obliterated, except in their ramifications directed towards the cavity; for no coloured matter can be made to penetrate the excavation. This fact was also observed by Storck and Baillie: Storck found these vascular extremities obliterated by coagulated blood. The vessels passing into the columns become impervious a little after they have entered into them.

The bronchial tubes, instead of being thrown aside, are evidently enveloped in the tubercular mass, and become involved in the general destruction of the portion of the lung in which they are placed, so that these vessels are not opened at their sides to transmit the softened matter, but appear clearly cut at the level of the parietes of the cavity.

One or many bronchial tubes always open into a tubercular excavation.

Tubercular Fistula. — As the excavation becomes emptied of its softened contents, its parietes

are covered by an opaque, thin, soft, and friable membrane, which may easily be detached. It usually lines the whole cavity: sometimes, however, a partial pseudo-membranous exudation is found, thicker, of greater transparency, less friable, and more adherent: its thickness is generally unequal.

Often the last described membrane is superimposed upon the previous one; it is then but loosely attached, and is even lacerated at many points, so that the second membrane appears to be only the primitive stage of the first.

The two membranes I have already described are succeeded by a third, which completely lines the parietes of the excavations; it presents a greenish tint, is unequal in its thickness in different parts of the cavity, and is of semi-cartilaginous density. The tubercular fistula may now be said to be completely formed.

Tubercular Cicatrix. — Tubercular fistula is evidently the result of nature's efforts to cure the disease, by establishing an internal cicatrix; but the healing process is sometimes carried on still farther, by the parietes of the tubercular excavation closing and uniting with each other, and completely obliterating it: I have seen many instances of this process, and there are before you preparations demonstrating it. In one case there had been a considerable cavity in the upper lobe of the right lung, and the parietes of its superior part being thin, and not adherent to the costal pleura, collapsed, and fell upon the floor of the excavation, and firmly united with it, so as to form a complete cicatrix. You perceive, gentlemen, that the lung offers a puckered appearance, arising from the

manner in which the parietes of the cavity had drawn with it that portion of the lung situated above it which had not been destroyed. On pressing the cicatrix, it gives the resistance of cartilage. The cicatrix is here in a horizontal direction.

I present you with a preparation in which the cicatrification of the cavity had proceeded in a direction perpendicular to the lung. The cavity had evidently been of considerable size; its sides had collapsed, united, and formed a large cicatrix. You perceive, by closely examining the section of it, that it consists of two layers of cartilage, almost completely united at all points; leaving, however, here and there slight separations, showing that the union had not been perfectly effected. In the same preparation there is a section of another cicatrix which takes an oblique direction.

It sometimes occurs, as I have already stated, that, when there is a vast accumulation of tubercles in the lungs, death takes place, whilst they are yet in the gray state, or, at least, not advanced beyond the period of crudity. It occasionally happens that, when they are but few, they are all found softened, or that excavations are completely formed; far more commonly, however, the tubercular matter is discovered in every state, from the gray and transparent to the final cicatrification of the excavations formed by them. Thus this deposit is almost invariably first placed in the superior lobes of the lungs; there, consequently, the process of destruction commences, the excavation is primitively formed, and the rest of the organ may be healthy, and perfectly free from tubercles; but it is more usual to find them crude or softened, and exca-

vated, in the upper lobes, and gray, transparent, and frequently in innumerable quantities in the middle or lower. From these appearances, it is inferred, that all the tubercular matter found in the lungs could not be deposited at once, but that it is formed in successive crops. It is indeed difficult to conceive that so large a quantity as is sometimes seen could have been deposited at one effort; or, being so, what could have retarded their destruction in some parts of the lung, and accelerated it in others. It is much more reasonable to suppose that these deposits occur at successive periods.

I have now, gentlemen, demonstrated to you the various forms and the mode of destruction of the tubercular adventitious deposit in the lungs: you have seen that it may exist in that organ in the isolated state, or infiltrated into its substance; that the matter is primitively of a gray colour and transparent, afterwards becoming yellow and opaque; that then nature endeavours to reject it to the exterior by softening it, and thus putting it into a form in which it can be readily expectorated; that the result of the softening is the partial destruction of the lung by the formation of a cavity within it; that nature endeavours to repair the loss by converting the cavity into a fistula; and that occasionally the reparation is rendered still more complete by the union of the parietes of the fistula, and the consequent formation of a cicatrix.

We have thus, gentlemen, studied the tubercular deposit as it occurs in the lungs; let us now examine certain conditions of the body, caused by, or concomitant to, this adventitious formation.

Tubercular matter is rarely found in the lungs alone; it coexists in various other organs. Laënnec did not neglect this part of the subject, but has given us a list of the different parts in which they may be found, placing the organs in the inverse order of the frequency of deposit; thus they are seen, in the following table, existing most commonly in the lungs, then in the bronchial glands, then the mediastinal, &c.

Lungs.	Epididymis.
Bronchic glands.	Testes.
Mediastinal glands.	Spleen.
Glandulæ concatenatæ.	Heart.
Mesenteric glands.	Uterus.
All the other conglobate glands.	Cerebrum.
Liver.	Cerebellum.
Prostate gland.	Base of cranium.
Peritoneum.	Bodies of vertebræ.
Pleura.	Intervertebral substance.
In the false membranes formed in the peritoneum and pleura.	The bones.
	In cancers.
	Voluntary muscles.

Louis has examined this subject still more minutely, and has formed the following table, after the examination of three hundred and fifty subjects who had died of phthisis.

In the above number, tubercles were found in the following proportions: — In the

Small intestines	-	-	-	-	$\frac{1}{3}$
Large intestines	-	-	-	-	$\frac{1}{9}$
Mesenteric glands	-	-	-	-	$\frac{1}{4}$

Cervical glands	- - - -	$\frac{1}{16}$
Lumbar glands	- - - -	$\frac{1}{12}$
Prostate	- - - -	$\frac{1}{13}$
Spleen	- - - -	$\frac{1}{14}$
Ovaries	- - - -	$\frac{1}{20}$
Kidneys	- - - -	$\frac{1}{30}$
Uterus	} In one subject only.	
Brain		
Cerebellum		
Spinal cord		
Ureters		

It has been observed, that tubercular matter has been deposited in the right lung more frequently than the left, and that it exists more commonly in men than women.

Lombard de Genève, on examining a vast number of children, who had died of various diseases, found that the mortality arising from phthisis, compared with other affections, varied according to their ages. Thus—

It was rare in the foetus, and in the first months of life.

From 1 to 2 years, it was found in $\frac{1}{8}$

— 2 to 3 - - - $\frac{2}{7}$

— 3 to 4 - - - $\frac{4}{7}$

— 4 to 5 - - - $\frac{3}{4}$

and from five years to the age of puberty, less frequently than from four to five, but more than before four years.

You perceive, by the above table, that tubercular disease occurred the least frequently before two years, and the most between four and five.

Bayle had previously examined the proportion of deaths arising from phthisis, compared with

other diseases, and he found it to be above one third, or as 244 is to 696. He found also that the periods at which death occurred from this disease after the age of puberty, in one hundred cases, were as follows:—

From 15 to 20 years of age, there died 10

— 20 to 30 - - - 23

— 30 to 40 - - - 23

— 40 to 50 - - - 21

— 50 to 60 - - - 15

— 60 to 70 - - - 8

Bayle examined two hundred patients, with reference to the duration of the disease from its commencement to its fatal termination. I think his table cannot entirely be depended upon, although it is no doubt as correct as the subject admits; for it is extremely difficult to obtain a precise history of a disease, especially from the lower classes of the people, who commonly date their complaints only from the period they became unable to attend to their occupations.

Table of the Duration of Phthisis before Death was produced, in 200 Cases.

Quarters of the Year.	Cases.	Half Years.	Cases.	Years.	Cases.
1st quarter	16	1st half year	60	1st year	124
2d	44				
3d	44	2d	64	2d	48
4th	20				
		3d	30	3d	6
		4th	18	4th	5
				5th	3
				6th	1
				7th	3
				8th	1
				9th	3
				From the 9th to the 40th	6
					200

In reference to the seasons of the year in which the mortality occurred, he found it as follows in 240 cases :—

There died in the spring	-	-	-	54
————— summer	-	-	-	68
————— autumn	-	-	-	64
————— winter	-	-	-	54
				—
				240
				—

We find that various lesions of different organs of the body are consequent upon the existence of tubercles in the lungs. Louis has investigated this subject with great patience ; and the following observations are principally derived from his work.

The absorption of the fat in the cellular tissue is often extraordinary : this secretion diminishes greatly in the orbits, so that the eyes sink ; it disappears between the buccinators and masseters, and the rotundity of the cheek is lost ; in extreme cases, the outer surfaces of the teeth are seen elevating the skin upon them, giving, as Areteus expresses it, the appearance of a ghastly grin or smile. From the loss of the soft parts, the malar bones appear projecting. The fat of the whole body becomes at last totally absorbed.

The intestines, brain, nerves, spleen, pancreas, and other glands, do not diminish.

The liver becomes larger, and is also fatty in about two thirds of the cases. This appearance occurs more frequently in women than men.

The specific gravity of the bones diminishes ; and where there is great marasmus, their circumference lessens.

The chest contracts at the sides from previous pleurisies, and often also at its anterior superior parts, in consequence of the presence of large and old excavations.

The muscles and the heart are smaller and redder than natural. The caliber of the aorta is generally diminished, and its internal surface is reddened in one fourth of the cases.

The mucous surfaces are peculiarly subject to alterations. Louis found, in examining 102 subjects who had died of phthisis, —

Ulceration of the larynx	- - -	in 23
———— epiglottis	- -	in 18
———— trachea	- - -	in 31

In most cases the ulcers occupied the posterior surface of the air-tube, and the laryngeal face of the epiglottis.

The stomach was affected in the following proportion in ninety-six cases of phthisis; and a comparison was also made by Louis of the same lesion of this organ in ninety-six cases of death from other chronic diseases.

Stomach enlarged to double or triple its volume	- - -	9 times.
Same lesion in ninety-six cases of other chronic diseases	- - -	2 do.
Softening with thinning of mucous membrane of the stomach	- -	19 do.
Same lesion in ninety-six cases of other chronic diseases	- - -	6 do.

The mucous membrane of the different parts of the intestinal tube was diseased in the following proportions : —

Duodenum—ulcerated, rarely.

The glandulæ agmenatæ of the jejunum and ileum ulcerated in five sixths of the cases.

The mucous membranes of the small intestines were rarely thickened or softened.

The large intestines not so frequently ulcerated, but their mucous membrane softened in three fourths of the cases.

Ileo-colic valve.—It is rare, indeed, not to find ulcerations there.

Fistula in ano occasionally coexists with tubercles in the lungs.

LECTURE XVI.

DISEASES OF THE PARENCHYMATOUS STRUCTURE
OF THE LUNGS.

PHTHISIS PULMONALIS.

TUBERCLE — *continued.*

Local Signs. — First, Accumulation of gray or crude Tubercles, Respiratory Murmur, Bronchophony, Percussion. — Second, *Softening* of Tubercles, Crepitation, Gurgling, Mucous Rattle. — Third, *Tubercular Excavation.* — Cavernous Cough and Respiration. — Percussion, “Souffle.” — “Souffle voilé.” — Pectoriloquy — its varieties and causes.

Functional Signs. — Dyspnœa, Cough, Expectoration. — Analysis of Pus — its value — Sources of expectorated Fluid. — Pain. — Oppression. — Paraphonia.

General Signs. — Hectic Fever, Rigors, Night Sweats. — Urine. — Anorexia, Diarrhœa, Catamenia, Pulse, Debility. — Aretæus’s Description. — Case. — *Causes.* — *Remote* — Individual — *External* — *Proximate.*

Treatment — Indications. — First, to prevent the Formation of secondary Crops. — Second, to favour softening of Tubercles. — Third, to cicatrise Ulcerations. — General Plan of Treatment.

WE have thus, gentlemen, described the morbid anatomy of tubercles in the lungs : we now proceed to their signs.

Local Signs.

1. *Of the Accumulation of gray or crude Tubercles — Respiratory Murmur.* — When but few gray or crude tubercles exist, the pulmonary structure between them is yet healthy, so that the respiratory murmur may be distinctly heard. There is then,

*few in
heard*

many, murmur lost upper lobes
in fact, no local sign of their presence; but when they are accumulated in masses, so as to encroach considerably upon the texture of the organ, the respiratory murmur is completely lost at the points of accumulation. I have already stated that tubercles are always primarily deposited in the upper lobes of the lungs; it is therefore immediately below the lines of the clavicles, and in the axillæ, that the absence of the respiratory sound occurs first. When you suspect tubercular disease, examine these regions with the greatest care; apply the stethoscope to the skin itself, press it firmly, and place the ear upon the instrument, so that no rustling sounds shall be produced, otherwise you may easily be deceived.

Bronchophony. — As the softening process has not yet commenced, the bronchial tubes traversing the tubercular matter are intact; and the consequence is, that, when the patient speaks, the voice is resounded in these vessels, constituting the bronchophony of Laennec. This sound is to be sought for immediately below and along the lines of the clavicles, in the supra spinal fossæ, and in the axillæ; but if it be heard at the internal and superior angles of the scapulæ, no inference should be drawn, because of the presence of large bronchial tubes, which sometimes occasion that sound there naturally.

many dull sound
Percussion. — When the tubercles are isolated, and at some distance from each other, the resonance upon percussion is natural; but as they increase in number, so as to form masses, then percussion elicits a dull sound. The diminished sound extends often from the clavicle to the fourth rib,

and is also found in the axilla. Absence of respiration, bronchophony, and dulness of resonance upon percussion, usually coexist in this stage of the disease at the same parts of the parietes of the chest.

2. *Softening of the Tubercular Mass.*—The above signs persist for some time; but there is now superadded a slight crepitating sound, varying in duration in different individuals. This crepitation is followed by a gurgling noise (the *gargouillement* of Laennec), as if produced by the movement of a thick matter. As the tubercular mass becomes still softer and thinner, a distinct mucous rattle becomes also daily more distinct.

3. *Tubercular Excavation — Cavernous Cough and Respiration.*—When the tubercular matter is expelled, a cavity is left in the lung. The emptying of this cavity is rendered evident by a cough or a forced respiration; in both cases loud sounds are produced, called the *cavernous cough and respiration*. While the softened matter is present, these cavernous sounds are accompanied by a mucous rattle. When the cavity is evacuated, these signs give the idea of the cough and respiration being resounded in a capacity of much larger dimensions than usual.

As the cavity empties, it occasionally happens that the sound produced by percussion becomes clearer; the contrary is most frequently the case, because a large quantity of tubercular matter generally surrounds the excavation.

When the softening matter is very near the surface of the chest, a gurgling sensation may sometimes be felt by the hand: this is caused by the rattling of the fluid, especially when the patient

coughs: he occasionally even feels it himself. When the anterior parietes of the excavation are very thin, and the patient is much emaciated, percussion elicits a sound something similar to that produced by striking a broken vessel — as a cracked earthenware pot.

If, also, the excavation be very near the surface, the blowing respiration, or “souffle,” takes place; that is, whenever the patient inspires quickly, he appears to draw the air from the ear of the observer, and, on expiration, he seems to drive it forcibly back again; every time he speaks, the same sensation is felt, although it rather immediately follows the voice than accompanies it. To distinguish this well, the patient should be made to speak in monosyllables. The modification of the blowing respiration, called the “souffle voilé,” is also given when the anterior parietes of the cavity are very thin, soft, and not adherent to the costal pleura. It appears, then, that every vibration of the cough, voice, or respiration, agitates a moveable partition between the excavation and the ear of the examiner.

When the cavity is completely empty, the cavernous cough and respiration are rendered perfect; the mucous rattle now disappears, sometimes to recur slightly, in consequence of the secretions going on in the parietes of the excavation.

Pectoriloquy. — The most interesting of all the signs of an evacuated cavity is the pectoriloquy. It may, however, exist in an imperfect state before the evacuation is complete. I have already endeavoured to account for this sign; but this is the time to describe the circumstances by which its

varieties are produced. I shall present to you Laënnec's observations upon this subject.

We should conclude nothing from doubtful pectoriloquy, when it occurs in the interscapular regions, in the axillæ, or at the union of the clavicles with the sternum, or even from the clavicles to the third ribs. The sign is doubtful if it exist equally on both sides of the upper part of the chest. The cause of these doubts is, that the upper lobes contain a larger proportion of bronchial tubes of greater diameter than the rest of the lungs, and some of them frequently take a superficial course; so that what is really bronchophony may be mistaken for a doubtful pectoriloquy.

If, too, the stethoscope be applied perpendicularly upon the upper surface of the clavicle, between it and the superior edge of the trapezius muscle, the resonance of the voice from the trachea and larynx is sometimes very similar to pectoriloquy.

But if a doubtful resonance of the voice be found below the third or fourth rib, or on one side only, there is then a strong presumption that it is pectoriloquy: if the sound does not exist at the same time at the points of the chest corresponding to the upper lobes, the sign is certain. The conclusion should then be, that the cavity is deeply seated, or that it is yet partly filled with incompletely softened tubercular matter.

On whatever part of the chest the voice is resounded more loudly than on the corresponding part of the opposite side, and particularly if it be so intense as to appear stronger and nearer to the ear of the observer than that which proceeds from

the mouth of the patient, the sign is then as certain as if the articulation were heard distinctly passing up the tube, and the pectoriloquy is *imperfect*, but not *doubtful*. Between perfect and doubtful pectoriloquy there are many intermediate degrees, which practice alone can teach, and which it would, perhaps, be impossible to describe.

The pectoriloquy is always most evident in persons having acute voices, as in women and children.

In men whose voices are very grave, the pectoriloquy is often imperfect and doubtful, even when the excavations are in a proper state to produce that sign. The voice then appears agitated and trembling; it seems to be unable to introduce itself into the stethoscope, but resounds loudly at its extremity, and is heard as if through a speaking trumpet. But it is a perfect sign of a cavity, especially if no such sound be audible on the opposite side of the chest.

The most evident pectoriloquy may present differences; thus the voice may pass continually through the tube, it may be intermittent, or some acute tones only may be heard: this intermittence occurs when the communicating bronchial tubes are temporarily obstructed by mucus. By making the patient cough, the pectoriloquy is easily reproduced.

In relation to the voice itself, the pectoriloquy may vary: the articulation of the words may be more or less distinct, the nature of the sound more or less altered: usually the voice is a little stifled. Sometimes the sound is fuller than from the mouth; it is commonly stronger, and the patient occasion-

ally speaks through the tube as if he placed his mouth to your ear.

Laënnec observes, that when the voice is reduced to a mere whisper, still the pectoriloquy exists.

The thinner the parietes of the excavation, the better is the phenomenon produced.

Moderate-sized unilocular cavities give the pectoriloquy best: small ones, however, occasion it to be very distinct. In a multilocular cavity the articulation of words becomes stifled and confused.

In cavities whose upper parietes are thin, and can collapse, the pectoriloquy may be entirely lost.

If the pectoriloquy be continued, evident, well articulated, and unaccompanied by any other sound, the excavation is complete, and its bronchial orifices are large.

When a cavity is very large, and is but little anfractuous, the pectoriloquy is entirely lost, but then the amphoric respiration and the metallic tinkling become evident; the first occurs most frequently. Laënnec states that the Hippocratic fluctuation rarely coexists with the metallic tinkling in these cases. I have tried to produce it in above twenty different instances, and have never succeeded.

It is necessary, for the production of the metallic tinkling, that the cavity should be exceedingly large, and that it should contain a certain quantity of fluid. If the quantity be very slight indeed, the phenomenon will not appear, but the voice, the cough, and respiration, are accompanied by the amphoric sound.

When an excavation bursts into the pleura, the pectoriloquy is usually lost; we shall advert to

this again, in speaking of the combination of *Pneumo-thorax with Empyema*.

Functional Signs.

Dyspnœa.—The degree of dyspnœa is often extremely variable. The patient, at the commencement of the disease, experiences more or less difficulty of breathing, which usually augments as the tubercles soften. The causes of the dyspnœa are not only the presence of tubercles, but also of catarrh. It might be supposed that the intensity of this functional sign depended upon the quantity of tubercles in the lung; but such is the general wasting of the body, such is the diminished quantity of blood in the system, the proportion sent into the lung is so small compared to the healthy state, that the necessity for respiration becomes extremely diminished; so that frequently, towards the termination of the disease, the patient suffers less from dyspnœa than might be expected from the organic lesions existing in the lungs.

Cough.—This symptom is present in all stages of the disease; it is slight and dry at its commencement, but as the tubercles soften, and the accompanying catarrh supervenes, it is attended by a more or less copious expectoration. The most violent coughs depend upon the presence of ulcerations of the larynx, epiglottis, and rima glottidis.

Expectoration.—It had long been thought that phthisis depended upon inflammation of the lungs, and that the matter expectorated was pus. Under this supposition, it became a subject of importance

to determine the difference between pus and the yellow secretion, or mucus, arising from inflammation of the bronchial tubes. The following differences were pointed out:—

<i>Pus.</i>	<i>Mucus.</i>
Dissolve both pus and mucus in sulphuric acid;	
pus precipitates, - - - - -	but mucus does not.
Oxymuriate of mercury does not coagulate pus; -	it coagulates mucus.
The microscope shows pus to consist of semi-opaque	
globules, - - - - -	mucus — of flakes.
Pus sinks in water; - - - - -	mucus floats.
Pus evaporates to dryness without first coagulating;	mucus coagulates first.

But the examination of these differences is of no use whatever in forming our diagnosis of phthisis, for the disease is not the result of inflammation of the lungs, and consequently the fluid expectorated is not pus, but a mixed matter derived from different sources.

The sources whence the expectoration is derived, are, 1st., the irritated bronchial tubes and trachea; catarrh is formed, and the mass of the sputa is constituted of mucous or pituitous matters, or a mixture of both. 2dly. The softened tubercular matter itself: this may put on a puriform appearance, or, what is still more common, it consists of whitish, curdy, irregular, small pieces, or flakes, mixed up in the catarrhal secretions. You should be careful, however, in examining whether they are not really curds of milk, for they are not infrequently the causes of this appearance. Certain curdy productions will also occasionally result from chronic inflammation of the amygdalæ; these glands should therefore be examined.

The quantity of the fluid expectorated is sometimes very considerable; for, though it may not

ordinarily be more than a teacupful, yet it occasionally amounts to more than a pint in a day. The mass of these secretions arises from the bronchial tubes. The softened tubercular matter can only be in very small proportion, since an excavation of the capacity of a pint measure is often many months in forming and evacuating its contents. The quantity, therefore, of softened tubercle contained in the matters expectorated has been supposed to be scarcely a thousandth part.

The third source of the expectorated secretions is from the parietes of the excavation; but the nature of this has not been sufficiently studied. It is probably but small in quantity; and if it be like the matter which may be scraped from the surface of the cavity, it is of a pituitous nature.

Blood in small quantity may be expectorated in any stage of the progress of tubercular matter. I have met with it before any expectoration had taken place, or, indeed, before the development of any local signs. It is commonly seen in streaks, or spots, in the expectoration. Hæmoptysis is rarely considerable; I have met with but few instances of its being the immediate cause of death: in one of them the bleeding arose from a ruptured vessel opening into the excavation.

Small quantities of ossific matters are sometimes mixed up with the expectoration; I shall show the sources of these when I speak of ossific concretions.

Pain — Oppression. — The patients rarely complain of pain, except slight pleurisies occur; they have not commonly, either, any sense of oppression in the chest, or, if present, it is but slight.

Paraphonia.— The voice, especially towards the close of the disease, often becomes hoarse, and reduced to a whisper; this, though partly arising from the exhaustion of the patient, is principally caused by the ulcerations in the larynx and trachea.

Such are the signs which may be referred to the lesion of the functions: we now proceed to those arising from the sympathetic disorder of the general system.

General Signs.

That general condition of the system known under the appellation of *hectic fever*, prevails in all the stages of this disease. It is least manifest during the crude state of the tubercles, most evident during the period of their softening, and diminishes when the excavation is completely formed.

Hectic fever has been classed among the remittents: it is said to have two accesses in the twenty-four hours, the one being in the middle of the day, and the other towards the evening; the latter being the longest. From long observation, I can say that the meridian access is rare. The access generally commences with horripilations (according to Louis, in five sixths of the cases), accompanied by great sensibility to cold, although the skin may feel warm to the observer. The hot stage soon begins, and lasts usually for many hours, terminating in the morning in perspiration.

These perspirations, or, as they are commonly called, *night sweats*, are highly characteristic of phthisis. The quantity is sometimes enormous, completely wetting the head and the whole body,

the night-clothes, and sheets: they have been seen penetrating deeply into the mattress or bed on which the patient is placed. These sweats often occur when the other symptoms of hectic fever are not marked. I have seen them in so early a stage of the disease, that the local signs were not present: indeed, their presence, if combined with frequency of pulse, is often alone a sufficient indication to the experienced practitioner of the approach of this fatal disease. There seems to be some relation between this sweating and sleep, for the profuseness of the one often depends upon the completeness of the other: even if the patient sleep at any part of the day, he almost always awakes in a state of perspiration.

The urine is usually high-coloured, and deposits a furfuraceous sediment. The thirst is rarely great, even during the febrile access. The tongue is often healthy at the beginning, but towards the termination of the disease becomes dry, of a deep red colour, its edges and point, and then its surface, and the throat, are covered by aphthous vesicles, leaving sores which distress the patient, and prevent free deglutition.

The lesions of the stomach are often shown by anorexia, nausea, and even vomiting: frequently these signs are absent, and the patient preserves a tolerable appetite to the last.

The ulcerated condition of the intestinal tube almost always causes *diarrhæa*: this is one of the most prominent and common signs of phthisis, and exists in every stage, but most frequently during and after the period of the softening of the tubercular matter.

The catamenia are usually diminished at first, and totally cease as the disease progresses. Females are apt to attribute the whole of their symptoms to this irregularity; but it is an effect, and not a cause.

The pulse is accelerated at all periods of the disease, but most during the hectic paroxysm.

Debility prevails from the commencement; yet this varies considerably. Some will take to their beds early, not to leave it; I have met with others who have been walking in the streets on the day of their deaths.

If the disease be slow and chronic in its course, extreme marasmus is produced. Aretæus gives us the following picture of it:—

The nose becomes thin, especially at its point; the malar bones project, and the skin covering them is of a brilliant red colour of a circumscribed form; the conjunctivæ shine, and are of a light pearly blue; the eyes are large, although somewhat sunk in their orbits; the cheeks are hollowed; the lips retracted, presenting often the appearance of a bitter smile. The neck is oblique, and as if oppressed in its movements; the shoulders are elevated; the ribs project; the intercostal spaces are depressed; the scapulæ are elevated like the wings of a bird; the belly is contracted and flattened; the articulations appear large; the nails are curved; and the hairs gradually fall from the head. No disease produces such great marasmus, excepting cancers, empyema, and fevers of long duration.

During the whole course of the disease all the mental faculties are perfect. If there be any thing

anormal, it is in the strong hope, nay certainty, which the patient has of his ultimate recovery.

Having described the symptoms of this formidable disease, let us now throw our observations into one point of view, by tracing a patient throughout its course.

I will imagine an individual of from 18 to 22, or 24 years of age, a female, brought to you, perhaps, by an anxious parent, whose solicitude is increased by having already lost some of her children by this insidious malady. You examine your patient; you perceive the eye to be of a bluish tint, and of a somewhat pearly lustre; that the cheeks are coloured with a red and unhealthy bloom, irregularly circumscribed; that her form is attenuated, the nails curved, the hair thinned and falling off, and the pulse frequent. Upon questioning her, she smiles at her mother's fears, and denies the existence of any ailment. You perceive she coughs slightly; that there is no expectoration, or merely a little pituitous matter — occasionally, perhaps, tinged with a spot of blood. She accounts for all this by attributing it to a recent cold; she admits that her respiration is somewhat quickened: even now there are frequent night sweats, irregularity of bowels, sometimes costiveness, sometimes diarrhoea. The catamenial flow is diminished in quantity, or irregular in its return.

Examine the local signs at this period: there may be none, for the tubercles may be yet gray, or crude, and not great in quantity; or there may be absence of respiration, bronchophony, and dulness upon percussion below the lines of the clavicles, or

in the axillæ, indicating the presence of tubercular masses yet unsoftened.

I will suppose that a few weeks have passed, and you are now called to visit your patient, for you are told she cannot leave her room.

The eye has still further assumed the pearly lustre I have described; the redness of her cheek is still more vivid, or there is a deadly paleness; the malar bones project; the cheeks have lost their rotundity; the dyspnœa is at its height; the cough constantly torments her; the expectoration is copious, consisting of a mixture of pituitous and mucous matters, with shreds of a curdy substance floating in it, and often also small spots or threads of blood. The hectic is now completely formed; in the evening a slight horripilation is succeeded by heat, which continues during the night. The patient sleeps, nevertheless, if the cough does not torment her, and awakes in the morning bathed in perspiration. Now, the urine deposits copiously a furfuraceous sediment; the diarrhœa is constant; the catamenia have totally ceased; the pulse beats from 100 to 130, or 140, in the minute; the weakness is great; but her never-failing hope still buoys her up: she is yet certain that she is labouring only from the effects of a common cold, from which she will soon recover.

This period coincides with the softening of the tubercular matter: the local signs evince it; for if you apply the stethoscope, a distinct crepitation, or the still louder sound of gurgling (*gargouillement*), is heard at the parts corresponding to the upper lobe of one or other of the lungs; an imperfect

pectoriloquy exists, and the cough and respiration are becoming cavernous at the same points.

Let us now suppose that the third and the last period has arrived: you will now find that the change is still greater; the malar bones are still more prominent; the cheeks still thinner; the lips are retracted, and give the bitter smile. The neck now appears somewhat awry, the ribs project, the intercostal spaces are deep, the scapulæ elevated, the abdomen is flat and contracted, the flesh of the whole body is extraordinarily reduced in quantity, the articulations appear large, and the nails more incurvated.

At this period the dyspnœa frequently diminishes, but the cough is often unceasing; not so much from increased fluid to be expectorated, as from the irritating ulceration of the larynx and parts adjacent. From the latter cause, also, the voice becomes hoarse, and reduced frequently to a whisper. The mouth, tongue, and fauces, become exceedingly tender from the aphthous eruptions.

The hectic fever now frequently becomes less; the night sweats even often cease, as if from diminished materials; the diarrhœa continues, and is colliquative. Hope still clings to the patient: she often will not hear of doubts of her recovery, but is constantly forming projects for the future. At last the pulse gradually sinks, the voice is lost, the power of expectorating fails, the head falls forwards, or laterally, by its own weight, and the patient frequently expires so quietly, that the attendants are for some time unaware of it.

If you examine the patient during the progress of this stage, you will find the excavation, or exca-

vations, complete; the pectoriloquy becomes, therefore, evident, and the resonance of the cough and respiration in the cavity cavernous. In some instances I have met with ravages so extensive, that nearly the whole of the lung on one side has been destroyed. In these cases the *bourdonnement amphorique*, and the *tintement métallique*, were perfectly distinct.

The three successive periods of the disease which I have just attempted to describe were denominated by the older writers — *phthisis incipiens*, *phthisis confirmata*, *phthisis desperata*.

Causes.

1. *Remote causes.* — Individuals of all temperaments are subject to tubercular deposits, but it most commonly occurs in those of the nervous and sanguine lymphatic. Persons thus constituted have the skin of a brilliant fairness of colour, and fineness of structure; the cheeks of a lively red; the neck narrow and long; the transverse diameter of the chest often diminished; the shoulders high; the scapulæ projecting; and, a general thinness of the whole frame. Phthisis attacks all ages, and both sexes, as I have shown you in the preceding lecture. There is, perhaps, no disease which results from hereditary predisposition so frequently as this. Moral causes, such as distress of mind, great seclusion, &c. may also be classed among the strongly predisposing causes.

The external causes are, — First, Cold, especially if combined with humidity. Phthisis is thus most common in the north of Europe and America; it

is a frequent disease also in France, Italy, and Greece, but generally less so in the meridional parts of Europe, and least of all in the tropics. In speaking of localities, it is found to be more frequent in large towns than in small ones, more common in the interior than on the coasts: it is rare on board ships. Secondly, Too slight clothing may be assigned as a cause of phthisis, by permitting the influence of atmospheric variations to act too suddenly upon the body. Thirdly, All debilitating causes, as venereal excesses, long courses of mercury, continued fevers of considerable duration, habitual intoxication, &c. Fourthly, Some trades have a tendency to produce the disease, as that of knife-grinding. Very sedentary occupations have a similar effect, as those of the milliner, tailor, weaver, &c.

It is thought, particularly in the south of France, that phthisis is contagious. I have seen no proof whatever of the correctness of this opinion. It is true the disease may occur in several individuals of the same family, about the same time; but this circumstance is much more readily accounted for from hereditary predisposition than contagion.

Proximate causes. — Although we shall be able to throw no light upon the proximate cause of phthisis, yet it will be of some advantage to refute the erroneous opinions that have been entertained upon this subject.

Thus, it was the opinion of the ancients that this disease was a consequence of an acute or chronic inflammation of the lungs; and this notion has been renewed by Broussais.

Can phthisis arise from inflammation of the lungs,

or *peripneumonia*? It is difficult to suppose so, since *peripneumonia* does not coexist with phthisis in a greater proportion than one tenth of the cases. Are the products of ordinary inflammation any thing like tubercles? Certainly not. *Peripneumonia* principally attacks the vesicular texture of the lungs, and often leaves the interlobular cellular substance intact; and yet, in the latter tissue the tubercular matter is most commonly deposited. *Peripneumonia* attacks primarily the inferior lobes of the lungs, in almost every instance; tubercles as frequently are placed in the upper lobes. The reverse should take place if tubercles were consequent upon inflammation of the organ. Tubercles are often seen imbedded in a perfectly healthy tissue, where there is no evidence whatever of inflammation. I have seen the lungs of a foetus studded with this deposit: was this from previous inflammation?

It is true that inflammation may accidentally accompany, or even be caused by, the irritation which the tubercular matter may produce; but it cannot be admitted to be the proximate cause of its deposition.

Can *catarrh* occasion the tubercular secretion? The latter is commonly attended by the former; but large tubercles have been seen without coexisting *catarrh*.

There is scarcely an individual in this country who is not even annually affected by *catarrh*. Phthisis, if depending upon that disease, should, therefore, be infinitely more common than it now even unfortunately is. *Catarrh* is most common

on the coasts ; phthisis in the interior. This order should be reversed if catarrh were the cause.

If tubercles depended upon catarrh, these deposits should not be found in the interlobular cellular tissue, since the bronchial tubes do not open into it ; but they are commonly formed there.

But catarrh and phthisis are two very distinct diseases ; the first is an inflammation of the mucous membrane of the air-tubes, having for its result a secretion, varying in character ; the second is the consequence of a peculiar adventitious deposit ; and, as Laënnec expresses it, no one has yet, scalpel in hand, shewed the conversion of catarrhal into tubercular matter.

Pulmonary Apoplexies have never been seen converted into pus or tubercles. Hæmoptysis often, indeed, seems to precede phthisis ; but, if the patient be carefully examined, it will commonly be found that the local signs of the latter disease are present at the same time. Tubercles frequently, by their pressure upon the vessels, cause spitting of blood ; but the quantity is usually small, and is derived from the bronchial tubes. This hæmorrhage must be considered as a consequence, and not as a cause.

Tubercles have been supposed to be a modification of *hydatids*. This opinion, I believe, has met with no supporters. I see no relation between the hydatid, even in its earliest budding state, and tubercle.

All we can say upon the subject of the proximate cause is, that this formation is a deposit from the blood-vessels. I am quite aware that this is no explanation, for all adventitious matters arise from

the same source. To determine the proximate cause, would be to determine the reason why the blood-vessels should deposit tubercle in one case, medullary sarcoma in a second, melanosis in a third, &c. The cause would probably be found in certain conditions of the system, or of the blood, peculiarly favourable for each particular deposit, the nature of which conditions we are as yet perfectly ignorant.

Treatment.

I now arrive at the least satisfactory part of this subject — I mean the treatment of phthisis; for I am convinced that art has done little more than to palliate this disease, and that when a cure has been effected, it has been by nature's efforts alone. I have shown the possibility of a cure by the cicatrization of the excavation; but I do not believe that this event occurs without the formation of a fresh crop of tubercles in one case in two hundred — perhaps not so frequently.

The following indications, which are proposed to be fulfilled by means sufficiently doubtful in their effects, have been laid down by authors.

1st Indication. To prevent secondary Crops of Tubercles. — *Bleeding* has been employed from remote antiquity, but it is rarely useful, except in intercurrent pleurisy or peripneumonia. I use the lancet only when there is considerable dyspnoea, and the patient yet possesses a certain degree of power, and when the pulse is full. Cupping, or leeches, may be applied under similar circumstances; but I do not believe that bleeding can

prevent the formation of a secondary crop of tubercles; I think it would rather tend to their production, by increasing the general debility of the system.

Counter-stimulants have also been extensively used, particularly by the ancients. Hippocrates applied the actual cautery at once to the axilla, back, and breasts; Celsus recommended it in six places — under the chin, on the throat, on each breast, and at the inferior angle of each scapula. These violent remedies have now subsided into blisters, setons, caustics, and occasionally also the moxa. Exutories relieve temporary pains and feelings of oppression in the chest, but cannot prevent the fresh formation of tubercles.

2d Indication. To favour the Softening of the Tubercles.—This indication was supposed to be accomplished by the use of certain medicaments believed to have a solvent power: among these were lime-water, sulphureous waters, either in baths or potations; the muriate of ammonia, the sub-carbonates of potash and soda; but these are very inert medicines. I have tried mercury, barytes, and iodine, extensively, but with no satisfactory results.

3d Indication. To cicatrize the Excavations.—A great variety of remedies have been recommended with this intention, as the following list will shew:—Antiscorbutic and aromatic plants, balsams of tolu, Mecca, and Peru, turpentine, and camphor dissolved in volatile oils; artificial atmospheres formed of the vapours of emollient, carminative, narcotic, and balsamic plants; also of certain substances burnt upon heated iron, as myrrh, ben-

zoin, petroleum, tar, resin, and wax ; sublimations of zinc, of sulphur.

Various gases have been employed : oxygen was recommended by Caillé ; hydrogen by Beddoes ; even sulphuretted hydrogen by Kortin ; carbonic acid by Beddoes also ; the vapours arising from stagnant pools, from stables ; the smoke of snuffed candles, heated air. The vapour of iodine, and chlorine gas, have been the latest remedies of this class proposed.

I have tried many of these remedies, and often with temporary advantage ; but I have no doubt that advantage has arisen from the relief they have afforded to the catarrh which accompanies the tubercular disease ; for, with a tolerable large field for observation, I must say that I never yet have seen a case whose *cure* could be attributed to any one of these medicaments. Most relief, I think, is produced by the inhalation of tar vapour, and of chlorine gas.

Many other means have been proposed, with the view of giving strength to the general system, or to relieve dyspnœa. Thus a milk diet is of very common use, and is often advantageous. The milk has been obtained from women, cows, asses, and mares : perhaps that from asses is the best. Charcoal, champignons, the boletus suaveolens, frogs, vipers, snails, oysters, chocolate, large doses of sugar, wine, &c., have all been lauded in their turns, although principally as articles of diet fitted to this disease. Electricity, cicuta, aconite, digitalis, ipecacuanha, and hydrocyanic acid, have been also successively extolled ; but practical men have

unfortunately experienced that their beneficial effects, if any, are rarely more than temporary.

Gymnastics, if not carried to excess, are excellent prophylactics, especially rowing and riding on horseback.

In the very early stage of phthisis, when it is yet incipient, or when the patient manifests a tendency to the disease, I believe a long journey, by such easy stages as the patient may not be fatigued, is of the greatest advantage; and I always recommend patients to travel whose means permit it. Sea voyages also frequently produce excellent effects. There is no doubt, too, that sojourning in a warm climate during the winter season is of great utility; I will not say that it cures the disease, but it often protracts life. I speak here only of the earlier periods of the affection; for I firmly believe that when a tubercular excavation is once formed, no means have yet been discovered which afford the patient any thing but palliation.

The object you should have in view in relation to climate, is to choose a locality where the winter season is temperate, and subject to the least vicissitude. The late Dr. Young observed, that the mean temperature, from October to March, was, from the year 1790 to 1794, as follows:—At London 43 deg. 5 min.; Penzance, 48 deg. 1 min.; Lisbon, 55 deg. 1 min.; Madeira, 63 deg. In this point of view, therefore, Madeira is the most preferable spot. There is the least vicissitude in the weather in England, at Undercliff, in the Isle of Wight, Torquay, and Penzance; and abroad, at Lisbon, Madeira, Hyères, Nice, in some parts of Italy, and at Montpellier. I refer you for an elaborate account

of this subject to Dr. Clarke's excellent work upon Climates.

My plan of treating phthisis is generally as follows :—

If I meet with an individual in the earliest stage of the disease, or in whom only a predisposition to it exists, I place him upon a very moderate animal diet, allowing him no wine or stimulants in any form ; I permit moderate exercise, as walking, riding on horseback, or sailing. As the month of September advances, I advise him to change his position entirely, and to winter in a warm climate ; if in England, at Undercliff, Torquay, or in some sheltered valley in the south. If his circumstances permit, let the change be still more complete, by sending him to Madeira, Lisbon, Hyères, or Nice. If the means of the patient do not allow him to effect this, I then recommend him to be confined during the winter months in chambers in which the temperature should be regulated according to his feelings.

At this early period of the disease, if the dyspnoea and cough be troublesome, and the pulse somewhat full and hard, I abstract a few ounces of blood occasionally ; but this, gentlemen, should not be too often repeated, as you should preserve the strength of your patient as much as possible. The digitalis, under such circumstances, is of use ; blisters, or the tartar emetic ointment, may now be employed. I commonly administer a combination of ipecacuanha, squills, and hyoscyamus, as an expectorant, and endeavour to alleviate the cough by emulsions and other demulcents.

You will often, very often, find, that such is the

weak state of the patient, even from the beginning, that he will bear no depletion. In those cases, support him with a bland and non-stimulant diet, — as milk, the white meats, jellies, &c. ; and you may administer the ordinary tonics, combining them with the acids, if there be night sweats and no diarrhœa.

When the tubercles soften, and the excavations commence forming, I should recommend you, gentlemen, not to send your patients abroad : it rarely, if ever, is of any service, but is often highly disadvantageous ; for they are removed from the comforts and attentions of home at the moment they are most required, and they too often, unfortunately, expire in a foreign land, without a friend to afford the last consolations.

As the disease advances, the remedial means diminish daily in their effects : the distressing symptoms alone can be attended to. Thus the cough may be somewhat allayed by demulcents, — as emulsions, the lichen islandicus, the Irish moss, &c. : the two latter also serve as articles of diet, Opium produces often the greatest relief in procuring sleep and abating the cough. The hyoscyamus and belladonna have been used with the same intentions, but rarely with such good effects.

I am satisfied that it is injudicious to permit the diarrhœa to continue at any period of the disease, since it produces great exhaustion. It often yields to the ordinary chalk mixture. I sometimes use small doses of the sulphate of copper, combined with opium, if the relaxation of the bowels be very obstinate.

A gargle, composed of borax, honey, tincture of

myrrh, and infusion of roses, often relieves the aphthous state of the mouth, tongue, and fauces.

I recommend the inhalation of chlorine gas if the concomitant catarrh be extensive and considerable; and, I think, with occasional advantage.

Such is the general plan by which phthisis may be *palliated*; but I repeat again, that I believe the disease not to be *curable* by any means proposed up to the present time.

LECTURE XVII.

DISEASES OF THE PARENCHYMATOUS STRUCTURE
OF THE LUNGS.ADVENTITIOUS DEPOSITS — *continued.*

Cartilaginous Deposits. — *Ossific Deposits* — imperfect, cretaceous. — Causes. — *Cysts* — serous, mucous, composed, or compound. — *Hydatids.* — *Medullary Sarcoma.* — Generalities. — Varieties — first, encysted; second, non-encysted; third, Infiltration. — *Melanosis.* — Generalities. — Varieties — first, encysted; second, non-encysted; third, infiltrated. — *Black Pulmonary Matter.* — Characters. — Differences between it and Melanosis.

I HAVE thus concluded the description of the tubercular adventitious deposit, and have directed much of your attention to that subject, in consequence of its great importance. We now proceed to the other species of accidental formations which occur in the lungs: they will occupy much less of our time, because of their greater rarity, of their signs being scarcely known, and their mode of treatment being very imperfectly understood.

You will recollect I divided the adventitious deposits into two classes — the one of inorganic, the other of organic substances. Tubercles, and the formation I am next to describe, are included in the first class; the second, or organic, contains cysts, hydatids, medullary sarcoma, and melanosis.

Cartilaginous and Osseous Adventitious Deposits.

Cartilage.—We have already spoken of the cartilaginous nature of the tubercular cicatrix, of the semi-cartilaginous structure of the lining of fistulous tubercular excavations, and of the formation of cysts of a similar character, occasionally enveloping tubercular matter. There are also found cysts of an analogous structure occasionally investing the adventitious deposits, which we shall presently describe; and, finally, portions of cartilage, of irregular forms and of moderate volume, are sometimes seen in the lung, presenting here and there points of ossification.

Osseous Deposit.—Accidental ossific deposits never present the same physical appearances, or proportion of chemical elements, as ordinary bone: no fibrous or cellular texture is seen within them, and the quantity of earthy matter is always greater in these formations than in healthy bone. The gelatine in them is sometimes so slight, that these concretions are like small stones, from which they have been called *calculous* or *tophaceous*; sometimes even there appears to be no admixture whatever of gelatine, so that the deposit is like half-dried chalk or mortar: hence Laënnec has denominated the first kind *imperfect ossification*—the second, *cretaceous concretion*.

Imperfect ossifications are found encysted or non-encysted. The first rarely occur in the lungs; they vary in size, from a pin's head to a nut, and adhere strongly to their cysts. The cysts are of a semi-cartilaginous structure.

The non-encysted are very irregular in their forms: their surfaces are uneven and rough; their centres white and opaque, and easily reducible into powder by a hammer; their external parts are of a yellowish colour, slightly transparent, as if horny, and are broken down with difficulty.

These ossifications are occasionally found in the pulmonary tissue, or in the midst of a cartilaginous mass, as of a tubercular cicatrix; they are found, also, in tubercles, particularly those developed in the bronchial glands. When a mass of tubercles containing these petrous substances soften, the concretion will remain free in the excavation until a bronchial orifice of sufficient size opens into the cavity to admit of its expectoration.

Cretaceous Concretions are of much more common occurrence: they present the appearance of chalk slightly wetted, or they are mixed with so much fluid as to render them like mortar more or less thick: in the latter case they are always encysted; in the former most frequently, but not always. When this matter is pressed between the fingers, it appears to consist of an impalpable powder; but frequently it contains small fragments of a firmer texture, giving the sensation of a mixture of small grains of sand with chalk more or less wetted.

Their cysts are usually cartilaginous; they are spheroid, or irregular in form. Laënnec saw one shaped like a pyramid of four sides. The rounded cysts are sometimes themselves imperfectly ossified, and present the characters of the external surface of the stony concretions previously described; that is, they are very hard, and slightly transparent, like

horn. Occasionally several concentric cysts are seen enveloping each other, either of a bony or cartilaginous structure: they are usually separated by layers of cretaceous matter.

A half liquid cretaceous substance is often found in the centres of tubercles, and particularly in those contained in the bronchial glands. This matter may easily be distinguished from the tubercle by its opacity and whiteness; and if it be dried, it becomes still whiter, and acquires more cohesion.

Laënnec observes, that these concretions are never larger than an almond: you perceive, however, one on the table larger than a walnut. This matter is often met with in filtrating the portion of a lung around a tubercular cicatrix.

Causes. — Cullen and others have attributed these formations to the inhalation of certain powders; and they state that starch-makers lapidaries, stone-masons, coachmen, &c., are most subject to them. A very simple and convincing answer may be given to this opinion; which is, that then the chemical composition of these concretions should be as various as the different kinds of powders which persons in these occupations inhale; but their chemical nature is always the same: they are always composed of *phosphate of lime*. Were any other proof of the incorrectness of this notion necessary, it might be inferred that these powders would remain and accumulate in the bronchial tubes; this, however, never occurs: the dust we inhale daily becomes entangled with the tracheal and bronchial mucus, and is expectorated.

Laënnec believed that, in the greater number of cases, these ossific substances were the result of

nature's efforts, who, in cicatrizing pulmonary excavations, has deposited in too great quantity the calcareous phosphate necessary to the formation of the accidental cartilages which constitute pulmonary fistulæ and cicatrices.

This explanation is insufficient, as he himself partly admits, inasmuch as cretaceous matter may exist in the centre of a tubercle before it has softened; and it may be found, also, disseminated in various parts of a lung otherwise healthy; in neither of which cases has there been a semblance of previous cartilaginous formation: nor is the phosphate of lime necessary to the accidental deposit of cartilage, although it be frequently superadded to it.

The occasional expectoration of cretaceous matter is the only sign we possess of the probable presence of a still greater quantity in the lungs.

Cysts.

By a cyst is meant an accidental membrane forming a sac without an opening, usually round, but sometimes irregular in form, and containing a matter more or less liquid, secreted by the membrane forming the cyst.

The cysts containing liquid matters vary as to the nature of the membrane or membranes which compose them: thus, they may be formed of a mere serous membrane, like the pleura or peritoneum; they are then thin and transparent, appearing like a vesicle sometimes not larger than a pin's head; at others of the size of a walnut. These are called *serous cysts*. I have never met with them in the

lungs, but generally in the kidneys. Occasionally the cysts are formed of a thin membrane analogous to a mucous membrane: these are often imperfectly surrounded by a layer of fibrous matter, or condensed cellular tissue, uniting them to the neighbouring parts: they may be called *mucous cysts*.

Sometimes the cysts containing liquid matters are formed of fibrous and condensed cellular tissue, enveloped more or less extensively by a cartilaginous layer, which is itself ossified at many points. The internal surface of these cysts is unequal, rough, and lined here and there by a semi-concrete albuminous or fibrinous substance. They are called *composed cysts*.

These cysts are the rarest of all the accidental deposits in the lungs of man: the serous forms are not infrequent in those of oxen and sheep. Laennec met with but a single instance of a composed cyst in the human lung, and that, probably, had contained an hydatid: it was situated in the right inferior lobe, large as an apple, and of very irregular form. Its parietes were from two to four lines thick, lined internally by a fibrinous or albuminous substance of a whitish-yellow colour, and similar in appearance to the middle coat of an artery; its external surface was of a perfectly fibrous texture, like to that of a tendon; here and there it had somewhat the structure of cartilage, in which there were many osseous points placed in various directions, some of which penetrated naked into the cyst, others plunged into the pulmonary tissue, which was, however, protected by a thick fibrous

layer covering the projecting spiculæ of bone. The cyst contained a liquid yellowish matter.

We have no specific signs of the presence of these accidental formations.

There are also cysts containing solid matters: I have described two species enveloping tubercular and cretaceous deposits; I shall speak of the rest in describing the substances they enclose.

Hydatids, or Acephalocysts.

These worms have long been confounded with cysts; they consist of a simple bladder of an ovoid or spheroid form, soft, and of the appearance of half-boiled white of egg; their parietes are usually uniform, although sometimes of unequal thickness; they are diaphanous, or semi-transparent; colourless, or of a milky tint, or slightly red, yellow, green, or gray.

The cavity of the hydatid always contains a fluid, which is usually limpid, but sometimes a little yellow or red. Frequently small hydatids are attached, like buds, to its internal parietes: these buds may be seen of various sizes, from a pin's head to a completely formed hydatid. When they have arrived at the latter state they fall off. A single hydatid may contain a considerable number of others in various degrees of development. This mother hydatid, as it has been called, sometimes acquires an enormous size: I have seen it of a capacity equal to three gallons.

Rudolphi has denied that the hydatid is an animal, from the extreme simplicity of its organis-

ation; Laënnec asserts that they are organised beings; Baron Percy, and my friend Mr. Langstaff, the latter of whom has paid great attention to the subject, declare that they have seen their contractions.

Hydatids are always contained in cysts, by which they are separated from the surrounding parts. The cysts are usually fibrous, although cartilaginous and osseous matters often enter into their composition. Their internal surface is commonly unequal and rough: most frequently it is lined by an opaque, semi-concreted, yellowish albuminous matter.

A single cyst may contain a number of hydatids; they are then found floating in a fluid, either limpid or troubled, yellow or red. If it contain but one hydatid, it completely fills its cavity.

The natural cure of the hydatid takes place from its spontaneous destruction; it then bursts, its fluid escapes into its cyst and becomes absorbed, and the cyst itself, no longer subjected to distention by the increase of the hydatid, gradually contracts, and it is probably ultimately obliterated by the adhesion of its parietes to each other.

Hydatids may develop in many of the organs; and, although they are rarely seen in the lungs, yet authors have occasionally described them situated there.

The signs of the presence of hydatids in the lungs are extremely obscure: they would be the same when arrived at a considerable volume as those of effusion into the chest; and it would be impossible to distinguish the two diseases. Hydatids have been expectorated: in that case there

would be no difficulty; and the previous absence of the respiratory murmur, and the dulness on percussion, would indicate their original situation. The result of the treatment of this disease is as uncertain as its signs: Laënnec imagined, that since sheep, who are so frequently affected by certain species of these worms, producing in them the *rot*, are cured by salt pasturages, that salt might be used with advantage in these cases by man.

Medullary Sarcoma.

This deposit has received a variety of names, as spongoid inflammation, fungus hæmatodes, soft cancer, cerebriform cancer, fungoid disease, encephaloïde, cephaloma.

Medullary sarcoma was first particularly described by Burns, of Glasgow, in 1800; since which period several have given their views of the disease, as Abernethy, Monro, Hey, Sir Astley Cooper, Mr. Wardrop, Laënnec, Cruveilhier, Andral, and Dr. Carswell. The last author, to whom the medical public is also so much indebted for his splendid plates on morbid anatomy, has, I fear, generalised incorrectly in including in the same genus the hard and almost inorganic scirrhus with the soft and highly organised medullary sarcoma. I have carefully examined the different authors upon this subject, and find that the description given by Laënnec accords best with what I have seen, and what I shall this evening show you.

From the resemblance which this deposit has to the cerebral substance, Laënnec has denominated it *encephaloïde*. He describes it as existing under

three forms: — 1. encysted; 2. non-encysted; 3. infiltrated into the tissue of the organs.

In whatever mode this matter is primarily deposited, when it is fully developed it is homogeneous, and very similar to the substance of the brain: it is here and there slightly tinted with a rose colour. Cut in fine slices, it has a slight semi-transparency, but is opaque in the mass. Its consistence is similar to that of brain, but is less supple, and breaks more easily. When the deposit has acquired a certain volume, trunks of blood-vessels run along its fissures, and from thence penetrate in ramifications into its substance. The coats of these vessels are thin, and often break; clots of coagulated blood are then extravasated into the medullary mass, presenting a striking analogy with the appearances found in the brain of persons destroyed by sanguineous apoplexy: these extravasations are sometimes so great as to destroy nearly the whole of the adventitious mass, a few portions of its peculiar substance being only left. When this occurrence happens near the surface, and the skin inflames and ruptures, a bleeding fungoid tumour appears, to which the name of *fungus hæmatodes* has been applied. This state has been confounded with tumours formed by accidental erectile productions bursting through the skin.

The mass now softens, and soon arrives at the consistence of a thick paste, and then of thick pus, still preserving its white or rosy tint. When, however, it contains extravasated blood, it becomes of a blackish-red colour; the blood soon decomposes, its fibrine concretes and combines with the medullary matter, while the serous part is absorbed. These mixed matters are yet, however, of a deep

black red, and present the appearance of a slightly dried and friable paste. Usually some portions of the cerebriform substance still remain, by which the nature of the original disease may be detected. Frequently, too, masses of medullary sarcoma may be found at the same time in different parts of the body, in various stages of destruction.

We now proceed to describe the three varieties in which the deposit is seen.

Variety 1.: Encysted. — The encysted medullary sarcoma varies in size from that of a nut to a middle-sized apple.

The parietes of the cyst have a tolerably uniform thickness, of about half a line; their colour is grayish-white, or milky, and they are more or less transparent, according to their thickness; they are of a cartilaginous texture, although of greater softness, and less brittleness, than cartilage in general.

The cerebriform matter adheres but slightly to the cyst; it is separated into lobules by a delicate and fine cellular tissue, analogous to the pia mater. A number of vessels ramify upon this membrane, and penetrate into the interior of the mass. The coats of these vessels are very thin, easily break, and the effusions of blood I have already spoken of occur. Sometimes the trunks of these vessels, while placed in the fissures separating the lobules, break, and the blood accumulates beneath the cellular membrane, and forms a clot between the membrane and the medullary mass.

It is in this early state that these tumours present the lobular form: the lobes are principally seen upon the surface, and then are very similar to the convolutions of the brain. To detect them, the cyst must first be dissected off.

At this period the medullary mass is firm : if cut in fine slices, it is slightly semi-transparent ; its colour is pearly gray, or yellowish. If the tumour be cut into, it appears to consist of a much greater number of lobules than its external form would indicate ; these smaller lobules are closely applied to each other leaving no intervals ; their separation is only expressed by red lines of injected cellular tissue. These lines rarely intersect each other, but are arranged in irregular curves.

As the tumour softens, it presents the greatest analogy to the cerebral substance ; its texture is now homogeneous ; there are no traces of the internal lobules, although the external are perfectly distinct ; the vessels on the surface are much larger, and it is at this period that their rupture takes place.

Finally, the tumour becomes still softer ; it is now like the brain, in a very humid and pultaceous state. Laënnec continues to observe that he never saw the encysted or non-encysted cerebriiform tumour soften much more, or its matter to be absorbed or evacuated, so as to leave an excavation.

This variety is commonly found in the lungs, liver, and mediastinum.

Variety 2.: Non-encysted. — The medullary deposits often appear in this form ; their size is extremely variable, existing as a mere point to the volume of a full-sized foetal head. Their forms are spheroid, ovoid, flattened, or altogether irregular. Their external surface is divided into lobes, although less regularly than in the encysted species. A cellular membrane invests them ; and if they be situated where that tissue is abundant, it is very distinct ; if placed in the interior of the organs of

the body, the cellular envelope is then scarcely apparent.

The non-encysted masses present, at first, a greater degree of semi-transparency than afterwards; they are almost colourless, or of a slight blue tinge; they are firm, and divided into many lobules; they have somewhat the aspect of fat, but do not grease the scalpel; sometimes they appear rather humid than fatty. After this period, the substance of the tumour becomes opaque and whiter; most of the lines separating the internal lobules disappear; the larger external lobes preserve their primitive texture a longer time, so that they may still be firm, while the other parts of the same tumour may be completely softened.

These tumours may be developed in all the organs of the body, but they are particularly found where the cellular tissue is lax and abundant.

Variety 3. : Infiltration. — Laënnec never saw this variety exist in the lungs. It consists in the molecular deposit of medullary matter into the tissue of an organ; at first the points of its formation are small, and irregularly disseminated; these points gradually enlarge, fresh ones accumulate between them, until at last the parenchymatous structure composing the viscus is more or less transformed into a medullary mass. The colour of this mass varies according to the colour of the organ in which the infiltration occurs; thus in the liver, where it is frequently seen, it is white, or of a whitish-yellow tint. The texture of the affected organ is at first somewhat firm, but it finally degenerates into a soft and pappy substance.

There are no specific symptoms by which this disease can be determined to exist in the lungs.

Melanosis.

This very peculiar accidental deposit has been successively described by Laënnec, Breschet, Langstaff, Cullen, Carswell, and Fawdington.

In its first state, or, as Laënnec calls it, the state of *crudity*, the melanotic deposit has the consistence of a lymphatic gland; its tissue is homogeneous, its colour deep black: as it softens, there may be pressed from it a ruddy thin liquid, mixed with grains, sometimes firm, sometimes friable; finally, these grains, and the whole mass of the tumour, break down, and are converted into a kind of black "bouillie."

Melanosis may exist in three forms: 1. encysted; 2. non-encysted; 3. infiltrated into the substance of an organ.

Variety 1.: Encysted. — The cysts are generally round; they vary in size, from that of a nut to a walnut; their thickness is equal, and generally of about half a line, and they appear to be formed of cellular substance alone. They adhere by a fine cellular tissue to the organ in which they are developed, and may be easily separated from it by dissection. The internal surface of the cyst is smooth, but the melanotic matter adheres closely to it; the means by which this union is effected is a fine cellular structure, which disappears as the mass softens.

This variety has been principally found in the liver and the lung.

Variety 2.: Non-encysted. — This has been seen in almost all the organs of the body.

The volume of the non-encysted masses varies from the size of a millet-seed to that of an egg, or

they may even become larger. Their form is irregular; they adhere strongly to the parts in which they are contained, although they are sometimes united to them by a delicate cellular tissue which permits them to be detached. In the latter case they are generally of a rounded form.

Variety 3. : Infiltrated. — Sometimes the melanotic matter is deposited in points between the integral molecules of an organ. The appearance of the matter, then, varies according to the colour of the organ in which they are placed, and according to the state of crudity or softening of the melanotic deposit.

In the first instance, the organ is dotted here and there with small black spots, arranged irregularly, or in striated forms; as these spots enlarge, they unite to constitute masses of various size and figure. The natural structure of the viscus in which they are placed gradually diminishes, and at last disappears entirely, leaving a perfectly black substance in its place. It is at this period that the adventitious deposit usually commences to soften: if it even should occur before, the natural substance of the organ begins to soften also, and in both cases is destroyed.

Too little is yet known of the special signs of this disease, to enable us to distinguish it when in the lungs.

Black Pulmonary Matter. — The lungs and bronchial glands are often tinted with a black colour; this is called the black pulmonary matter, and may easily be mistaken for melanosis; and, indeed, is often very difficult to distinguish from it. Laënnec has established the following differences:—

Softened melanotic matter tinges the fingers of

a black colour, which is easily washed off; but black pulmonary matter, if allowed to dry previously, will remain for many days, even with frequent washings. Black bronchial glands, according to Fourcroy, contain a large quantity of hydrogen and carbon, which are not found in melanosis; the latter being composed almost entirely of albumen and a peculiar black colouring matter.

Melanosis is evidently a deleterious formation; black pulmonary matter is not: the first produces malignant symptoms; the second is innocuous.

The black pulmonary matter effects no alteration in the structure of the lung, except its colour. When the melanotic matter is deposited in a slight degree, it may produce no other change also. Laënnec then gives us the following rules by which we may distinguish them:—

We ought not to admit the existence of melanosis in the lungs, except we meet with masses of a certain nature and volume, already softened, and so placed and formed, that they cannot be mistaken for bronchial glands.

We ought not to admit that the lung is infiltrated with melanotic matter, unless the tissue of the organ be as dense and hard as that of the liver; but when this density has a doughy character, and the hardness is caused by osseous or cartilaginous points, the colour should be considered as produced by black pulmonary matter.

I have now, gentlemen, concluded the diseases of the parenchymatous substances of the lungs, and shall proceed to those of the third section, or diseases of the pleura.

LECTURE XVIII.

ON DISEASES OF THE LUNGS.

SECTION III. — DISEASES OF THE PLEURA.

PLEURITIS.

First, Acute. — Second, Chronic or latent. — Third, Gangrenous. — *Morbid Anatomy of Acute Pleuritis.* — First Stage — *Redness* — uniform — spotted — Guersent's Experiment — arborescent — Extent of Redness. — Second Stage — *Effusion* — Colour — Hæmorrhagic Pleurisy — Transparency — Quantity. — Effects of Pressure of the Fluid on the surrounding Parts. — Old Adhesions — Effects of partial or circumscribed Pleurisy. — Effects in displacing the Heart, on the Diaphragm, on the Intercostal Spaces and Ribs — General Dilatation of the Side of the Chest affected. — Third Stage — Formation of *False Membranes* — Change of Characters of effused Fluid — Deposit of Albumen to form False Membrane — its Colour, Appearance, Thickness. — Relative Proportion of Serum to False Membrane. — Fourth Stage — Conversion of False Membrane into *Cellular Tissue, or Fibro-Cartilage* — Absorption of Fluid — Expansion of Lung — Organisation of Albuminous Bands and False Membranes into *Cellular Tissue* — Mode of Formation of *Fibro-Cartilage* — Empyema. — *Morbid Anatomy of Chronic Pleurisy* — When to be called Chronic. — Appearances of the Effusion of False Membranes. — *Empyema of Necessity.* — *Morbid Anatomy of Gangrenous Pleurisy* — combined with Gangrene of the Lung — and Serous Effusion. — Theory of Formation of False Membranes.

THE pleura is a fine serous membrane investing the lungs, and reflected over the diaphragm and the inner surfaces of the ribs. It secretes a fluid, for the purpose of lubricating the parts, and allowing their free play upon each other. Almost all the diseases of this membrane have for their

results an alteration in the quantity and quality of this fluid. We shall consider the diseases of the pleura in the following order: —

Pleuritis.

Hydrothorax.

Pneumothorax.

Pneumothorax, with effusion of fluid.

Adventitious deposits.

PLEURITIS.

By *Pleuritis* or *Pleurisy*, we mean an inflammation of the pleura. Laënnec has given several minute divisions of this disease, which I think unnecessary, as they all may be included in the three following forms: —

Pleurisy, { 1. Acute.
2. Chronic or latent.
3. Gangrenous.

1. *Acute Pleurisy.*

We shall proceed in our usual order, by first describing the morbid anatomy of the parts affected.

Morbid Anatomy. — The inflamed pleura presents in the first instance a red colour: after a short time, probably only a few hours, an effusion takes place from its surface, varying exceedingly in its quantity: to this effusion succeeds a deposition of coagulable lymph, which forms pseudo-membranes covering the inflamed parts: these false membranes finally unite, become organised, and converted into cellular membrane, or fibro-cartilage. We shall consider these conditions, as indicating different

stages of the disease, in precisely the order described: viz. —

1. Redness.
2. Effusion of fluid.
3. Formation of false membrane.
4. Conversion of false membrane into
Cellular membrane.
Fibro-cartilage.

First Stage — Redness. — In the first stage of pleurisy, the inflamed membrane is of a red colour, sometimes appearing uniform and diffused in its tint, at others in spots of various sizes. This last appearance is probably a post-mortem result, for there is every reason to suppose that the redness is uniform in the living subject, and that the absence of colour between these spots or stains arises from the partial transudation of the blood through the vessels after death. Thus we see that in persons dying of erysipelas on the surface of the skin, the blood transudes the vessels of the inflamed parts, and the skin returns almost to its natural colour, leaving a few spots only here and there. Guersent injected stimulant liquids into the serous membranes of living animals, and found that the redness produced by their inflammation was uniform, and not spotted. The red colour penetrates through the membrane.

Besides this diffused coloration, vessels are seen ramifying in an arborescent form, in the same manner as upon the conjunctiva, when that membrane is inflamed. The extent of the redness varies considerably; it may occur only in a small point, it may occupy the whole of the pleura on one side of

the chest, but it very seldom affects both sides at the same time.

Some authors have thought that the pleura becomes thickened during this stage, but a careful examination will shew that it is not.

Second Stage — Effusion. — It is very probable that at the commencement of the red stage, the pleura presents a dry surface or nearly so, because we have then no signs of effusion; the secretion commences in a few hours.

The effusion consists of a citrine, straw, or yellow-coloured fluid; it is sometimes slightly reddened, from the admixture of a little blood, small clots of which may be occasionally seen floating in the liquid. When this accidental coloration takes place, Laënnec has called the disease *Hæmorrhagic Pleurisy*. The fluid is at first limpid, and inodorous, and varies in quantity from an ounce or two to seven or eight pints.

It may readily be conceived that this effusion of fluid must produce certain mechanical effects upon the parts within which it is contained. It is of the highest importance that we should be well acquainted with these effects, as they afford the most valuable signs to direct us in the diagnosis and treatment of this stage of the disease.

The pleura bounds the cavity containing the effused secretion; that membrane invests the lungs and pericardium, covers the upper surface of the diaphragm and the inner surface of the ribs and intercostal muscles. The effect of the accumulated fluid must be to compress these parts, and increase their distance from each other. Let us examine these results.

As the fluid is secreted, although a portion of it

gravitates to the lower part of the pleuritic cavity, a considerable quantity spreads over the whole surface of the lung, and compresses it, and in proportion to its accumulation, so the lung becomes diminished in size, until at last it is reduced to so small a volume, that it is not larger than the closed hand: the pressure of the fluid upon the organ is directed from without to within, from before backwards, and from below upwards: the lung is flattened against the vertebral column, and, in consequence of its compression, becomes denser and less crepitant than usual; it contains but little air, its blood-vessels are flattened and empty, and the bronchi are contracted: the pulmonary tissue is, however, perfectly distinguishable, and if air be forced down the trachea, the viscus may be completely inflated, and restored to its original size.

Old adhesions modify the form and situation of the lung when compressed. If these adhesions have existed between the upper lobe and its corresponding costal pleura, the organ can only be pressed from below upwards, and it then occupies the superior part of the pleuritic cavity, while the fluid is accumulated beneath it. If old adhesions have formed between the lower surface of the organ and the diaphragm, the reverse takes place; the pressure of the fluid is from above downwards; the lung occupies the lower part of the chest; and the fluid is placed above it; finally, when there are adhesions between the outer surface of the lungs and the costal pleura, the pressure is directed from within outwards, the organ is flattened against the ribs, and the fluid collects nearer to the median line of the chest.

If the previous adhesions are so situated as to

isolate the effusion in a diminished space, the disease has been called *partial or circumscribed pleurisy*.

The heart becomes also displaced if the fluid be considerable in quantity: thus, if the effusion be situated on the left side of the chest, that organ is pressed towards the right; and its beatings can no longer be felt or heard in his natural position. If the accumulation be in the right side, the heart is pressed still farther to the left.

The pressure of the fluid upon the diaphragm forces it to descend, but that effect is inconsiderable on the right side, in consequence of the support which the liver affords to this muscle. In extreme cases, the compression may be sufficient to force the edge of the liver below the margins of the cartilages of the false ribs, but I have rarely seen it. If the left side of the diaphragm be compressed, the stomach is pushed before it, and a diffused and irregular swelling becomes very distinct below the cartilages of the false ribs on that side.

We must now consider the effect of the pressure of the fluid upon the intercostal muscles and ribs. The internal surfaces of these muscles are forced outwards, and are brought to a level with the external surfaces of the ribs, so that the intercostal spaces are altogether effaced, and the whole side of the chest is rendered perfectly smooth and even; nay, some have said that these spaces have been seen projecting outwards: I have often sought for, but have never met with, this appearance. The pressure of the fluid forces the ribs directly outwards, by which the whole of the affected side becomes enlarged, and sometimes to a considerable degree; so much so, that in measuring and com-

paring the two sides of the chest, I have seen a difference of two inches. This difference of size affords us a most important sign of the disease.

Third Stage—Formation of False Membranes.
—The effused fluid, which in the first instance was transparent and of a straw colour, now changes its characters, although the precise time at which it does so has not been discovered, and probably varies in different individuals. Small particles of white and opaque albumen, like curds of milk, are now seen floating in the secretion; these particles enlarge, unite, and form masses of various sizes up to that of an egg; sometimes they present the appearance of fresh mesenteric fat. The colour of the fluid itself also changes; it becomes milky; the floating albuminous matter breaks down into small fragments, and mixes with it, so as to give the whole the appearance of flour diffused in water; finally the comminution of the albumen becomes still more complete, and the whole effusion then presents a puriform character.

As these changes take place, the albuminous matter gradually precipitates and adheres to the surface of the pleura, forming a false membrane upon it, to an extent of surface proportionate to the extent of the inflammation. This false membrane is at first of the consistence of thick pus, or the curds of milk, then of boiled white of egg, or of the inflammatory crust of the blood; and, finally, it acquires the cohesion of the elastic coat of an artery. Its colour is whitish-yellow, and, according to its thickness, it varies from semi-transparency to opacity. Its thickness, at the period of its primary formation, is inconsiderable, but gradually increases, so that it has been seen to measure two or three

lines : its surface is occasionally smooth, although more frequently irregular. It would seem that after the membrane had been formed, fresh albuminous matter had been deposited upon it in irregular and intersecting lines, giving it a reticulated appearance ; these lines are often so numerous, and cross each other so frequently, that the points of intersection become so close and elevated as to appear like granulations, and the surface has been mistaken for an ulcer.

The relative proportions of false membrane and of fluid vary exceedingly ; in some instances the effusion is considerable, and the false membranes are but thin, and slightly extended ; in other cases, the membrane exists alone, and there is scarcely any serous exudation ; the last case being considered as a variety, under the name of *dry pleurisy*. It may be established as a general rule, that the greater the power of absorption, the less is the quantity of serum. Thus, in individuals of firm fibre, or in children, the activity of whose functions is great, there is very often but little serum, compared to the false membrane ; while in the aged and in those of lymphatic temperaments, the latter is generally inconsiderable.

It may also be observed here, that, usually, in proportion to the limpid and transparent state of the effused fluid, the false membranes are thin, but as the serum becomes turbid and similar to pus, they gradually grow thicker, and of greater density and firmness.

Conversion of False Membranes into Cellular Tissue, or Fibro-Cartilage.

Cellular Tissue. — The effusion being accomplished, and the false membrane formed, nature endeavours to effect a cure by absorbing the thinner matter. In proportion to the activity of the absorption the fluid gradually diminishes, and the compressed lung expands. The degree, however, to which that organ can dilate, not only depends upon the diminution of the fluid, but also upon the thickness and density of the false membrane which has formed upon it, and by which it is bound down to its anormal situation. If the false membrane be very thin, the lung can freely dilate until it arrives at or very near the costal pleura: in that case, bands of albuminous matter cross through the remaining fluid, nearly at right angles from the false membrane lining the costal pleura to that which covers the pulmonary pleura: these bands are at first of a yellowish-white colour, and opaque, sometimes round, sometimes irregularly angular, and fimbriated at their edges; they vary in thickness, not only as compared to each other, but in the different parts of their own extent; thus they may be thickest at their costal or pulmonary extremities, or at their centres: they are in the beginning very soft, so that you may pass the finger between the two pleuræ, and rupture a series of them with the greatest facility. These bands gradually increase in number, encroaching constantly upon the space previously occupied by the effusion.

These bands, and the pseudo-membranes to which they are attached at their extremities, gra-

dually become organised : an irregular streak of blood, arising apparently from the healthy pleura, which membrane is spotted with red at the corresponding point, penetrates the band, and soon assumes a cylindrical form of a certain diameter, and ultimately becomes a vessel. If the rudimental vessel be examined at this period, it is very red, soft, and formed of blood scarcely coagulated. In cutting into it, a round, white, and fibrinous mould may be extracted, formed of concreted fibrine, whose centre is perforated to form a small canal : this fibrinous mould, in becoming thinner, appears to form the parietes of the new vessel.

The false membranes and their bands now become thinner and more transparent, their consistence is yet softer than cellular tissue, and they appear as if injected with fine injection ; finally, they become perfectly thin and transparent, and lose their vascularity ; their firmness is increased, and they cannot be distinguished by their structure from the cellular tissue of any other part of the body. They are subject also to the same physiological and pathological laws as the cellular and serous membranes ; like them they secrete and absorb, and may become the seat of inflammation.

Fibro-cartilage.—But when the false membrane is thick and firm, the lung is not only bound down by it, but the function of absorption is very imperfectly carried on through its inorganic texture. The circumstances necessary to the cure of this disease are then extremely unfavourable.

If the patient die in this state, and you examine his chest, you will find the lung reduced to a very

small volume, and covered by a thick, yellow, and opaque false membrane. The cavity of the pleura is filled with a turbid fluid, often as if mixed with flour, often also of a completely puriform appearance, constituting that form of pleuritic effusion called *empyema*. Similar bands to these I have already described pass at right angles from one false membrane to another, the only difference being that they are much longer, in consequence of the greater distance now between the pleura pulmonalis and costalis.

It occasionally happens, however, that the absorption of the fluid is effected to a certain degree, and that the false membrane enveloping the compressed lung is not so thick as to prevent its expansion, although it may be sufficiently so as not to allow it to extend to the costal pleura: in that case nature makes an extraordinary and beautiful effort to effect the contact of the opposed false membranes, by forcing the whole of the diseased side of the chest inwards upon the lung, by which that side becomes contracted. Laënnec was the first to discover this singular phenomenon.

Let us now suppose that the thick and apposed false membranes have by this means become in contact: what is their mode of union?

They adhere, and if a transverse section be then made, it will be seen that they form a single solid membrane composed of three layers, the two external being the original membranes, now appearing white and opaque, fibrous or cartilaginous, or of a mixed character, and sometimes even partly osseous. A third and intermediate layer is the means of union; it is semi-transparent, and, as Laënnec

expresses, perfectly similar to the most transparent parts of the intervertebral cartilages.

The *fibro-cartilaginous* membrane thus formed varies in thickness from two lines to half an inch, but it gradually diminishes as the time advances from its original formation.

Laënnec supposed that this species of membrane could only be formed from hæmorrhagic pleurisy, or at least that the presence of blood was necessary in the effused fluid. This opinion is hypothetical, and unsupported by facts.

It is, gentlemen, when the fibro-cartilaginous membrane is completely formed, that the contraction of the chest becomes the greatest. You perceive, in this cast, that the shoulder of the affected side has become depressed; that the inferior angle of the scapula is an inch and a half lower than the opposite; that the intercostal spaces are diminished; and that the last rib approaches closely to the crista of the ileum. The sternum and dorsal vertebræ have deviated from the median line, so that the former bone is inclined over to the right or healthy side, and the spine is curved, the convexity being directed to the same side also: finally, the transverse diameter is so diminished, that the circumference of the diseased half of the chest is less by two inches than that of the healthy.

2. *Chronic Pleurisy.*

We now proceed to the consideration of the morbid anatomy of Chronic Pleurisy.

Pleurisy may be said to be chronic when the signs of the red stage have passed, and those of

effusion have continued for some time : at least, if the fluid be not rapidly absorbed, the false membranes soon form, and then the progress of the disease is usually tedious and prolonged. It sometimes occurs that all the signs of the red stage are absent from the commencement, and the disease assumes a chronic form in its origin.

The morbid anatomy of chronic pleurisy is nearly the same as that which I have described of the acute, the only anatomical difference being that the effused fluid is never seen in the limpid or transparent state.

Laënnec, however, thought that, when the disease commenced in the chronic form, there were some differences in the red coloration, and in the appearance of the effusion and false membranes. He observes, that the pleura is more highly reddened ; that the effused serum is more abundant and less limpid, and has the odour of garlic or gangrene ; that there is a much larger quantity of small albuminous flocculi floating in it, so that they give the fluid the aspect of its mixture with coarse flour. He states also that the false membranes have not the same cohesion ; that they are friable, and easily broken by pressure with the fingers ; and that they sometimes appear to be formed only of very thick pus ; that there is but a slight tendency to the conversion of the pseudo-membrane into cellular tissue ; and that it is in this case the dilatation of the chest is greatest, and the compression of the lung most complete.

But, gentlemen, although these varieties in the appearance of the effused fluid and false membranes do exist, yet they are not necessarily depended

upon the disease having commenced in the chronic form, as Laënnec supposed. I have had many opportunities of observing pleurisy in all its stages, and I can state with certainty, that I have seen the appearances just described frequently consequent to the acute form of the disease also.

It sometimes happens that an abscess forms in the parietes of the chest, in consequence of the internal effusion, and it bursts externally: this constitutes the *empyema of necessity* of the ancient writers. I have seen seven instances of these ruptures, and most of them have occurred at the anterior superior part of the chest.

3. *Gangrenous Pleurisy.*

Morbid Anatomy.—This is a very rare disease, and is usually but slight in extent, although, in the specimen I present you, it involves nearly the whole of the pulmonary pleura. This state is almost always consequent to gangrene of the lung, or to chronic pleurisy.

This affection appears in the form of round or irregular stains upon the surface of the lung, of a brown or black colour, and horribly foetid; the parts are soft, and easily broken down by the fingers: sometimes the gangrene scarcely extends deeper than the pleura; at others, the sub-serous tissue is infiltrated with a greenish or blackish serosity of a gangrenous odour.

The irritation of the gangrenous affection always causes a general inflammation of the pleura: abundant serous effusion then takes place, followed by

false membranes, all presenting a dirty green or dark colour, of the same smell I have already described. Gangrenous eschars have been known to form upon the pleura pulmonalis, which, when they fall, have occasioned a communication between the cavity containing the fluid, and the bronchial tubes, allowing a free expectoration of the secreted matter; or the intercostal spaces may be penetrated, and a copious discharge take place from the orifice; constituting another cause of *empyema of necessity*.

Such, gentlemen, is a general account of the morbid anatomy of the different stages of pleurisy. You have seen that the pleura is at first reddened; that it then throws out a serous fluid, from which an albuminous matter is formed, which becomes converted into cellular tissue, or fibro-cartilage. You have seen, also, variations in the degree of redness of the pleura, in the qualities of the serous secretion, and in the nature of the albuminous and pseudo-membranous deposits.

When the disease commences with pain in the side, and fever, symptoms denoting the red stage, it is denominated *acute pleurisy*; it may already be called *chronic pleurisy*, when the fluid has accumulated, and the albuminous matter has begun to separate; and this state may occur without the previous signs of the *acute*. Finally, a gangrenous state of the pleura occasionally, though fortunately rarely, takes place.

Laënnec, in speaking of the products of inflammation of the pleura, states that the secretions are of two kinds; the one fluid, the serum; the other solid, the false membrane. I doubt exceedingly that the false membrane is a secretion; I believe

it rather to be a deposit from the serum, from the following reasons :—

It must be admitted that the functions of secretion (with a single exception) takes place by means of the orifices of the capillary extremities of the arteries ; but these orifices are so small, that they are beyond microscopic powers to discover. We infer their existence by reasoning, but cannot prove it by demonstration. Is it not almost impossible to conceive that such minute orifices can permit the passage of a solid material? Is it not more likely that they deposit fluids, charged with the specific nutritive matter which the organs in which their vessels ramify require? Do we not see that the phosphate of lime itself is not arranged in amorphous forms in the bones, but in the crystalline? And are not all our notions of crystallisation associated with those of previous solution? If, then, the orifices of the capillary vessels are too small to allow the *matériel* of nutrition to permeate them, except in solution, so must they also be too small to carry an adventitious solid, to be deposited upon the surface of an inflamed serous membrane, except by the solution of that matter also.

But without adverting to arguments derived from the general theory of nutrition, we may adduce others which apply more specifically to the case in question.

It is evident that the first result of the inflammation of a serous membrane is a limpid and transparent secretion, and that the adventitious membrane is the second ; the latter never precedes the former. It is a principle in philosophy, that,

when two or more events succeed each other, *always* in the same order, the first must be the cause of the second, the second of the third, and so on. I believe the solid deposit is effected in the following manner:—The serous effusion is charged with albuminous matter; nature soon commences to absorb the fluid; the thinnest parts of it are the soonest taken up, leaving the thicker matter, or albumen, floating in the remaining mass: this albumen becomes attached to the surrounding inflamed parts, covers them in the form of a thin and soft layer at first, which gradually becomes thicker from successive deposits. Irregular lines of albumen, like those floating in the fluid, are also attached to the false membrane thus formed, and give it a reticular appearance. The lowest part of the pleuritic cavity is covered most thickly, because of the gravitation and entanglement of the albuminous matter, until at last the pseudo-membrane is completely deposited, from separation, and not secretion.

If this be not the mode, why is the false membrane always the thickest at the lower part of the inflamed pleura? Are we to be satisfied with the gratuitous assumption that there the inflammation is always of the greatest intensity?

Why is it, too, that we find the relative quantities of serum and false membrane always proportionate to the activity of the absorbing function? Thus in weak and lymphatic individuals, the serum is great in quantity, and there is scarcely any solid albumen formed; absorption is here inactive, the serum remains, and no separation takes place. In

children, in whom all the functions relative to their organisation are extremely energetic, it often happens that all the serum is absorbed, and false membranes alone remain. From these circumstances, permit me to say, at least, that, if I have not proved my own, I doubt Laënnec's explanation.

LECTURE XIX.

PLEURITIS — *continued.*

SIGNS OF PLEURITIS.

First, or Red Stage. — *Local Signs.* — Pain — Situation of — augmented by deep Respiration, or Coughing. — Pleurodynia. — *Respiratory Murmur.* — *Functional Signs.* — Dyspnœa. — Cough. — *General Signs.* — Inflammatory Fever.

Second, or *Stage of Effusion.* — *Local Signs.* — Increase of the Effusion — Respiratory Murmur. — Œgophony. — Percussion. — Effects of Pressure of Fluid upon the Lungs, the Heart, the Diaphragm, Intercostal Spaces, Ribs. — Measurement of the Chest. — Immobility of Ribs. — Position.

Third, *Formation of Cellular Tissue, or Fibro-Cartilage.* — Decrease of Effusion. — Respiratory Murmur. — “Œgophonia Redux.” — Percussion. — Immobility of Ribs. — Effects upon the Size of the Chest — in the Formation of Cellular Membrane, compared with that of Fibro-Cartilage. — *Functional Signs.* — *General Signs.*

Causes of Pleuritis.

Treatment of Pleuritis. — First, Red Stage. — Second, Stage of Effusion. — Operation of Paracentesis Thoracis.

WE now, gentlemen, proceed to consider the signs of the different stages of pleurisy.

1. *Red, or Acute Stage.*

Local Signs — Pain. — One of the most constant signs of this stage is a violent pain in the side. It occurs generally at the point corresponding to the inflamed part, which is usually along the line of the 6th, 7th, or 8th ribs. Laënnec observes, that this sensation sometimes is felt on the healthy side only. I cannot say that I have ever yet distinctly met with an instance of this

variation. The pain is always augmented by inspiration and coughing, in consequence of the lungs being pressed against the inflamed pleura by these movements.

Rheumatism of the muscles of the chest, or *pleurodynia*, often produces pain similar to that from pleurisy. The diagnosis may easily be determined by desiring the patient to move his arm in various directions: these acts will be rendered difficult if the pain be caused by pleurodynia, but not so if it arise from pleurisy. The inflamed muscles are also tender upon pressure, and there is generally rheumatism in some other part of the body at the same time.

In severe cases of pleurisy, the patient endeavours to breathe with the healthy lung only, in consequence of the pain which the inflation of the organ on the diseased side occasions. Examine the affected side, and you will perceive that the ribs are almost immoveable. The same circumstances occur also in pleurodynia.

Respiratory Murmur.—As the patient rarely fully dilates the lung corresponding to the inflamed pleura, the respiratory murmur in it becomes very obscure and indistinct.

Functional Signs.—*Dyspnœa* is always present, but it varies greatly in its intensity, according to the degree and extent of the inflammation. This sign depends not upon any disease of the lung itself, but upon the pain which a full or even ordinary dilatation of it gives, so that the patient breathes by short inspirations, which must necessarily be frequently repeated to establish the balance between the quantity of air and the quantity of

blood in the lungs. It is in this case that the inspirations are shorter in their duration than the expirations.

The dyspnœa would, of course, be more severe if any disease of the bronchial tubes, or pulmonary structure, were combined with the pleurisy.

Cough is by no means a constant sign of this stage of the disease; if it be present, it is usually slight, dry, and infrequent, except there be catarrhal combination; then there will be an expectoration, more or less abundant, of mucous or pituitous fluids.

General Signs—Fever.—Intense inflammatory fever frequently accompanies this stage; it usually lasts but a few days, and ceases upon the disappearance of the pain in the side. It does not follow, however, because the fever and pain cease, that the disease is cured; for effusion will frequently still continue. The dyspnœa then slowly increases, a slight febrile action reappears, and upon a local examination of the chest, you will find the signs of effusion becoming daily more manifest. The slightest dyspnœa and fever occurring after the apparent cure of acute pleurisy, require your utmost attention.

2. Stage of Effusion.

We shall first describe the signs of the increase and accumulation of the fluid, and secondly of its decrease.

Local Signs.—Increase—Respiratory Murmur.
—As soon as a thin layer of fluid is formed between the pleuræ, the respiratory murmur almost instantly

ceases: its cessation is so sudden, that it would appear that the lung was at once arrested in its movements by a sort of suffocation. The fluid may even afterwards slightly increase in quantity, and the respiration be occasionally heard, although at a distance, appearing as if the organ had accommodated itself to the pressure to a certain degree, so that it could perform its movements, although to a slighter extent than natural. As the fluid increases considerably, the lung becomes so compressed, that very little air enters it, and the sound of the respiratory murmur is then totally lost.

There is, however, a certain point of the chest at which the respiratory murmur is almost always present during this stage; it is between the base of the scapula and the vertebral column. This space corresponds to that occupied by the compressed lung. Whenever, also, the organ is retained at a spot by previous adhesions, there also the murmur may be heard: thus it is sometimes distinct at the summit of the lung, or even at the side of the chest; in the latter case it is circumscribed to a place of inconsiderable dimensions. Occasionally, too, the effusion is so slight as not to reach as high as the upper lobe, to compress it. In that case the sound is there distinct also.

In proportion to the diminution of the respiratory murmur on the diseased side, the necessity for respiration increases in the healthy lung; the functions of the latter become more energetic; its movements quicker; the expansion and contraction of its cells more active, and consequently the respiratory murmur is louder, and assimilates to that of children, from whence it is called *puerile respira-*

tion. The respiratory sounds of the healthy lung are sometimes so intense, that they may be even slightly heard through the accumulated fluid, giving the sensation of their arising from the compressed lung. A little practice will soon, however, correct any error that might arise from this circumstance.

As a total absence of the respiratory murmur occurs also in a hepatised lung, it is necessary to observe, that in the latter case it is never sudden, and is always preceded by crepitation, the sign of pulmonary engorgement.

Ægophony. — When a slight layer of fluid exists between the pleuræ, ægophony becomes its sign. I have entered so largely upon this sign in my lecture on Auscultation, that it is unnecessary to repeat it here; but I refer you to that lecture. I shall, however, state, that ægophony appears when the effusion commences; that it disappears when it becomes abundant; that it may remain for many months when the fluid continues in a small quantity; that it is most complete at the upper edge of the effusion, or where the layer of fluid is thinnest; that it may be accompanied by *bronchophony*; and, finally, when the sound is heard all over the chest, it may be affirmed that the effusion is slight, and uniformly spread all over the lung. In the latter case the respiratory murmur may still be slightly heard, because the layer of fluid is not sufficient to compress the lung, so as to prevent the air penetrating it; and if these latter circumstances continue during the whole period of the disease, you may be certain also that the lung is retained at a small distance from the ribs by cellular bands disposed here and there between their surfaces.

Percussion.—Although the sound on percussion is usually loud in proportion to the quantity of air contained in the lungs, yet in this case, a small quantity of fluid placed between the pleuræ seems as effectually to destroy the natural sound as a larger. Thus it is, that as soon as a thin layer of fluid is formed, indicated by œgophony, percussion gives a perfectly dull and fleshy sound, which, of course, remains during the augmentation of the fluid.

Signs arising from the Pressure of the Fluid upon the surrounding Parts.—You recollect, gentlemen, that when I described the morbid anatomy of this stage of pleuritis, I described also the effects of the pressure of the fluid upon the surrounding parts. We must now attend to the signs of these effects.

The lung is first acted upon, diminished in volume, and pressed against the vertebral column, accounting, therefore, for some of the signs we have just mentioned, as the absence of the respiratory murmur all over the affected side of the chest, excepting the space comprised between the base of the scapula and corresponding vertebræ; accounting, too, for the dulness of sound on percussion. The heart is also pushed from its situation, so that if the effusion exists in the left side of the chest, that viscus is forced over to the right; consequently the sounds of its beatings are often entirely lost at its natural position, and are heard only immediately beneath the sternum and at the right side of that bone. If the fluid be on the right side, the heart is forced beyond its natural position to the left, and the sounds are heard farther in that direction. The fluid then depresses

the diaphragm, but that pressure is resisted on the right side by the liver, so that the latter organ is rarely sufficiently displaced to force its anterior edge below the margins of the cartilages of the false ribs ; but when the left side of the diaphragm is pressed down by the fluid accumulated upon its superior surface, it forces the stomach before it, and a considerable irregular and diffused swelling is produced below the margins of the cartilages of the left false ribs.

In examining the effects of the pressure of the fluid outwards, you will remember that I stated that the internal surfaces of the intercostal muscles were brought to a level with the external surface of the ribs, by which all appearance of intercostal depressions between the ribs is entirely effaced, giving to the whole side a general smoothness singularly contrasting with the healthy side of the chest. The ancients considered œdema of the surface to be a sign of effusion ; but I have no doubt they have confounded that state with the smoothness and fulness I have just mentioned. Be that as it may, I have never seen the skin pit upon pressure in more than two or three instances in above thirty cases. You will recollect, also, gentlemen, that I described the ribs as projecting outwards, whereby the diseased side becomes more or less enlarged : this may be readily detected by the eye. Confirm this sign by measuring the chest as I have directed, and you will find that the difference in the circumferences of the two sides will often be considerable : I have seen it from a quarter of an inch to two inches. These variations, of course, arise from the different quantities of fluid contained.

Immobility of the Ribs.—Another valuable sign of effusion is the immobility of the ribs of the diseased side. The reason of this immobility is obvious: if the lung be so compressed as to receive no atmospheric air, the respiratory functions are not carried on in it, and of course the action of the ribs is unnecessary. If the quantity of fluid be large, you will see that the side affected is perfectly immoveable, forming a striking contrast with the opposite, where the ribs are elevated and depressed with considerable rapidity, in consequence of the respiration being carried on in the healthy lung only.

Position of the Patient.—If the patient place himself upon the healthy side, the fluid compresses the mediastinum and opposite lung, and produces a sense of suffocation. He almost invariably, therefore, places himself upon the diseased side.

Such, gentlemen, are the signs of effusion, upon its increase and utmost accumulation. I will now mention those by which its diminution can be ascertained, and the formation of cellular membrane or fibro-cartilage determined.

Signs of Formation of Cellular Tissue, or Fibro-Cartilage, or of Decrease of Effusion.—As the absorption of the effused fluid takes place, if the lung be not bound down too firmly by a false membrane, that organ gradually dilates, and in proportion to its expansion the respiratory murmur becomes again audible: the sound is at first most distinct where it had never entirely ceased—that is, between the base of the scapula and the corresponding vertebræ. As the fluid recedes from the upper part of the pleuritic cavity, the murmur

is heard at the anterior-superior part of the lung, on the summit of the shoulder, and in the axilla; and, as the secretion still farther diminishes, it becomes distinct successively at the various parts of the chest, from above downwards. If, however, any previous adhesions exist, such as I have already described, the murmur is first heard at the points of the chest corresponding to them.

Let us suppose that the absorption continues: the layer of fluid between the pleura then becomes thinner, and the *œgophony* returns; the *œgophonia redux* of Laënnec. This sign ceases with the total disappearance of the fluid.

The sound on percussion is often a long time before it becomes natural, and in some cases, perhaps, never.

The mobility of the ribs returns in proportion to the degree of expansion of the lung.

All the symptoms I have described are proofs of the absorption of the effused fluid; but the only pathognomonic sign I am aware of, denoting the union of the pleuritic surfaces by cellular tissue, and not by fibro-cartilage, is, that in the first case the chest does not contract, nor do the sternum and vertebræ deviate from their natural position on the median line; while, in the second case, the contraction and deviations are always more or less considerable. The reasons of this difference appear to me to be as follow:—

If the adhesions be of a fibro-cartilaginous character, it is always a proof that the original false membranes were thick, that the lungs were bound down proportionably, and that consequently the freedom of the expansion of that organ was dimi-

nished. It is under these circumstances that nature makes her great effort to approximate the parietes of the chest to the compressed lung; and when that is effected, which it occasionally is, then the union of the two false membranes form fibro-cartilage. As the extent of the contraction is in some degree a measure of the compression of the lung, and of the thickness of the membrane binding it down, so it becomes a sort of measure of the thickness of the united false membranes.

On the other hand, when the lung fully dilates, the compression inwards of the diseased side of the chest is unnecessary; the false membranes then do not exist at all, or are very thin. Thin false membranes do not form fibro-cartilage, but cellular tissue; therefore, when you find that, after the signs of effusion have disappeared, the chest is not contracted, you may pretty certainly infer that the adhesions are of cellular tissue. Look to this subject for yourselves, gentlemen, and you will find your post mortem examinations will bear me out in my assertion.

Functional Signs of Effusion.—The principal functional sign of effusion into the cavity of the pleura, is *dyspnœa*; which is generally of an intensity proportionate to the quantity of fluid accumulated. As, however, the disease becomes chronic, the general emaciation of the body and diminution of the mass of the blood is so great, that a less quantity of that fluid is sent into the lung than in the healthy state, and consequently less air is required; and for this reason the difficulty of breathing is not always in proportion to the quantity of fluid effused. If a catarrh coexist,

the dyspnœa is, of course, increased. A *Cough*, if it be present at all, is usually dry, or the expectoration consists only of a small quantity of pituitous matter.

General Signs of Effusion.—As the effusion becomes chronic, great emaciation is the consequence. I know of no disease in which the body becomes so attenuated, except in phthisis, or after fevers of long duration.

Causes of Pleurisy.

The causes of pleurisy are the same as those of other inflammatory affections. The disease may take place in individuals of all temperaments and ages; those of thin stature and of narrow chests are the most predisposed. Excesses of all kinds favour the action of the external causes, by diminishing the general powers of the system. The external causes are exposure to long-continued cold, or sudden transitions from heat to cold, or from cold to heat; suppression of habitual evacuations, blows, fractured ribs, &c.

Treatment of Pleurisy.

Acute or Red Stage.—Our indication here is to destroy the inflammatory orgasm; and one of the most powerful means of effecting it is by venesection: the moment, therefore, that the pain in the side presents itself, a bleeding should be practised proportionate to the intensity of the disease and to the power of the patient. I need not repeat here, that one copious bleeding is more effectual than a

number of small ones ; thus, it is the practice in this country to bleed until the patient be completely relieved, so that he can breathe freely and without pain, or until he faints : and it often occurs that by this bold proceeding a farther abstraction of blood is unnecessary. This means should be repeated while the pain and fever continue with any degree of intensity. When the force of the pulse is diminished, and the pain in the side, although lessened, nevertheless continues, topical bleedings are useful, as cupping or leeches ; these may be followed by hot fomentations, although the latter are of little use unless they be continued for a considerable time, as an hour or two, or until the patient falls into a copious perspiration.

The tartar emetic has been employed in this stage of the disease, but its effects are not equal to those produced by mercury ; and I state here, what I believe I have mentioned before, that the tartar emetic seems to have most power in reducing inflammation of the parenchymatous structures, and mercury that of the serous membranes ; at least, such is the result of my experience.

From the commencement of the disease, after clearing the bowels, I begin the free use of mercury, by administering calomel every three hours, in doses of two or three grains, combined with a quarter of a grain of opium, if the bowels are irritable ; if that irritability be great, the safest method is, then, to rub in freely the strong mercurial ointment instead ; and you will almost invariably find that, when the mouth becomes tender, the inflammatory symptoms are all subdued.

Blisters are also advantageous when some relics of pain are left, and when the inflammatory fever has subsided; but if the pulse be quick and full, and the skin hot, I think they only irritate, without producing any good effect upon the disease. Mustard cataplasms are the best applications for children.

It is hardly necessary to state, that during the treatment of this stage the diet should be strictly antiphlogistic.

Stage of Effusion.—But, gentlemen, when we arrive at the stage of effusion, and when that state has become chronic, our indication is altogether changed: we have no longer an inflammatory action to subdue, but we have to facilitate the absorption of a fluid. Nature is most frequently sufficient to effect this herself; but when the fluid largely accumulates, and especially when the false membranes form, then her process is too slow to be compatible with the safety of the patient, and art must supply the remedies, which frequently, although not always, are successful.

The indication, then, is to cause the absorption of the effused fluid. Various means have been adopted with this view as purgatives, particularly the elaterium. Mercury, powerful as it usually is in effecting absorption, rarely succeeds in this case; diuretics, too, are but faithless remedies here. The acetate and nitrate of potash, the squills, digitalis, &c., have all been successively used, but they have not fulfilled the indication. Counter-stimulants, as blisters, setons, and cauteries, rarely either render much service.

Why are these means, usually so active in pro-

moting absorption, so powerless in these cases? It is because the false membranes lining the inflamed parts, and enclosing the fluid as in a bag, are themselves inorganic, or nearly so. How is the function to be performed through so thick a structure of inorganised lymph? It is scarcely possible. If, then, after having tried these remedies, — and it is proper you should try them, for you cannot be certain at this moment that the false membranes exist at all, — you find you make no progress, and the patient daily becomes worse, you should then evacuate the fluid by mechanical means — by performing the operation of *paracentesis thoracis*. As I have had occasion to direct this operation frequently, and have proposed certain modifications in effecting it, perhaps I may be excused from entering into some details upon the subject.

Paracentesis Thoracis.— Our time does not permit us to enter into the history of this operation: it was evidently performed by Hippocrates, and those of his school; for the directions they give are too practical not to have depended upon actual experience. The history of the operation of *parecentasis thoracis* resolves itself into the description of the various opinions as to the place where it should be performed, and the means by which the chest should be perforated. I refer those who wish for information upon this subject to the ninth volume of Sprengel's "History of Medicine." I have proposed the following means of evacuating fluid from the chest: —

In the first place, re-examine your patient with the utmost care; satisfy yourself that percussion

gives a dull sound; that there is no respiratory murmur; that the heart is pushed out of its situation; that the movements of the affected side are nearly or wholly lost; that the intercostal spaces are effaced, giving to the whole side a round and smooth appearance; and, finally, assure yourself by mensuration that the diseased side is the largest. If all these signs exist, you may be certain that fluid is present.

I have proposed an instrument by which you may prove the existence of the fluid with but little pain, and no danger, to the patient: it is the needle I present to you: it is somewhat thicker than that used for acupuncture; it is about an inch and a half in length, pointed like a trochar, and has a groove running nearly to its point: the groove should be made as deep and as wide as the thickness of the instrument permits. Selecting the centre of the most bulging part of the chest, which is usually between the seventh and eighth ribs, and about a hand's breadth from the spine, I then carefully examine for the intercostal space, by feeling for the edges of the ribs (for you recollect that the appearances of these spaces are lost to the sight), and introduce the instrument as nearly as possible into the centre; for, if you pass it upon the superior edge of a rib, you will find that the moment the intercostal muscles are pricked by it, they will contract and bring the two ribs together, so that, instead of penetrating the space, the point of the needle will strike the bone itself, be thrown up, and you may be foiled in your attempt, simple as it is, and endanger also the intercostal artery. If, however, you perforate the centre of the inter-

costal space, you can easily penetrate into the chest.

The needle being introduced to about three-quarters of its length into the pleuritic cavity, determines at once the presence of the fluid, and, what is equally important to the future steps of the operation, its nature; for, if it be serous, it will pass readily along the groove, and trickle down the back of the patient; if it be puriform and thick, it will not freely flow, but a thick drop or two will be seen at the external orifice; and on withdrawing the instrument you will find its groove filled with pus. In the first case, you may satisfy yourself that the false membranes scarcely exist at all, or at least, are so slight, that the lung may expand freely, and therefore more fluid may at once be evacuated; in the second, the probability is that the membranes are of greater thickness; that the lung will consequently be less free in its movements; and, therefore, that but a small quantity of fluid should be allowed to flow at a time. The different degrees of thickness of the matter shew you also the size of the trochar you should subsequently use, as a small one is sufficient for the evacuation of a serous fluid, but a large one only permits the continuous flow of thick puriform matter.

The advantage of this instrument is, that it demonstrates the presence of the fluid, and allows, therefore, the future steps of the operation to be conducted unhesitatingly; that it causes but little pain to the patient; and is as harmless in its effects as an acupuncture needle.

Having withdrawn the needle, you may then

introduce a trochar, the size of which, as I have already hinted, should be according to the thickness of the fluid you have discovered: if it be mere serum, a small hydrocele trochar is sufficient; if it be a thick puriform matter, a larger instrument must be introduced, otherwise the flow will be tedious. I recommend you, gentlemen, to be extremely careful in having the instruments sharply pointed; for, if they be not, after the pleura has been penetrated by them, the false membrane, from its imperfect attachment, may be driven before the point, and the operator foiled.

The quantity of fluid you should permit to escape must be according to its thickness: if it be mere serum, you may evacuate two or three pints, and the lung will freely expand; if puriform, I generally allow from twelve to sixteen ounces to flow before I stop it,—recollecting that the lung is then, to a certain degree, bound down by the false membranes.

It now becomes necessary to establish a communication between the pleuritic cavity and the exterior; for this purpose, I fit a portion of gum-elastic catheter, previously cutting off its rounded extremity, into the canula of the trochar, and pass it through that instrument into the chest, to the distance of about three inches; the canula may then be withdrawn, allowing about four inches of the catheter to remain at the exterior, which may be secured in its position by tapes attached to it, and passed round the body: the whole should be still farther secured by adhesive plaisters.

The fluid now passes through the catheter instead of the canula of the trochar, and you may

stop the flow by introducing a small plug, made of a portion of bougie, into the external extremity of the former instrument. Having thus a free communication at will, I daily evacuate a certain quantity of fluid, retaining the catheter in the wound for two, three, or four weeks; in fact, until I can obtain no more fluid. You need not be apprehensive of the presence of the gum-elastic catheter in the cavity of the pleura; I have never seen it do harm. From its elastic nature, it soon bends, and places itself closely against the inside of the ribs.

You will often find that after the second day sufficient ulceration is effected, by the presence of the catheter in the wound, to enlarge it; in that case the fluid flows, though very slowly, by the side of the instrument. I endeavour to favour this by directing the patient to lie upon the diseased side. Often, after the instrument is entirely withdrawn, a fistulous orifice will remain for some weeks, from which a slight discharge passes. I have always seen that this circumstance is favourable, and have never observed danger to arise from it. Some have feared that air would enter, and occasion inflammation. No doubt air frequently does enter into the cavity; but I have not perceived it produces this result: indeed, the slightest reflection must show us, that the false membranes are as yet incapable of inflammation, in consequence of their inorganic nature.

Laënnec states that the operation for empyema is rarely attended with success. I cannot agree with him in that opinion. If he had said that the operation, when performed for the cure of pneumothorax combined with effusion, was generally un-

successful, I most fully agree; for it has occurred to me, that, out of sixteen individuals in whom paracentesis thoracis has been performed for simple effusion of serum, or puriform matters in the chest, twelve have perfectly recovered, and four only have died. Where, however, there has been a combination of pneumo-thorax and effusion of fluid, the operation has been invariably unfortunate in its results. The reason of this difference will be rendered obvious when I treat of the latter disease.

The causes which render the operation for simple effusion unsuccessful are, the existence of tubercular or other disease in the lungs, and, above all, the degree of thickness of the false membrane, which prevents the contracted lung from expanding. I have found that children most frequently recover, probably from the lungs being more commonly healthy in them, and from the great pliability of their chests, by which the parietes fall in more readily upon the contracted lung.

I shall in the next lecture offer a tabular view of these operations, with their results.

LECTURE XX.

DISEASES OF THE PLEURA—*continued.*

HYDROTHORAX.

Definition. — Idiopathic — Sympathetic — Character, &c. — Mechanical Effects of Fluid. — Signs. — Treatment.

PNEUMO-THORAX, simple or combined with Effusion.

Definition. — *Characters* of the accumulated Air. — *Causes* — from Chronic Pleurisy — Rupture of Tubercular Excavation into the Pleura — Gangrene of Lung — Blood effused — A blow rupturing the Air-cells — Rupturing of Cells of an emphysematous Lung — Spontaneous Exhalation of Gas.

Signs of Simple Pneumo-Thorax. — *Functional.* — Dyspnœa — Cough. *Local.* — Percussion — Respiratory Murmur — Size of the Chest on the diseased Side. — *Diagnosis.*

Signs of Pneumo-Thorax with Effusion. — Respiratory Murmur — Percussion — Fluctuation — “Tintement Métallique” — “Bourdonnement Amphorique.” — *Treatment.* — *Table* of Operations for Paracentesis Thoracis. — Results.

Pleuro-Peripneumonia. — First, Peripneumonia, combined with slight Pleurisy. — Second, Pleurisy, with slight Peripneumonia. — Third, Pleurisy and Peripneumonia of equal Intensity.

Accidental Productions deposited in the Pleura. — Diaphragmatic Intestinal Hernia — Hernia of the Lungs through the intercostal Muscles.

By hydrothorax we mean an effusion of limpid serosity into one or other, or both, the cavities of the pleuræ, not depending upon previous inflammation.

This disease has been denominated *Dropsy of the Chest*; more commonly also *Water in the Chest*; and has often been mistaken for other affections—as diseases of the heart, aneurism of the aorta, emphysema, and œdema of the lungs.

Hydrothorax may exist in the idiopathic or sympathetic forms : the first is so rare, that Laënnec calculated he had not met with it more than once in 2000 post mortem examinations. The fluid then occupies one side of the chest only. The sympathetic form usually affects both pleuritic cavities, and is a common consequence of acute and chronic diseases ; especially of scarlatina, and, above all, of organic lesions of the heart. Laënnec states, that sympathetic hydrothorax indicates the rapid and fatal termination of the disease which is its cause ; that it rarely commences to develop before a few days or hours preceding death, and that nothing is more uncommon even in diseases of the heart, accompanied by universal leuco-phlegmasia and ascites, than a hydrothorax whose signs have appeared eight days before death. I entirely differ from him upon this point, for I have seen the signs of the sympathetic affection presenting themselves two or three months before the fatal termination of the disease, and have afforded temporary relief by tapping the chest in two individuals who had long laboured under sympathetic hydrothorax.

In both forms of the disease the effused fluid presents the same characters ; it is limpid, and of a fawn colour : the pleuræ are perfectly healthy. The quantity of the secretion varies from a few ounces to many pints, and the effects of its accumulation are precisely the same upon the surrounding parts as I have described in effusion from inflammation of the pleura ; the signs are also similar. Thus there is absence of the respiratory murmur, dulness on percussion, œgophony, altered position of the

heart, enlargement of the side affected, effacement of intercostal spaces, immobility of ribs, with dyspnoea and cough.

General Signs.—Sympathetic hydrothorax is itself one of the general signs of some other disease—as of fevers of long duration, and of affections of the heart. I shall again advert to this subject when we arrive at the consideration of the latter class of disorders.

Treatment.—Idiopathic hydrothorax is so rare, that I have not had occasion to treat it; but the indications would be the same as for any other species of dropsy. The treatment of the sympathetic form should be subservient to that of the disease of which it is the consequence, and will be spoken of hereafter. I have twice directed the operation of paracentesis thoracis in these cases, and have thereby produced temporary relief.

PNEUMO-THORAX.

By pneumo-thorax is meant an effusion of aëri-form fluids into the cavity of the pleura. This disease, in its simple form, is rare, although it is not very infrequent when combined with serous or puriform secretions in the same cavity. This affection was first mentioned by Riolanus, and afterwards became the subject of the inaugural thesis of the celebrated Itard, physician to the hospital of “Sourds-Muets,” at Paris.

The gases accumulated in the pleuritic cavity are sometimes inodorous, at others foetid, and similar to sulphuretted hydrogen. Their quantity is occasionally very considerable, and they then produce

the same effects upon the surrounding parts as serous or puriform fluids placed in the same situation: the lung becomes compressed; the heart is pushed from its natural position; the thoracic parietes are dilated; and the diaphragm depressed.

Causes.—Air is frequently met with in the pleura, combined with fluids resulting from chronic pleurisy. In that case, when there is no communication between the pleura and the bronchial tubes, we must suppose that it is formed from the decomposition of that fluid: the gas then has a most foetid odour, assimilating to that of sulphuretted hydrogen.

A tubercular excavation has been seen to burst into the cavity of the pleura, producing a free communication between it and the bronchial tubes: atmospheric air then of course passes into the pleura, which in its turn produces pleurisy and consequent effusion of fluid. An excavation has also been seen to burst into the pleura, and not into the bronchial tubes; the softened tubercular matter then irritates and inflames the serous membrane, and a liquid secretion is again the result, the decomposition of which affords air sufficient to produce pneumo-thorax.

Gangrene of the lung causes pneumo-thorax; not only from the decomposition of the structure of the organ, but also from its establishing a communication between the bronchial tubes and the pleuritic cavity. Puriform secretions are always present in this case, the decomposition of which also produces aëriform exhalation.

Blood effused from any cause into the pleura may decompose, and give rise to the formation of air.

A violent blow upon the chest may rupture the pulmonary pleura, and occasion extravasation of air into its cavity. Hewson and Laënnec mention instances of this.

The rupture of an emphysematous lung may cause effusion of air into the pleura. I have seen a remarkable instance of this case in a child, in whom the necessity for respiration had so increased in one lung, in consequence of the total hepatisation of the other, that it had become completely emphysematous; one or more of the enlarged air-vessels had ruptured, and air had effused and accumulated in the pleura, in such quantity, that the lung was compressed to a very small size.

Finally, an aëriform fluid may be generated in the cavity of the pleura, without any visible change in the membrane, or appearance of any other effusion. The serous membrane has then occasionally appeared drier than usual; Laënnec has seen it as dry as parchment.

You thus perceive, gentlemen, that simple pneumo-thorax is a rare affection, and that there are only two or three apparent causes of it: first, from the rupture of an emphysematous lung; secondly, from a blow rupturing the pleura pulmonalis; thirdly, from a spontaneous exhalation of air: while, on the other hand, the disease frequently arises from the decomposition of liquid effusions, or from the direct passage of air from the bronchi into the cavity of the pleura, constituting cases of what Laënnec calls pneumo-thorax with effusion. Let us first examine the signs of simple *pneumo-thorax*, and then those of *pneumo-thorax with effusion*.

Signs of Pneumo-Thorax.

Functional.—Dyspnœa is a constant sign of this disease. Cough is not always present, except the bronchial tubes be also affected.

Local.—As the loudness of the sound upon percussion depends upon the quantity of air contained in the chest, so in this case the diseased side of the thorax is always more sonorous than natural. But this sign alone is insufficient to determine the nature of the affection, because the sound is also as loud upon percussion in emphysema of the lung. An error, too, might arise from the supposition that the most sonorous side was the most healthy one; when, in fact, it is that which is diseased.

But the conjunction of the signs derived from auscultation and percussion give certain proofs of the presence of air in the cavity of the pleura: thus, if the respiratory murmur be entirely absent on the side which is most sonorous by percussion, you may fairly infer that it is a case of pneumothorax. It is scarcely necessary to state, that the respiratory murmur may be heard, as in empyema, between the base of the scapula and the corresponding part of the vertebral column; and for the same reason, because of the lung being compressed against the vertebræ at that part.

In consequence, also, of the pressure of the air upon the intercostal muscles and ribs, the side affected becomes larger, and the degree of increase may be determined by measurement.

The only affection which produces similar signs is emphysema of the lungs; but there are differences which render the two diseases easily distin-

guishable: for in pneumo-thorax the absence of the respiratory murmur is complete, except at the points indicated; but it may still be heard, although feebly, at different parts of the chest, in emphysema. In the latter disease a slight rhoncus is occasionally apparent, even close to the ear, which never occurs in pneumo-thorax. The dry crepitating rhoncus is also pathognomonic of emphysema. Finally, pneumo-thorax is always a sudden affection; but dilatation of the air-cells is a chronic disease.

Signs of Pneumo-thorax with Effusion.

In this combination the respiratory murmur is absent all over the surface of the affected side, except at the part corresponding to the situation of the compressed lung. The sound on percussion varies: thus, at the lower part of the chest, where the fluid has gravitated, it is dull; but at the upper part, where the air is accumulated, it is loud.

There are three other signs, which, when united, are pathognomonic of air and fluid matters co-existing in the pleura. These are the *Hippocratic fluctuation*, the *tintement métallique*, and the *bourdonnement amphorique*.

Hippocratic Fluctuation.—*Succussion.*—Hippocrates proposed, for the purpose of discovering fluids in the chest, that the patient should be placed on a solid seat, his arms being extended by an assistant; and that he then should be shaken by the shoulders, to determine a fluctuation.

This method of succussion had fallen entirely into disuse, although, no doubt, it had been often

tried, but without success, in the great majority of cases. We have, however, several instances on record, in which a fluctuation has been felt by the patient himself upon a sudden and spontaneous movement of his own; as in turning quickly in bed, or in descending a staircase, or in riding on horseback. There can be no doubt that, when a fluctuation is thus accidentally produced, it might also be caused by the Hippocratic mode of succussion, as I have verified in a great number of cases.

What is the reason that succussion will induce fluctuation in some cases of fluid in the chest, and not in others? The reasons are obvious. In empyema or hydro-thorax nothing is contained in the cavities of the pleura but liquids; no movement in such cases can produce fluctuation, no more than it could in a perfectly filled bottle; but if air coexist with the fluid, as in pneumo-thorax with effusion, then the fluctuation becomes distinct, as it would upon shaking a bottle partly filled; for the fluctuating sound can only be produced by the reciprocal action of air and liquids upon each other in motion.

Fluctuation upon succussion is, then, a sign of pneumo-thorax with effusion. The liquid matter should always, of course, be in a certain quantity; but the loudness of the sound depends upon the air being in the greatest proportion; for we constantly find that if the pus or serum predominates, the sound of the fluctuation is slight; if the air be in excess, it is considerable.

This sound is always distinguishable by the stethoscope; and even when slight, it may be detected by it when it cannot be heard by the

intermedium of the air. If the hand be also applied to the surface of the chest, the shock or vibration produced by the fluctuation may easily be felt.

There is another case in which air and fluid may coexist in the same cavity, and in which, consequently, fluctuation should be produced by succussion: I mean a very large tubercular excavation, containing a certain quantity of secretion within it. I have essayed in many of these cases to produce this sound, but never could effect it. I believe this arises from the fluid being in very small quantities in such cavities; perhaps, also, their soft parietes are not equally favourable to the production of fluctuation.

Tintement Métallique.—I have already described the nature of this sound, in my lecture on Auscultation. It is like that produced by frequently and gently striking a thin metallic or glass vessel with a pin. It is heard during the acts of respiration, speaking, or coughing. The tintement métallique indicates the presence of air and fluid in the pleuritic cavity; but Laënnec asserts still more,—that it is a proof of a fistulous communication between a pulmonary excavation, opening at once into the bronchi and pleura. I have often, it is true, met with a fistulous opening when the tintement métallique has been present; but often also I could not, after a most careful examination, discover any such fistulous communication. I have therefore arrived at the conclusion, that this sign, although perfectly indicative of pneumo-thorax with effusion, is not pathognomonic of a fistulous opening between the bronchial tubes and the cavity of the pleura.

The tintement métallique is also a sign of a large tubercular excavation, containing a certain quantity of fluid; for here the reunion of the circumstances necessary to its formation obtains; that is, there is a large cavity containing air and liquid.

Bourdonnement Amphorique. — This is a sound like the buzzing of a bee in a large vase. There are, however, great varieties in this sign, especially in its loudness. It is always somewhat argentine, or metallic; it is occasionally like a thin, shrill, and scarcely audible silvery whistle, softly resounding afterwards, as if echoing in a metallic vessel. This sound is always indicative of air and fluid contained in a large cavity in the lung, or of pneumo-thorax with effusion. I believe the bourdonnement amphorique never occurs in the latter disease, except where there is the fistulous communication before spoken of: it appears to me to be formed by the air passing into the bag of the pleura through the fistula, and resounding afterwards. It is produced during respiration, speaking, and coughing.

As the presence of a certain quantity of liquid and air in an excavation in the lungs, or in the pleuritic cavity, causes these sounds, have we any diagnostic signs by which we can distinguish whether they arise from one lesion or the other?

In the first place, I have never met with an instance of fluctuation in a tubercular excavation, although I have always attempted to produce it when I have heard the tintement métallique, or bourdonnement amphorique. I do not, however, deny the probability of its occurrence, but I may be fairly allowed to infer it must be very rare

indeed. In the next place, when there is a tubercular excavation, the tintement métallique is usually confined to the upper half of the lung; and in pneumo-thorax with effusion it is much more extensive, although I once met with so large a tubercular cavity, that scarcely any of the lung remained, except a small mass attached to the larger divisions of the bronchial tubes, and a thin lining of its substance adhering to the costal pleura: in this case, these two sounds were audible all over the side affected, but still there was no fluctuation. Thirdly, I have observed that patients having tubercular excavations, however large, lie indifferently on either side, whilst those afflicted with pneumo-thorax and effusion prefer resting on the diseased side.

In the present state of medical science, we can effect but little, indeed, by any treatment, because the disease is almost constantly combined with phthisis: at least, I have not met with a single instance in which it was not. I have frequently tapped the chest on these occasions, but never produced any thing but temporary relief: in fact, I have now ceased performing the operation of paracentesis thoracis in these cases, except the patient be very distressed by dyspnœa. I would recommend you, gentlemen, when you have determined upon evacuating the fluid, always to explain to the friends the nature of the case with as little technicality as possible; state that you cannot save, but may ease, the patient, and possibly protract his life for a little time.

I now present a tabular view of the number of times I have directed the operation of paracentesis

thoracis, the nature of the case for which it was performed, the names of the operators, and the results:—

Nature of the Cases.	Operators.	Recovered.	Under Treatment.	Died.
Empyema -	Mr. Sturkey, Lung } Infirmery - - }	—	—	1
	Mr. Martin, Surgeon, } R. N. - - - }	1	—	—
	Mr. Headington - -	1	—	—
	Mr. John Scott - -	4	—	1
	Mr. Kiernan - - -	2	—	—
	Mr. Herring, Lung } Infirmery - - }	3	—	1
	Dr. B. Babington - -	1	—	—
	Mr. Skey - - -	—	—	1
	Total Empyema - -	12	—	4
Pneumo-thorax with effusion	Mr. Headington - -	—	—	3
	Mr. John Scott - -	—	—	2
	Mr. Kiernan - - -	—	—	3
	Mr. Bryant, Clapham } Road - - - }	—	—	1
	Total Pneumo-thorax - -	—	—	9
Hydro-thorax	Mr. Herring, Lung } Infirmery - - }	—	—	1
	Mr. Kingdon - - -	—	—	2
	Total Hydro-thorax - -	—	—	3
Total of Operations - - - -		28		

OBSERVATIONS.

Empyema. — Of the sixteen cases, twelve have recovered, and four have died.

Of the twelve individuals cured, five were under six years, one at eleven, one between eighteen and nineteen, and five above twenty-five years of age.

In three of the unsuccessful cases, the lungs could not expand after the evacuation of the fluid, in consequence of the thickness of the false membranes covering them; of the fourth I have no account.

Pneumo-thorax with Effusion. — All died, from tubercular combinations.

Hydro-thorax. — These cases were of the sympathetic form consequent upon diseases of the heart. These patients were tapped merely with a view to temporary relief.

Note. — This Table being revised in December, 1834, varies in the number of cases from that given at the time the lecture was delivered — in March, 1834.

PLEURO-PERIPNEUMONIA.

Inflammation of the pleura and of the substance of the lungs often coexist. Laënnec has considered these combinations under three forms or varieties.

1. Peripneumonia complicated with slight pleurisy; 2. Pleurisy with slight peripneumonia; 3. Where the inflammation of the pleura and the lungs is of nearly equal intensity.

1. *Peripneumonia combined with slight Pleurisy.*

There are few cases of peripneumonia in which the pleura is not slightly affected; for when the inflammation of the lung has reached the surface of the organ, the pleura covering it inflames, and a thin false membrane is formed, and is exactly bounded to the part where the disease has gained the surface; sometimes even a false membrane is also formed upon the costal pleura, exactly opposite. When hepatization attacks a certain portion of the lung only, there will be effusion of fluid if the pleura be also inflamed; but if the organ be almost entirely consolidated, it presents so firm and incompressible a mass, that there will be no effusion, but merely a very thin and incomplete false membrane; thicker, however, at the edges and fissures and on some points, than on the generally inflamed surface.

Signs. — If the patient were seen for the first time in this state, it would be very difficult to distinguish whether he laboured under pleurisy with effusion, or hepatization of the lung; for the dullness on percussion, and the absence of the respira-

tory murmur, would be the same in both cases. But when a lung is completely hepatised, there is always a very loud and distinct bronchophony, almost similar to pectoriloquy, at various points, particularly at the summit and the root of the lung; a sign which does not exist, to the same extent or degree, either in pleurisy or pleuro-peripneumonia.

But if the disease has been seen from the beginning, it is almost impossible to mistake it; for the rhoncus crepitans always precedes the absence of the respiratory murmur, and the dulness of the sound on percussion only appears gradually. In pleurisy the dulness is sudden on the whole of the side affected. The œgophony also occurs, at least for a day or two, in pleurisy.

2. *Pleurisy, with slight Peripneumonia.*

It occasionally happens, when a pleurisy is severe, and accompanied by a considerable and rapid effusion, so that the lung is extensively and speedily compressed, that inflammation develops itself in some points of the organ, usually in the inferior lobe: these points are often isolated, and of slight extent, constituting what has been called *lobular pneumonia*. It is probable that the inflammatory action in the lung is moderated by the pressure of the effused fluid, so that it is confined to a few lobules, and cannot extend farther. The lobular pneumonia rarely proceeds to the stage of purulent infiltration; its resolution is slower than that of simple peripneumonia, and it presents very peculiar anatomical characters: thus the lung is less firm and more flabby than when in a state of hepatiza-

tion; the pulmonary tissue is of a red, violet, or gray tint; a section of it presents the granular appearance of hepatization; it has altogether the aspect and consistence of muscular flesh which has been bruised to render it tender. Laënnec denominates this state *carnification*. There is now no trace of air-cells; no air can be passed from the part, and it is but slightly humid.

The resolution of this state is slow, under the pressure of the pleuritic effusion. When it occurs, the affected part becomes less red, then changes into a pale violet, which latter becomes of a grayish colour; at the same time the vesicular texture of the lung reappears.

The resolution of peripneumonia under the influence of the pressure of fluid, in the stage of purulent infiltration, is very rare indeed. Laënnec describes instances in which he had seen carnified portions of lung of a yellowish tint, in which the air-cells were distinguishable, and appeared filled with semi-concreted pus.

Signs.—The complication of even a slight peripneumonia with an abundant pleuritic effusion, may generally be distinguished by the crepitating rhoncus, which usually occurs at the root of the lung, in the axilla, or a little below the clavicles.

This complication can hardly take place, except at the commencement of the disease, and when the effusion is not yet great; for when the lung is completely compressed, it is no longer susceptible of inflammation.

3. *Pleuritis and Peripneumonia of equal intensity.*

This conjunction is much less frequent than the two preceding cases. The pleurisy does not increase the danger of the peripneumonia: it even diminishes it, in moderating the inflammation by the compression of the effused fluid. On the other hand, the peripneumonia augments at first the danger of the pleurisy, which rarely threatens life at its acute period, and it renders the absorption of the fluid more rapid, and does not permit so great an accumulation of it as occurs in simple pleurisy; for this liquid is thrown out between two bodies, neither of which cedes to the pressure exercised upon them; viz., the hardened lung on one side, and the firm thoracic parietes on the other. Laënnec therefore infers that this form of pleuro-peripneumonia ought to be regarded as less dangerous than either simple pleurisy or peripneumonia.

Signs. — The reunion of the signs of pleurisy and peripneumonia renders these combinations easily determinable; many of their pathognomonic signs are even more permanent than in the simple, or uncombined affections; thus the rhoncus crepitans, and ægophony, often persist even to the period of convalescence. The latter sound is usually only apparent at the root of the lung, and in the vicinity of the inferior angle of the scapula; and from the presence of the large bronchial divisions, as well as from the density of the pulmonary tissue, it is rarely simple, but is generally accompanied by a loud and noisy bronchophony; it is in this case particularly that the united sounds imitate so perfectly the voice of *Punch*.

Treatment. — The treatment of pleuro-peripneumonia ought to be regulated according to the predominance of one or other of these diseases; it is therefore unnecessary, after what has already been said, to repeat what I have stated in speaking of the uncomplicated forms of the separate affections.

ACCIDENTAL PRODUCTIONS DEPOSITED IN THE PLEURA.

Laënnec has divided these formations into three classes.

1. *Productions which develop themselves on the inner Surface of the Pleura, ordinarily accompanied by liquid Effusion.*

These are usually cancerous or tubercular deposits. The first generally offers the characters of medullary sarcoma, presenting masses of various sizes, but rarely, nevertheless, beyond that of an almond: they are strongly adherent to the pleura, which membrane is of an areolar redness around these deposits, for some distance; sometimes melanotic lines are seen to pass from the tumours to the surface of the pleura: the tumours are rarely numerous.

Tubercles are occasionally deposited in great numbers upon the surface of the membrane: they approximate closely, and are often united by a soft and semi-transparent false membrane, in which they seem at first imbedded. At a remoter period from their formation, this false membrane can no longer be found, because it is organised and united to the pleura: the tubercles are then very adherent, and

appear implanted into the thickness of that membrane. Sometimes the tubercles are gray and semi-transparent, sometimes yellow and opaque, but they have not been seen softened: the interstices between them are generally reddened. In this state the pleura has the aspect of certain miliary eruptions on the skin; and in the midst of the redness lines of melanotic matter may be seen. Although tubercles are commonly first deposited in a false membrane, yet they may occur in the pleura without any signs of previous inflammation.

Small, white, opaque, and flattened granulations, of a firm and fibrous texture, are occasionally seen on the surface of the pleura; the membrane is then thickened.

These two productions are more commonly observed upon the peritoneum than the pleura: they are accompanied by effusion, which is generally sanguineous: cancerous productions sometimes form an exception to this rule. The signs of effusion are, of course, apparent, but there are none of the organic causes which have occasioned it.

2. Solid Productions.

The pleura, instead of secreting its serosity, sometimes deposits tubercular or cancerous matters in large quantities, which accumulate and gradually compress the lung against the vertebral column, and thus fill up one side of the chest: these cases are very rare. Boerhaave found a white matter filling up one side of the chest of a patient; Corvisart mentions a similar case; Recamier found an enormous tubercular mass occupying the cavity of the pleura;

Cayol and Laënnec had each observed analogous instances: Haller appears, also, to have met with a considerable mass of softened melanosis in the same situation.

3. *Accidental Productions developed between the adherent Surface of the Pleura and the neighbouring Parts.*

Medullary sarcoma and tubercles, imperfect or petrous ossific deposits, and large cartilaginous incrustations, have occasionally been found in this situation. Haller and Dupuytren have described large cysts, placed between the pleura and the intercostal muscles, filling almost entirely the pleuritic cavity, and compressing the lungs.

DIAPHRAGMATIC INTESTINAL HERNIA.

A wound, a rupture from a fall, or a malformation of the diaphragm, may occasion an opening sufficiently large to admit a portion of the intestines, or even the stomach, into the cavity of the chest. Herniæ of this kind could only be distinguished by the absence of the respiratory murmur at the points of the chest corresponding to the position occupied by these viscera, and by the borborygmata which might be heard in the same situations.

The lungs have been seen forming hernia through the intercostal muscles. Grateloup mentions one produced by a violent cough; Boerhaave speaks of another, produced by the efforts of accouchement; Sabatier a third, which appeared after

the cicatrization of a bayonet wound between the fifth and sixth ribs; Richter gives a fourth instance; and two cases have lately been observed at Paris.

The respiratory murmur would be most distinctly heard in tumours of this kind, by which their nature could easily be determined.

LECTURE XXI.

DISEASES OF THE HEART.

THEORY OF THE SIGNS OF DISEASES OF THE HEART.

Generalities. — Position of the different parts of the Heart, in relation to the Chest. — *Percussion.* — *Auscultation.* — First, *Extent* of Pulsation — in the healthy and diseased States. — *Rules* — *Exceptions* to them. — *Cautions.* — Second, *Impulsion* given by the Cardiac Movements. — Impulsion proportionate to Thickness of Parietes of the Organ. — Natural State. — Diseased State. — Mode of Impulsion. — Back Stroke of Dr. Hope. — *Rules.* — *Exceptions.* — Third, *Rhythm* of the Movements of the Heart. — Volume of the Heart. — Comparison of the Size and Thickness of the Ventricles. — Impulse with first Sound and arterial Elevation — isochronous with the Systole of the Ventricle. — Second Sound — with that of Auricle. — Rhythm, according to Laënnec's Theory. — Error of Laënnec. — Dr. Hope's Experiments. — Rhythm, according to their Experiments. — Rhythm altered in Diseases of the Heart. — Fourth, *Sounds* produced by the Movements of the Heart. — Two Sounds — their Character — where heard — when weakest — when loudest. — *Anormal Sounds* produced by the Action of the Heart and Arteries. — "Bruit de Soufflet." — "Bruit de Scie ou de Râpe." — "Bruit de Soufflet musical ou sibilant." — Character of these Sounds. — *Frémissement Cataire*. — Characters — where found — accompanied by Bruit de Soufflet. — Causes. — Beatings heard at some Distance from the Chest. — Cause.

THE diseases of the heart, like those of the lungs, were involved in great obscurity from the earliest period of medical history to the present century. It is true, more had been effected towards the description of the morbid changes of the heart than of those of the pulmonary organs, by the suc-

cessive labours of Lancisi, Morgagni, and, above all, of Senac ; but whilst these authors pretty tolerably described the organic changes which took place in this important viscus, they overlooked almost entirely the physical causes which commonly produce them. Since the time of these illustrious anatomists, the morbid anatomy of the heart has been so accurately described, that, perhaps, there is nothing left undone. We owe this perfection to the works of Corvisart, Burns, Bertin and Kreysig, and Laënnec.

But although the lesions of the tissues composing the cardiac organ were earlier understood than those of the lungs, yet their signs were, if possible, still more obscure than those of the latter viscus. It may be said that there were then no local signs, and that the functional and general symptoms also were extremely uncertain ; thus palpitations, or irregularities of the pulse, may occur, either with or without a change of the structure of the heart ; and dyspnœa being a common sign of the affections of both the thoracic viscera, is insufficient alone to demonstrate which of them is diseased. The great discovery of Laënnec has, however, so completely unveiled all obscurity relative to the signs of cardiac diseases, that we are now enabled to distinguish even which particular cavity or valve of the organ is affected.

In prosecuting this subject, we shall first treat of the theory of the local signs of the diseases of the heart ; and, secondly, of the diseases of that organ, of the pericardium, and of the large blood-vessels.

Before we consider the theory of the signs, it

will be necessary to describe the situation of the different parts of the heart in relation to the parietes of the chest.

Laënnec divided the precordial region into two parts; the right corresponding to the inferior third of the sternum, and to the right side of the heart; and the left comprising the space between the cartilages of the fourth and the seventh sternal ribs, having beneath it the left side of the organ.

I have followed the example given by Dr. Hope, of passing needles into the chest, for the purpose of ascertaining the relative positions of the thoracic parietes to the different cavities and valves of the heart, and to the large blood-vessels. Placing a subject upon its back, I dissected the skin from the anterior part of the chest, and drilled a hole through the centre of each bone of the sternum, and passed long knitting needles through them as perpendicularly as possible to the vertebral column, and fixed them there. The intercostal and pectoral muscles were then removed, so that the anterior surfaces of the lungs and the pericardium could be seen. The sternum was carefully removed, leaving the needles transfixing the soft parts beneath it. The centre of the first bone was seen to correspond to the lower edge of the left subclavian vein, and to the arch of the aorta crossing the trachea. The centre of the second bone corresponded to the right side of the ascending aorta, and to the upper edge of the appendix of the right auricle. The third needle passed into the right side of the right auricle, and the fourth into the right ventricle. Another needle penetrating the chest at the upper edge of the cartilage of the

fifth rib, close to its costal extremity, entered into the septum of the ventricle; the point of the heart was about an inch and a half below this, and inclined to the left side.

The pulmonary semilunar valves corresponded to a spot a little below the centre of the third bone of the sternum, the aortic valves were a few lines below and behind the pulmonary, and the mitral were still more deeply seated, and a little lower also than the aortic. The origins of the pulmonary artery and aorta were slightly below, and to the left of, the centre of the third bone. The pulmonary artery passed upwards, touching the sternum, inclining to the left, and was found between the second and third ribs, close to the sternum; the aorta ascended to the first bone, crossed it, and then formed the arch.

One third of the heart, consisting of the upper part of the right ventricle, and of the whole of the right auricle, was situated immediately beneath the sternum; and the rest, being the remainder of the right ventricle, the left ventricle, and auricle, to the left of that bone.

Nearly the whole of the anterior surface of the heart is formed by the right auricle and ventricle; a small portion only of the left ventricle and its point, and the appendix of the left auricle, forming a part of that surface. The left auricle, and the greater portion of the left ventricle, form the posterior surface.

The pericardium invests the heart from the diaphragm to the commencement of the arch of the aorta.

Percussion.

The heart being a solid body, at least in relation to percussion, ought to give a dull sound upon striking the parietes of the chest covering it; but, perhaps, from its being imbedded, as it were, in the lungs, and from its being partially also covered by them, the sound is not so fleshy as that produced by striking over the liver, or the pulmonary substance, consolidated by hepatisation, or compressed by a fluid. The sound on percussion is something intermediate between that produced at the upper part of the sternum and the muscular parts of the body. The sounds, also, are still duller and more uncertain when the parietes of the chest are charged with fat, or are infiltrated with serum.

All lesions, however, which by their action distend the pericardium, increase the dulness of the sound upon percussion, as hypertrophy and dilatation of the heart, the dilatation of the auricles or ventricles, hydro-pericarditis, accumulations of blood in the pericardium, &c.

Auscultation.

There are four points of view in which the heart may be examined with reference to auscultation.

1. *The extent* to which the beatings of the heart are heard upon the thoracic parietes.
2. *The impulse, or shock*, communicated to the ear of the observer by the movements of the heart.
3. *The rhythm* of the movements of the heart.

4. The nature of the *sounds* produced by the heart's action.

1. *Extent of Pulsation.* — When the ear or the stethoscope is applied to the precordial region, the beatings of the heart are usually heard most distinctly all over it. There are but few exceptions to this rule. It sometimes, however, occurs, that, in individuals who are very fat, or whose thoracic parietes are infiltrated with serosity, these beatings are confined to a space not greater than the circumference of the stethoscope; or the sounds of them may even be almost entirely lost. If extensive catarrh be united to the above-described conditions, the deep sonorous wheezing tends still farther to mask the cardiac sounds. In all ordinary cases, the pulsations are perfectly evident over the space corresponding to the inferior half of the sternum, and to the cartilages of from the fourth to the seventh ribs. The movements of the right side of the organ are heard in the former region; those of the left, in the latter. If the sternum be short, they can also be distinguished in the epigastrium.

If the heart be heard beating only at the points I have just described, it may be considered to be of good proportions.

When the heart increases in its volume, the sounds of its movements are heard, with equal, or nearly equal, intensity, to an extent over the surface of the chest proportionate to that increase: thus, if it be slightly enlarged, its pulsations are distinguishable on the left side of the chest from the axilla to the region corresponding to the stomach; if larger, its sounds may be heard to the

same extent on the right side; if the organ be of very large proportions, then its beatings are audible on the left side of the spine, and, finally, though rarely, on the right.

When, also, the heart is of its normal size, or nearly so, the extent of its pulsations may be modified by the thickness of its parietes: thus, if they be very thin, the sounds are more extensively heard than in their natural state; if they be thick or hypertrophied, the pulsations are often concentrated in a small space. When, however, the cavities of the organ are dilated, and their walls are thickened, then the heart is occasionally enlarged to an enormous volume, and its beatings may be heard all over the chest.

The following observations may be made upon this subject:—1. That the extent of the pulsations of the heart is increased whenever its volume increases, especially if the augmentation of the size of the organ depends upon the enlargement of its cavities, combined with hypertrophy of their parietes. 2. That, although the heart be of its natural size, or nearly so, if its parietes be thin, the sounds of its beatings will extend beyond the precordial region; this probably depends upon the increased *loudness* of the sound, which we shall hereafter describe to exist, under these circumstances. 3. That in concentric hypertrophy of the heart, where its walls are thickened, and the capacities of its ventricles proportionately diminished, the pulsations are not heard, or at least but extremely slightly, beyond the cardiac region; and sometimes even they are not distinguishable, except upon a spot equal in surface to a square inch.

Are there any exceptions to the rule, that the extent of the pulsations of the heart is proportionate to its volume? There are several, and it is of the greatest importance to understand them, as I find that, practically, constant errors are made upon this subject.

In the first place, any cause which renders the lung solid, will occasion it to be a better conductor of the sounds of the heart, and consequently increase the extent of the surface of the chest over which they may be heard; as hepatization, or even the firm parietes of a tubercular excavation. Fluids contained in the pleuritic cavities will produce the same effect. The extent of the sounds is greater in persons having narrow and contracted chests; the heart then, although, perhaps, of its natural size, has not sufficient space to perform its movements freely, and, therefore, is too large for the diminished capacity of the part in which it is placed. The hearts of children are also larger proportionately than those of adults, so that their pulsations can be distinctly heard all over the thorax; and, finally, the extent of the pulsations is always increased by any cause tending to accelerate the movements of the organ; as nervous emotions, fevers, &c.

To determine the volume of the heart, attention, then, should be paid to the configuration of the chest, to the age of the patient, to any alarm he may feel from the mode of your examination, and to febrile action augmenting the frequency of the pulse. Even then do not hurry to a conclusion, but make repeated and daily examinations before you form a diagnosis.

2. *Impulsion*.—By *Impulsion*, or *shock*, is meant the sensation of elevation communicated to the ear of the observer, in consequence of the heart striking the parietes of the chest with a certain force.

The stethoscope renders this elevation much more evident than the application of the hand; the shock often cannot even be distinguished by the sense of touch. Sometimes, too, in those persons who are agitated at the moment of examination, a considerable impulsion will be given to the hand, which the stethoscope shows to consist of but little real increased force of the action of the heart. Care must be taken not to mistake the impulsions of the heart's movements for the short and rapid inspirations which occasionally take place in dyspnœa. A very slight attention will render such an error impossible.

This proposition may be advanced relative to the force of the heart's action, — that the intensity of its impulse, or shock, is in a direct ratio to the thickness of its parietes, and in an inverse ratio to the extent of its pulsations.

In the natural state of the heart, its impulse is but slight, and in persons of a certain degree of obesity it is not to be distinguished; but if the organ be slightly thickened, and its movements quickened by nervous agitation, running, palpitations, or febrile action, then its impulsions become exceedingly evident: the shocks are still greater, if the hypertrophy be considerable; they then distinctly elevate the head of the observer when applied to the chest. If the hypertrophy be extreme, the impulsion is affected in a progressive manner: the heart, at first, seems to swell, is applied upon a

point only to the corresponding surface of the chest, then upon its whole surface; finally, it gives its shock, and instantly recedes, to recommence the same series of movements: to this recession, described by Laënnec, Dr. Hope gives the name of *back stroke*. The impulsion is felt during the contraction of the ventricles.

The force of the impulsion is always proportionate to the thickness of the parietes of the heart: if the hypertrophy be great, the shock is great also; if the walls of the heart be thin, no impulsion whatever can be distinguished.

When the heart is in a state of simple or concentric hypertrophy, its impulse is felt in the precordial region only, and sometimes even in a small part of it; if, however, the hypertrophy be eccentric, or combined with dilatation, the heart then acquires the largest possible proportions, and the shock of its pulsations may be felt all over the chest. It is in the latter case that the impulsions are so great as to communicate their vibrations to the person of the patient, and even to the bed on which he is placed.

All causes by which debility may be produced diminish the impulsion of the heart's movements,—as repeated bleedings, continued diarrhœa, low diet, digitalis, &c. Even in every marked hypertrophy of the organ, a severe dyspnœa may produce the same effect, particularly when the difficulty of breathing arises from pneumonia, pleurisy, œdema of the lungs, a paroxysm of asthma, or the congestions which form during the agonies of death. Nothing, therefore, should be concluded from an examination under such circumstances.

3. *Rhythm of the Beatings of the Heart.*—By rhythm is meant the order of succession in which the contractions and dilatations of the heart occur, and the relative time occupied in effecting each movement.

It is, perhaps, impossible to determine the size of the heart, compared to the rest of the body: usually, Laënnec asserts, it is a little less, or equal, or it slightly exceeds, that of the closed hand of the subject. The parietes of the left ventricle are almost double the thickness of those of the right, and their tissue is firmer and more compact than that of the rest of the muscles of the body. The walls of the left ventricle should not depress upon being cut into. The right ventricle is somewhat larger than the left, its *carneæ columnæ* are more voluminous, and a section of the parietes of the cavity causes them to fall inwards upon it.

If the finger be placed upon the radial artery, and the stethoscope be applied at the same time to the region of the heart, the following phenomena may be readily distinguished, if the organ be in good proportions.

The instant the artery strikes the finger, a slight elevation is communicated to the ear through the stethoscope, by a movement of the heart: this is accompanied by a dull sound. The isochronism of the movement and sound, with the elevation of the artery, shows distinctly that the phenomena are due to the contraction of the ventricles, since the arteries cannot be elevated except by the ventricular systole.

Immediately after this ventricular movement and sound, a louder noise is heard, the duration of

which is considerably less than of that just described: it is unaccompanied by any sensation of impulsion, or shock, and is similar to that produced by the clacking of a valve, the smacking of a small whip, or the lapping of a dog. This sound Laënnec attributed to the contraction of the auricles: we shall presently examine the correctness of this opinion.

Directly following the second sound, there is a period of repose, the duration of which is equal, or nearly so, to the second sound itself. At the termination of this repose of the movements of the heart, the ventricular contractions recommence.

According to Laënnec, the rhythm, or relative proportion of time, occupied by the beatings of the different parts of the heart, and the period of repose, is as follows; and, for the purpose of elucidating this subject, let us suppose that the successive movements and repose occur in one second: that time would be thus divided:—

The impulse and first sound, caused by the ventricular contraction, half a second.

The second or louder sound, caused by the auricular contractions, quarter of a second.

The period of repose, quarter of a second.

Such are the succession of phenomena which may be distinguished by auscultation. The cause of one of the sounds is, however, still a subject of dispute. Laënnec asserted, that the feeling of slight impulse, accompanied by the first, or dull sound, was caused by the contraction of the ventricles: this is accorded to him; for the simultaneous elevation of the arterial pulse incontestably proves it. He ascribes the second, or clicking

sound, to the contraction of the auricles: this is denied by Dr. Hope; and I think he has succeeded in establishing his refutation, and in proving its real cause.

Dr. Hope examined the movements of the heart in several living animals, of various sizes, after depriving them of sensation, and keeping up respiration by artificial means. He saw, upon opening the pericardium of the ass, that the auricles first contracted by a vermicular and slight action, which was continued to the parietes of the ventricles; that when that movement arrived at the ventricles, they instantly contracted, and propelled the blood into their emerging vessels; the point of the heart then striking the parietes of the chest. Dr. Hope, at the moment of the ventricular contraction, applied the stethoscope to the naked surface of the ventricles, and distinctly felt the impulsion of their movement, and heard the first sound, and found that they accorded, or nearly so, with the elevation of the arteries; proving that the first sound, the impulsion, and the arterial pulsation, depended upon the contraction of the ventricles. The moment the ventricles ceased to contract, they receded from the parietes of the chest, and became dilated and swollen. The stethoscope, according to Dr. Hope and numerous assistants, then communicated the second sound, — proving, if the facts were well observed, that it did not depend upon the auricular contraction, but upon the ventricular dilatation. It was then observed that the heart remained for a short time in a state of repose, during the latter part of which the auricle again commenced its vermicular contraction, which was

immediately again followed by that of the ventricles, &c.

The time occupied by these states will now be as follows:—The ventricular contraction, half a second; the ventricular dilatation, a quarter of a second; the period of repose, a quarter of a second, the latter part of that period being interrupted by a certain quantity of blood entering into the ventricles, in consequence of the contraction of the auricles.

Such are the leading points of Dr. Hope's theory of the movements of the heart; for a more complete account, I recommend to you the perusal of his excellent work.

Impressed with the importance of this theory, especially in a pathological point of view, I repeatedly attempted to verify these experiments. I perfectly satisfied myself that the contraction of the ventricles coincided with the first sound, the impulsion, or shock, and the elevation of the pulse; but I confess I could not as satisfactorily ascertain that the dilatation of the ventricles occasioned the second: I much doubt even whether I heard the sound at all; but it was evident that the successive movements of the heart were performed in accordance with Dr. Hope's description; and it was clear, also, that the contractions of the auricles were too slight to produce that sound. Although, therefore, I could not succeed in observing all that Dr. Hope describes, yet I fully believe his explanation to be the most rational. This theory singularly corroborates the description of the symptoms of the diseases of the heart given by Laënnec, since it

not only does not invalidate a single sign, but confirms and explains them all.

The rhythm of these movements is modified by the disease; thus, if the ventricles be in a state of moderate hypertrophy, their contractions are less sonorous, and are of longer duration than usual, the period of repose being still well marked. If, however, the hypertrophy be considerable, the ventricles occupy a still longer time in contracting; these contractions are at first obscure, deep-seated, but augment gradually, elevate the ear, and terminate in a shock, unattended by any noise, or, if any, it is a slight murmur, like that of respiration; the second sound is then of short duration, and nearly inaudible: sometimes it cannot be heard: the period of repose is then lost. In extreme cases of hypertrophy, no sound whatever is heard, except, perhaps, the slight murmur before described; but the ventricular contractions are manifested by a strong rolling movement, as if a large mass of solid flesh were irregularly and tumultuously revolving within the cavity of the chest. The second sound, in this case, is heard with great difficulty; now and then, however, two or three ventricular dilatations succeed each other rapidly and convulsively.

When the parietes of the ventricles are thin, the interval of repose is lost; the sounds of their contractions are louder than natural, and approximate in character to that of their dilatations; so that these movements often cannot be distinguished. The shock of the ventricle is also diminished: indeed, it may be said not to exist. If to these signs be superadded an increased extent of pulsa-

tion over the parietes of the chest, it may then be inferred that the walls of the ventricles are not only thin, but that their cavities are enlarged.

4. *Of the Sounds produced by the Movements of the Heart.*

The sounds produced by the movements of the heart cannot be heard by the patient himself, except he be suffering from febrile action, or nervous palpitation, or in the case of these sounds being heard at a certain distance from the chest.

If the stethoscope be applied to the precordial region, two distinct sounds may be heard in the natural state; the one clear, short, like the clicking of a small valve, and corresponding to the dilatation of the ventricle; the other dull, longer in duration, coinciding with the beating of the pulse and with the sensation of impulsion previously described, and indicating the contraction of the ventricle.

The sounds heard in the right precordial region are produced by the right ventricle; those in the left by the left ventricle. In the natural state the sounds of both ventricles are similar; in their diseased state, they are often very dissimilar.

These sounds are the only phenomena which the beatings of the heart present, when they are heard on any other point of the chest than in the precordial region; for the shock, or impulsion, is generally confined to the latter space alone, except in cases of hypertrophy with dilatation of the ventricles.

The sounds produced by the beatings of the

heart are always the loudest when the parietes of the ventricles are thin, and the impulsion feeble. When the ventricles are in a state of moderate hypertrophy, their contractions produce a dull sound like an inspiratory murmur, and the clicking noise of their dilatations is less than natural. If the hypertrophy be extreme, the sound of the ventricular contraction is lost; a shock only is felt, and the dilatation is also to be distinguished with difficulty.

When the parietes of the ventricles are thin, the sounds of these contractions are clear and loud, and similar to that of their diastole. If their cavities be also dilated, the sounds become still more similar and equal in intensity; so that finally they cannot be distinguished from each other.

In the natural state, these noises are always most evident in the precordial region, becoming feebler at the remoter parts of the chest. In some diseased states of the heart, they may be louder at other parts; as we shall hereafter describe.

It frequently happens that, in cases of hypertrophy, when nothing is felt in the precordial region but a noiseless impulse, and where the sound of the diastole of the ventricle can scarcely be heard, the latter can be distinguished under the clavicles, and even on the back; and in almost all slight cases, it is more evident in these situations than in the precordial regions, especially in persons possessing thin and narrow chests.

Of certain Anormal Sounds produced by the Action of the Heart and Arteries.

Bruit de Soufflet.—The heart, either in the systole or diastole of its cavities, and the arteries in their diastole only, occasionally produce a peculiar sound, often exactly similar to that of the blowing of a bellows, from which analogy Laënnec has denominated it “*bruit de soufflet*.” There are certain modifications of this sound; hence he divided them into three species.

1. *Bruit de soufflet*, properly so called.

2. *Bruit de scie ou de râpe*.

3. *Bruit de soufflet musical ou sibilant*.

1. *Bruit de Soufflet*, properly so called. Laënnec has most singularly stated that the bellows sound occurs only during the diastole of the heart and arteries; when, in fact, it occurs frequently during the systole of the ventricles. This sound, although generally precisely analogous to that produced by blowing a bellows, yet sometimes changes in its character both in the heart and arteries: it then becomes similar to the continued murmur of the sea heard at a distance, or to that caused by holding a large univalve convoluted shell to the ear. This latter sound is sometimes heard in the carotid and subclavian arteries on one side, while the ordinary *bruit de soufflet* occurs in those of the opposite side. It is isochronous with the arterial diastole, and most frequently exactly circumscribed by the caliber of the artery or ventricle producing it; sometimes it is, however, more diffused.

Bruit de Scie ou de Râpe.—This sound is much rougher, and, as its name indicates, is like those

caused by the sawing, rasping, or filing of wood, when heard at a distance.

Bruit de Soufflet musical ou sibilant.—The musical or sibilating bellows' sound is, according to my experience, very rare: many of you have heard it very distinctly lately, in a lad in this hospital. This sound is generally preceded by the ordinary bruit de soufflet: it was so in the case I have just alluded to. Its characters vary: it is sometimes like that caused by the wind blowing through a key-hole, or to the resonance of a metallic cord which vibrates a long time after it has been touched.

These sounds, although not very intense, are yet perfectly appreciable; sometimes they consist of two or three notes: the instance I have mentioned consisted of two only.

The musical or sibilating bellows' sound is very rarely produced by the heart, and never in a very distinct manner.

The bruit de soufflet may exist in the heart and arteries with or without an increase of the force of their impulsion.

The bruit de soufflet may manifest itself at once in the cavities of the heart, and to a great extent of the arteries. Nothing is more uncertain than its duration, for it may be heard in the left carotid, and not in the right, on one day; it may be reversed, or altogether absent, on the following; and so of the rest of the arteries. The musical sound continued in the right inguinal artery of the boy I have mentioned many days after it had disappeared from the carotids.

*Proximate Causes of the Sounds produced by the
Movements of the Heart and Arteries.*

It is not my intention to enter here into any elaborate detail of the various opinions entertained of the efficient causes of the sounds produced by the movements of the heart and arteries, because the time permitted me in delivering this portion of the course is not more than sufficient even to state all the facts, without entering into hypothetical speculations, and because also these speculations are of minor importance, compared to a perfect knowledge of the facts themselves; yet it may be necessary to say a few words upon this subject, although I shall be as brief as possible.

What are the efficient causes of the sounds produced by the movements of the heart in its natural state; and what are the causes of the anormal sounds denominated *bruit de soufflet*?

In relation to the first question, there are two sounds to explain: the first, dull, and corresponding to the contraction of the ventricles; the second, louder, and synchronous with the dilatation of the same cavities.

There are two elements which enter into the consideration of these questions; which are, that these sounds can only be produced by the action of the solid structure of the heart, or by the blood entering into its cavities; or, perhaps, by the united movements of one and the other.

I believe that the first sound is caused by the point of the heart striking against the ribs. One, however, has thought that it was produced by the rushing of the blood into the ventricles during their

dilatation; a second supposed that it was during that dilatation that the point of the heart arose to strike the ribs; a third, that the closing of the mitral and tricuspid valves, during the contraction of the ventricles, was a sufficient cause to produce the sound; a fourth, that it was effected by the rushing of blood into the arteries; a fifth, that it was to be accounted for by the collision of the particles of blood itself in the ventricles, and, finally, that the contraction of the muscular fibres is the cause.

I believe, also, with Dr. Hope, that the second sound is caused by the ventricular dilatation; at least, it is certainly coincident with it. Laënnec supposed it to be formed by the auricular systole: we have, however, sufficiently discussed that question, and it is unnecessary to revert to it. But, as there have been a variety of opinions as to the proximate cause of the first sound, so have there been also of the second: thus it has been supposed to be caused by the contraction of the ventricles; by the flapping of the mitral and tricuspid valves against the sides of their corresponding ventricles; by the striking of the blood against the parietes of the pulmonary artery and aorta; by the movement of the base of the heart against the chest; by the action of the arterial blood in closing the semilunar valves; by the impulse given by the blood in dilating the ventricles during the contraction of the auricles; and, lastly, by the mere muscular action of the parietes of the ventricles in their dilatation.

Such, gentlemen, is a summary of the opinions entertained upon this subject. I can here enter into no further details, but refer you to the ap-

pendix of Dr. Williams's work upon Diseases of the Lungs and Pleura. Recollect, however, that these *facts* are proved: — that the impulse of the heart, the first sound, and the pulsation of the arteries, occur at the same moment, and therefore must be caused by the ventricular contraction; also, that the second sound, and a part of the time between two successive elevations of the pulse, are isochronous with the period of the dilatation of the ventricles, and that that sound is caused, in all probability, by the ventricular diastole.

The anormal sound denominated “bruit de soufflet” is equally difficult of explanation. When, however, it, or any of its modifications, exist, either in the heart or arteries, it may always be inferred that there is a mechanical obstruction to the current of the blood, or that these organs are under the influence of some peculiar and inexplicable state of the nervous system. If the sound be caused by mechanical obstruction, it is permanent, since the cause is so; but if it be the consequence of nervous disorder, it disappears with the affection that has caused, or, at least, is concomitant with it.

The efficient cause of the bruit de soufflet must be sought for in the parietes of the heart and arteries, or in the blood flowing within them. Laënnec, after adverting to Wollaston and Erman's discoveries of the sounds produced by muscular movements, some of which are precisely similar to the bruit de soufflet, attributes the bellows sound to a vibratory action of the heart and arteries. Dr. Hope, and others, believe the sound to be caused by the movements of the blood; but, in the latter case, it is impossible to explain why it should

occur in one carotid, and not in the other ; or in the subclavian, and not in the carotids ; or in the inguinal artery, and not in the subclavian ; or that, in different days, or in different parts of the same day, it should so frequently change its situation. We cannot suppose that the blood changes its qualities so rapidly, or that it should differ so materially in vessels of similar caliber and distance from the heart, as to produce the sound in one carotid artery, for instance, and not in the other.

Frémissement Cataire.

Laënnec gives the following description of this sensation, which is occasionally detected by the application of the hand upon the heart and arteries.

The *frémissement cataire* is almost exactly similar to the tremulous sensation produced in passing the hand upon the skin of a cat while she is purring ; it may also be assimilated to the feeling caused by gently rubbing a hard brush over the palm of the hand covered by a glove. This tremor is commonly felt over the precordial region, or a part of it ; or over the whole surface of the chest ; and frequently, also, in the course of the larger arteries.

The arterial purring tremor is usually similar to that already described, and is exactly bounded by the caliber of the artery. It is felt by a slight pressure upon the vessel ; but if the force be increased, the trembling diminishes : in this case it is saccaded, and not continuous. Sometimes, and especially in the carotids, the purring extends beyond the dia-

meter of the artery, and is superficial, continuous, and not saccaded. The vessels in which the *frémissement cataire* is commonly found, are the carotids, subclavians, brachials, and inguinals. Laënnec observes that it is rare in the ascending aorta, but I certainly have not found it so: it is, however, infrequent in the abdominal aorta, and it can only be felt there by considerable pressure. The purring is but little sensible in the smaller arteries; yet when it exists in the heart, or a great blood-vessel, or even when the bruit de soufflet is formed in them without the *frémissement cataire*, the pulse often presents a slight tremor corresponding to the arterial diastole.

The *frémissement cataire* of the heart and arteries is constantly accompanied by the bruit de soufflet, and appears to be caused by it. According to my experience, the intensity of the trembling is always proportionate to the loudness of the bellows' sound. Laënnec, however, asserts that it is not always so, and, consequently, infers that the two phenomena are not determined by the same cause. I cannot help believing, after several years' observation, that, considering that these sensations are always perfectly synchronous, and that the force of the *frémissement cataire* is always in a direct ratio to the loudness and roughness of the bruit de soufflet; it is, in fact, but one phenomenon, rendered evident to the tact, by the vibrations communicated to the hand, and to the ear, by the sounds they occasion.

The two sensations, then, have, I presume, identical causes; they may arise from organic obstruction to the circulation, or from nervous agitation.

In the latter case, the purring is as uncertain as to its seat and duration as the bruit de soufflet. If the cause be organic, like that sound it is always present in the same situation.

Of the Beatings of the Heart heard at some distance from the Chest.

It requires the application of the ear, or the stethoscope, to the surface of the chest, to hear the sounds produced by the pulsations of the heart. It very rarely occurs that these movements can be heard at a distance from the thorax. It sometimes, however, happens that these noises may be distinguished at a certain distance from the body. I have heard them two or three times, and, in one instance, at five or six yards from the patient.

I have also occasionally heard the bruit de soufflet at a distance from the individual; and have little doubt that, if this sound were constantly attended to in this point of view, it might frequently be heard when intense.

The cause of this phenomenon is extremely obscure. Laënnec believed it depended upon the presence of air in the pericardium, or that air might even be formed in the cavities of the heart during the agonies of death, so as to occasion this sound. I mention it as a singular circumstance, that, in the cases of the beatings of the heart heard at a distance by myself, they occurred in females who were at that period under the influence of the catamenial discharge. I perceive, however, no connection of cause and effect between these two events.

Laënnec mentions another cause of this phenomenon, which is doubly interesting, on account of the fact itself, and on the probability of his having noticed it on his death-bed. It is the last page of his work, which I shall transcribe entire.

“ At the moment they brought me the last proof of my work, indisposed for some days, I have observed in myself the phenomenon of the noise of the heart, sensible also to those around me ; and I was enabled to distinguish an evident cause, altogether physical, and which, being of the same nature as that we have already spoken of, ought certainly to be much more frequent.

“ I had just been bled in the foot, and placed myself in bed, where I rested a few minutes in a sitting posture, the back but slightly supported, and the head upright : finding myself very easy in this position. Suddenly I felt the contractions of my heart (a very rare circumstance with me), and I heard them also very distinctly. These contractions were regular, not unusual in their force, but had the frequency which a slight degree of fever occasioned. It seemed to me that, at each contraction, the heart slightly repelled a veil moderately tightened. I examined the region of the stomach, which I found very distended by gas, and strongly resonant by the slightest percussion. A person placed his head about six inches from my chest, and heard distinctly the beatings of my heart. From thence I began to think, that a certain degree of flatulent distention of the stomach, from its lying in such close contact with the diaphragm, might produce the phenomenon of which I speak. An instant after, I doubted no more, for an eructation of gas occasioned it to disappear.”

LECTURE XXII.

DISEASES OF THE HEART.

General Classification. — Tabular View of Diseases of the Substance of the Heart, and of the Valves. — *Hypertrophy* of the Substance of the Heart. — Simple Hypertrophy of left Ventricle — of right Ventricle — of both Ventricles. — *Dilatation* of the Ventricles. — *Hypertrophy*, with *Dilatation* of the Ventricles. — *Hypertrophy*, with *Dilatation* of the Auricles.

Diseases of the Valves. — Mitral Valves. — Cartilage, or Ossific Deposits. — Aortic Valves. — Ossific Deposits. — *Vegetations* on Valves, and internal Parietes of the Heart — verrucous — globular.

Causes of Hypertrophy and Dilatation of the Heart. — Proposition — exemplified. — First, Obstructions from Chronic Diseases of the Lungs. — Second, from diseased Aortic Valves. — Third, from diseased Mitral Valves. — Fourth, from a contracted or dilated Aorta. — Fifth, from habitually bent Positions — from a Malconformation. — moral Causes — Plethora — Anemia.

HAVING thus, gentlemen, given you an account of the local signs of the diseases of the heart, we now proceed to the description of the affections: —

1. Of the substance of that organ, and its valves.
2. Of the pericardium.
3. Of the large blood-vessels.
4. Of the nervous disorders of the heart and arteries.

And, finally, of malconformations of the heart.

In describing the diseases of the heart and its valves, I shall proceed according to the following arrangement: —

Tabular View of the Diseases of the Substance of the Heart.

Hypertrophy, simple	{	left ventricle.	
		right ventricle.	
Dilatation, simple	- {	left ventricle.	
		right ventricle.	
Hypertrophy, with dilatation of ventricles.			
Hypertrophy, with dilatation of auricles.			
Valves	{	mitral	- -
		semilunars of aorta	
			1. cartilage, bone.
			2. vegetations. { verrucous.
			globular.
Carditis.			
Ulcers of the heart.			
Rupture.			
Hardness of the substance.			
Softness of the substance.			
Surcharge of fat.			
Fatty degeneration.			
Cartilaginous, bony, and other adventitious deposits.			

Hypertrophy of the Substance of the Heart.

I shall, in the description I am about to give of the morbid anatomy of the heart, adhere closely to the texts of Laënnec, Bertin, and Kreyseg, because of their extreme accuracy : these descriptions will also be at once verified and illustrated by the series of preparations I shall place before you.

By *hypertrophy*, is meant an increase of nutrition of the substance of the heart, the augmentation of the thickness of its muscular structure, and consequently of the parietes of the cavities of the organ : this thickness is usually combined with increased firmness of the muscular fibres ; sometimes, however, though rarely, they are softer than in their natural state.

Hypertrophy may affect the parietes of either cavity of the heart ; thus it may exist in one ventricle or in both, and in the auricles also, at the same time, or in a single auricle.

Simple Hypertrophy of the left Ventricle. — The parietes of this ventricle occasionally acquire an

extraordinary thickness ; sometimes, at the base of the ventricle, even to one or two inches. This thickness gradually diminishes from the base to the point, so that at the latter part it may not measure more than a line. The point, however, occasionally participates in the same condition, and increases to three or four lines. The carneæ columnæ are usually augmented in volume, although not always. The interventricular septum generally, though not invariably, is affected in the same manner. The hypertrophy may vary in different parts of the same ventricle, so that it may affect exclusively the point, the base, the septum, the carneæ columnæ, or the external parietes. In proportion to the increased thickness of the walls of the ventricle, its cavity diminishes in its capacity : Laënnec had seen it so small as to be incapable of holding an almond enclosed in its shell. The right ventricle, in this case, is flattened along the interventricular septum, and does not descend so nearly to the point of the heart as usual ; and, in extreme cases, it appears even to be hollowed out of the parietes of the left ventricle.

It is from the increased thickness of the parietes of the ventricle being directed towards the interior or centre of its cavity, that Bertin has denominated this form of the disease *concentric hypertrophy*.

Simple Hypertrophy of the right Ventricle. — The parietes of this ventricle become thicker and firmer than usual ; they do not collapse upon their cavity upon a section of them being made. The thickness of the muscular structure is nearly uniform, although it is generally greatest towards the base of the ventricle. The carneæ columnæ are

always very large. The size of the cavity is rarely much diminished, because the thickness of the walls of the ventricle is not often very considerable. Laënnec never found the thickness above four or five lines; Bertin describes a case in which it was from eleven to sixteen lines. I present you here, gentlemen, a preparation in which the parietes of the right ventricle are fully three-quarters of an inch thick, and its cavity is so diminished as to be capable only of holding a small pea: it is the heart of an infant, in whom the pulmonary artery was obliterated at its origin.

Hypertrophy of both Ventricles. — When hypertrophy attacks both the ventricles, they each descend to form the point of the heart. The other anatomical characters are the same as above described.

Dilatation of the Ventricles. — This affection is the *passive aneurism* of Corvisart. The cavities of the ventricles become now enlarged, and their parietes much thinner than natural. The muscular tissue is softer, and generally pale; sometimes it is of a violet colour; occasionally it assumes a yellow tint. The softening of the fibres is often so great, especially of the parietes of the left ventricle, that they may be broken down easily with the fingers. The thickest part of the left ventricle may now measure no more than two lines, and its point not more than half a line. The point of the right ventricle often becomes thinner still, seeming, indeed, to consist of the external and internal membrane of the heart only, separated by a small portion of fat. The *carneæ columnæ* of the left ventricle appear as if increased in number, although they are extremely thinned, presenting, by their crossing

each other, a finely reticulated character. The intervertebral septum loses less of its thickness and firmness than the rest of the parietes of the heart.

This dilatation and thinning may affect a single ventricle, or both at the same time. When one only is dilated, its point descends lower than usual, although the increase of its cavity is in the direction of its transverse diameter rather than of its length. When both ventricles are affected, the heart is rounder in its form, and is almost as large at the point as at the base.

Burns believed that rupture of the heart might be caused by its dilatation; Laënnec conceived also such an event to be possible; Dr. Hope mentions an instance of such an occurrence, and cites another case from Dr. Williams. I present you here with a preparation of a rupture of the upper part of the left ventricle. It is, however, rather a consequence of softening, or ulceration of the muscular fibres, than of excessive dilatation.

Laënnec has averted us from the mistake which might be committed, by confounding the dilatation of the heart with its distention from blood. In the latter case, the moment the cavities of the heart are opened, the distending blood escapes, and the organ collapses to its natural size: this does not occur if there be really dilatation.

Hypertrophy, with Dilatation of the Ventricles.—This combination constitutes the *active aneurism* of Corvisart, and the *eccentric hypertrophy* of Bertin.

Hypertrophy with dilatation of the ventricles is a very common affection; it is much more frequent than simple dilatation, and still more so than

the concentric or simple hypertrophy. This disease may affect either of the ventricles, or both simultaneously. In the latter case, the heart often acquires an enormous volume. The muscular fibres become firmer. The heart is rendered rounder at its point. In some cases the ventricles are dilated, and their parietes are of their natural thickness, being what Bertin has considered dilatation of the ventricles, with hypertrophy in the direction of the extent of their surfaces. Dr. Hope states that this variety was unknown to Laënnec; but, although no specific name was given to it by him, yet the following passage clearly denotes that he was aware of it, for in speaking of hypertrophy with dilatation of the ventricles, he says, "A un degré médiocre les ventricules sont dilatés, et leurs parois semblent seulement n'être pas amincies."

It happens, though very rarely, that some parts of the walls of the same cavity present the characters of hypertrophy, and others those of dilatation.

Hypertrophy, with Dilatation of the Auricles. — It is very easy to mistake organic dilatation of the auricles with their simple distention from blood; it is as easy, also, to correct the error, by compressing the distended cavities, and thereby forcing the blood from them into their communicating cavities or vessels. If they are merely distended, the auricles will then return to their natural volume; if they be permanently dilated, they preserve their size. Laënnec also observes, that if their cavities be fully distended, their parietes will appear tense, and the colour of the blood is seen through them; but if they be permanently dilated, their walls are not so tense, they appear capable of holding more

blood, and the colour of that fluid can no longer be distinguished.

The dilatation of the auricles, although not so rare as Laënnec believed, is yet not so common as that of the ventricles. This dilatation is generally accompanied by hypertrophy, or by hypertrophy and dilatation, of the ventricles. Sometimes the left auricle may be dilated, while its corresponding ventricle is in a healthy state. Laënnec states also, that he never met with evident dilatation of the auricles without a certain degree of hypertrophy. There is before you a specimen of hypertrophy of the left auricle without dilatation. It requires a certain degree of habitude to distinguish hypertrophy of the auricles; for as their parietes are very thin, it might escape the observation of an unpractised observer even if they were doubled in thickness. The preparation I hold in my hand shows the walls of the left auricle to be nearly three lines thick.

The following combinations of organic changes in the parietes and cavities of the heart most frequently occur:—

1. Hypertrophy with dilatation of the left ventricle, with simple dilatation of right.
2. Hypertrophy with dilatation of the left ventricle, with simple hypertrophy of the right.
3. Hypertrophy with dilatation of right ventricle, with simple dilatation of the left.
4. Simple hypertrophy of right ventricle, with dilatation of the left.

There are other varieties, which we shall hereafter mention when we consider the causes of these affections.

It occasionally, although very rarely, occurs that a partial dilatation of the cavities of the heart takes place — a dilatation which presents all the characters of aneurism. Corvisart mentions a singular instance of this in a negro, in whom the partial dilatation of the left ventricle was so large as to form a tumour of the size of the ventricle itself. My friend Mr. Langstaff possesses two beautiful specimens of these partial dilatations; the one is of the upper portion of the left ventricle, forming a tumour of the size of a large walnut, which presents itself between the base of the ventricle and its corresponding auricle, the coronary artery crossing it; the other is a still larger aneurism of the left auricle, situated between the appendix and the mitral valves: it contains a considerable quantity of fibrinous substance, precisely similar to that which is so commonly found in arterial aneurisms.

Diseases of the Valves.

The valves of the right side of the heart have been rarely seen diseased. Occasionally, however, they have been noticed. Vieussens, Bertin the elder, Horn, Crewel, Burns, Laënnec, Bertin the younger, Drs. Hope and Latham, all give instances of cases which they have observed. It must be noticed, however, that the indurations of the valves which they describe are almost all of the cartilaginous character, and that bony concretions have very rarely been seen. In many of these cases, also, a free communication has existed between the auricles, in consequence of the foramen ovale being open.

The cause of the frequency of disease of the valves of the left side of the heart yet remains in great obscurity. Corvisart has supposed that it arises from the fibrous structure partly forming these valves being more complete ; and consequently he infers that that structure is necessary to the cartilaginous or ossific deposit. Others think that it arises from some peculiar influence of the arterial blood. I am inclined to the latter opinion, because we see that the parietes of all the vessels carrying venous blood, including the right side of the heart, are rarely subject to cartilaginous or osseous induration, while nothing is more frequent than their formation in the valves of the left side of the heart, and between the internal and middle tunics of the vessels carrying arterial blood.

Cartilaginous Deposit in the Mitral Valves. — When cartilage is deposited in the mitral valves, it is always situated within the duplicature of the membrane forming it. The base of the valve is generally the seat of the adventitious matter ; the auriculo-ventricular opening then becomes contracted, and assumes various forms, but usually that of a narrow chink or slit. The edges of the orifice are commonly smooth and polished, and of firm structure. Occasionally the cartilaginous matter occupies the free edge, the middle, or even the whole, of the valve.

Ossific Deposit in the Mitral Valves. — If phosphate of lime be deposited, it is also in the duplicature of the membrane forming the valve, and may be situated, like cartilage, either at the base, point, or throughout the valve. The bony matter soon perforates the membrane covering it, and

forms irregular granular projections, or asperities, bathed by the blood. This osseous deposit never presents the cancellar appearance of healthy bone: it is usually whiter, and breaks down with greater facility. The mitral orifice is often extremely contracted by the ossific formation: it becomes a small and irregular slit, or an opening more or less round, or sometimes even a canal, which has been seen bending like the carotid in its passage through the temporal bone. The chordæ tendineæ have, although very rarely, been observed ossified.

Ossific Deposit in semilunar Valves of the Aorta.—The ossific matter generally commences to be deposited in the tubercula Arantii, then at the base, then on the free edge, and, finally, on the whole structure of the valve.

When the ossification affects the free edges of the valves only, their movements are yet tolerably complete, and the flow of blood through them is not much impeded; but when their bases and other parts become diseased, the valves unite, curve inwardly or outwardly, like certain shells, or sometimes a single valve is curved in one direction, and another in a different. They then become nearly immoveable, and remain in the position they have taken: thus they may fall inwards towards the heart, or they may lie along the internal surface of the aorta, or they may be placed permanently across the origin of that vessel. I present you with a preparation in which the natural orifice between the valves is reduced to a very small size; but, at the base of one of them, ulceration and perforation has taken place, so that two distinct,

although small, columns of blood must have passed into the aorta instead of one. I need hardly state that the obstruction of the circulation must be in proportion to the diminution of the opening left by the diseased state of the valves.

Cartilaginous or ossific deposits in the valves have been supposed to depend upon inflammation of the lining membrane of the organ, or of the pericardium investing it, or of the substance of the heart itself, or from any cause rapidly and frequently increasing the circulation, or from some peculiar diathesis. Nothing, however, is positively known upon this subject.

Vegetations formed on the Valves and Parietes of the Cavities of the Heart. — There are two forms in which fleshy vegetations grow upon the valves and internal surfaces of the heart; the first is that of warty excrescences, which have been called, by Corvisart, *vegetations* on the valves; the second, that of globular bodies, often of considerable size, which have been denominated, by Laënnec, *globular vegetations*. The latter author gives the following description of them: —

Verrucous Vegetations on the Valves. — These often present a very similar appearance to ordinary or to venereal warts; hence they have also been named *verrucous vegetations*. They bear occasionally a striking resemblance to a small raspberry. Sometimes these bodies are greater in length than breadth, and have the form of small irregular cylinders, more or less approximated to each other. They have been seen covering the valves, chordæ tendineæ, and the internal surfaces of the auricles; rendering them rough and granulated. I have ob-

served them extending only in a single row: their length is rarely greater than two or three lines.

The colour of these vegetations, although sometimes whitish or yellowish-white, is usually that of muscular fibre, or a little lighter. Their texture is fleshy, sometimes similar to venereal warts, although less firm. Their adhesion varies, for it may be so strong that they cannot be torn off, or it may be so slight that they may be easily separated and scraped off by the handle of the scalpel or the finger-nail. In the latter case the vegetations are soft, and of very humid texture.

These substances have been found on all the valves, and on the internal surface of the auricles and ventricles.

Corvisart believed the verrucous vegetations to be of venereal origin: there are no proofs whatever in support of this supposition. Laënnec conceived them to be formed by the organisation of coagula of blood adhering to the valves and the parietes of the heart: his reasons were, that their texture closely approached to that of the most compact polypiform concretions of the blood; nay, even that a slight coagulum is occasionally found within them. Bertin and Bouillaud attribute their formation to the organisation of false membranes, produced by inflammation of the membrane lining the cavities of the heart and forming the valves.

Globular Vegetations. — The size of these vegetations varies from that of a pea to a pigeon's egg. I present you a specimen, where there are two attached to the mitral valve, of nearly the latter volume: their form is spheroid, ovoid, or round: they are hollow. The external surface of

these cysts is smooth, the internal is rougher : their parietes are about half a line in thickness, and have somewhat a firmer consistence than boiled white of egg. The matter contained within them varies ; it is sometimes similar to half liquid blood, containing also a few coagula of that fluid — it is opaque, and occasionally of a pale violet colour, or of the colour of lees of wine, and of a pultaceous consistence ; finally, it is like thick pus, or to a substance precisely similar to the polypiform concretions which are frequently found interlaced between the carneæ columnæ. These cysts always adhere by a pedicle which is slightly transparent, and occasionally contains small clots of blood.

Laënnec never found the globular vegetation except in the ventricles, and in the sinuses of the auricles. The two I present to you are attached, by a common base, to the mitral valve : the disease occurred in a girl of fourteen years of age. I believe it to be a very rare affection.

The same opinions have been entertained by Laënnec, by Bertin, &c. of the formation of this disease as of the preceding species.

Causes of Hypertrophy and Dilatation of the Heart.

I have thus, gentlemen, described the morbid anatomy of the heart, as far as relates to the increased thickness of its parietes, to the dilatation of its cavities, and to the diseases of the valves : I have preferred this order, for these affections are intimately connected with each other, and because

valvular obstruction is so commonly, although certainly not the only cause of hypertrophy and dilatation of the heart.

To understand the causes of hypertrophy and dilatation of the heart, the following principles must be advanced: that whenever any frequent necessity exists for an increased force or exertion of muscular power, the muscles increase in their size to a certain extent, if the subject be healthy. Thus we see that in the workman who wields the heavy sledge-hammer, the arms acquire an extraordinary volume; that the same occurs to the waterman; that the muscles of the back and shoulders of the porter become greatly developed; that the legs of the chair-porter become of a great size.

If we apply this principle to the movements of the heart, we may assert that, whenever any permanent or even frequent obstruction exists to impede the current of the blood, that obstruction creates a necessity for an increased force or power of muscular action to overcome it, otherwise the circulation would cease. The muscular parietes of the heart then increase in thickness, or, in other words, become in a state of hypertrophy.

But, as in the aged, and in persons of lax and weak fibre, or of delicate constitutions, the constant application of physical force only tends to diminish the size and firmness of the muscles, and exhaust their power; so I apprehend that in these individuals the parietes of the cardiac cavities do not increase in thickness and in force to overcome the resistance afforded by the obstruction, but yield, dilate, and become thinner, from the disten-

sion produced by the blood accumulating within them. These constitute the cases of simple dilatation of the cavities of the organ.

Mixed cases, however, frequently occur, in which nature lends her aid to overcome the obstruction, by thickening the parietes of the heart, while, at the same time, the cavities dilate; for if the heart, although thickened in its walls, has not sufficient power to send the due quantity of blood through its orifices at each of its contractions, that fluid must accumulate in its cavities, and ultimately dilate them. These, then, are cases of hypertrophy with dilatation, or the eccentric hypertrophy of Bertin.

Let us now proceed to the particular cases of obstruction to the current of the circulation, and their effects upon the different cavities of the heart.

1. *Obstructions caused by Chronic Diseases of the Lungs.* — It is scarcely necessary to state that the lungs should be in a perfectly healthy condition to admit of a free circulation through them. If these organs become affected by chronic disease, more especially by chronic catarrhs and emphysema, then the blood is impeded in its transit from the pulmonary artery into the pulmonary veins, obstruction is established from the engorgement of the capillary vessels, and the pulmonary artery and right side of the heart become unnaturally filled with that fluid. Hypertrophy, or dilatation, or a combination of both states, now occurs of the right cavities of the heart; while the left ventricle and auricle remain in their natural condition.

You will understand, however, that these affections of the right side of the heart can only be

produced by pulmonary diseases of long standing. Probably many months — nay, perhaps two or three years — are required before such consequences visibly take place. These changes do not occur either from protracted hepatisation of the lung, or chronic effusions into the pleura, compressing that organ; for these diseases destroy life before hypertrophy or dilatation are produced. It might be supposed that the tubercular deposits would cause sufficient obstructions to the course of the venous blood through the lung; but the quantity of that fluid is then so extraordinarily diminished, that no engagement ever occurs, sufficient, at least, to require any increased force to overcome it; indeed, the heart then generally, like the rest of the muscles of the body, decreases in volume.

2. *Obstruction caused by diseased Aortic Valves.* — This obstruction, when considerable, produces the most extensive alteration in the parietes or cavities of the organ, — as you may perceive by the various preparations placed before you. Let us trace its effects.

The *left ventricle*, being placed immediately below the obstruction, becomes first affected: its parietes may be simply hypertrophied, or they may simply dilate from the engorgement of blood within it; or both states may coexist. The *left auricle* being a continuation of the ventricular cavity, becomes similarly diseased. As, therefore, the blood cannot flow freely from, but accumulates in these cavities, it follows that the four pulmonary veins cannot either freely pour their contents into the left auricle, and they consequently be-

come permanently filled and engorged to their capillary extremities. If the pulmonary veins are engorged to their extremities, it follows also that the blood in the capillaries of the pulmonary arteries passes with difficulty into those of the veins; and the pulmonary arterial branches, as well as the artery itself, becomes preternaturally filled. The right cavities of the heart, being continuations of the pulmonary artery, become also in an engorged condition; obstruction exists; their parietes are hypertrophied, or their cavities dilate, or there is a coincidence of both conditions; and the heart acquires an enormous volume.

Thus, gentlemen, an obstruction placed at the commencement of the aorta may affect the parietes and dimensions of all the cavities of the heart.

3. *Obstruction caused by diseased Mitral Valves.*

— If the mitral valves be diseased, so as to cause obstruction, the same effects will be produced in all the cavities of the heart except of the left ventricle, which is placed before the impediment. Thus the left auricle becomes gorged with blood, its walls thicken, and its cavity dilates; the four pulmonary veins and the pulmonary artery are habitually distended; the cavities of the right side of the organ are similarly affected; and hypertrophy or dilatation, or both states combined, are the results.

[Numerous specimens were here shown, exemplifying the facts of obstruction of the aortic valves causing hypertrophy or dilatation of the different cavities of the heart, and obstruction of the mitral valves causing the same effects, except that in the latter case the left ventricle was in a comparatively healthy condition.]

But, gentlemen, diseased valves are not the only obstruction to the circulation; there are others, which, because they are not so obvious, are often overlooked: so that the heart often appears enlarged and thickened without apparent cause.

Obstruction from a contracted or dilated Aorta.—The circulation of the blood cannot proceed in a healthy manner except there be a due relation between the caliber of the vessels carrying that fluid to and from the heart, and the capacities of the cavities of the organ itself. The vena cava, the pulmonary arteries and veins, are rarely diseased; but it is not uncommon to find the aorta considerably diminished or enlarged in its diameter; and either of these states becomes a cause of obstruction as much as diseased valves.

Let us suppose that the diameter of the healthy ascending aorta in the adult be an inch, the circulation proceeds with regularity; but if it be diminished to half an inch, the column of blood passing through it can only have the same diameter, or half an inch; but as the same quantity of blood has always a tendency to enter into the left ventricle, and the aorta is insufficient in breadth to discharge it, it accumulates; there is an obstruction before it in its course, the parietes of the left ventricle thicken, its cavity distends, &c. In fact, precisely the same effects are produced upon the walls and cavities of the heart as if diseased aortic valves were the cause of the obstruction. You see in this preparation that the ascending aorta and its arch is extremely contracted, that the left and right ventricles and auricles are hypertrophied and dilated,

and that the heart has acquired an enormous volume.

Let us now suppose that the aorta, instead of being of the normal diameter of an inch, acquires that of two inches.

We will assume that the left ventricle, in its healthy state, can readily overcome the inertia of a column of blood contained in an aorta of an inch in diameter, and that then the force of the ventricle and the resistance of the fluid are so balanced, that the circulation proceeds without difficulty. Conceive, however, that the diameter of the aorta becomes increased to two inches, the column of blood within it will be, of course, of the same breadth — its mass is increased — its inertia is greater — it offers more resistance — it is a cause of obstruction — and the left ventricle becomes hypertrophied or dilated, to overcome it. The rest of the cavities of the heart participate in the same state, and for the same reasons as I described in speaking of the obstruction from diseased valves.

I believe, also, that obstructions to the circulation often exist at remote distances from the heart, causing that organ to be diseased. Thus it is by no means uncommon to meet with individuals labouring under hypertrophy or dilatation of the heart, who, from their occupations, are almost perpetually placed in bent positions, — as tailors, shoemakers, weavers, &c. The tortuous course of the arteries must be as much a cause of obstruction to the current of blood circulating within them, as the sinuosities of the bed of a river retard the flow of the waters within it. Cer-

tainly these persons are frequently subject to diseased hearts, and they are often permanently relieved by ceasing their occupation, or by making them work in an upright position.

The parietes of the left ventricle are thicker than those of the right, because the blood has to be propelled to a greater distance from the former than from the latter. I present you with a curious specimen of malformation of the heart, in which the emerging vessels are reversed in their position. The aorta arises from the right ventricle, the parietes of which are hypertrophied; the pulmonary artery from the left ventricle and its walls are very considerably diminished in thickness. Do moral causes affect the dimensions of the cavities, or the thickness of the parietes of the heart? I believe so. But the organ probably then becomes diseased in consequence of the rapid movements it is often thrown into from the frequent anormal influx of blood into its cavities; the heart then has to propel forwards a fluid, and consequently to overcome a resistance too frequently, and finally becomes hypertrophied or dilated.

To sum up our views of the causes of increased thickness of the parietes of the heart, or of dilatation of its cavities, or of a combination of both states, we may observe that these conditions arise from obstructions at some point of the circulation within the organ, or anterior to it in relation to the course of the current of the blood. We may enumerate these causes as follows:—Obstruction from chronic diseases of the lungs; from diseased aortic or mitral valves; from a contracted or dilated aorta;

from an habitually bent or curved state of the arterial system; from moral causes, inducing too frequently an excessive influx of blood into the heart; finally, too plethoric or too weak a state of the system may ultimately produce an anormal condition of the heart, as they do of the rest of the muscles of the body, so that it may become in a state of hypertrophy or atrophy.

LECTURE XXIII.

DISEASES OF THE HEART — *continued.*

SIGNS OF DISEASES OF THE HEART.

Signs of Diseases of the Heart. — Substance of the Organ. — Simple Hypertrophy of the left Ventricle — of the right — of both Ventricles. — Simple Dilatation of the left Ventricle — of the right. — Hypertrophy, with Dilatation of the Ventricles — of the Auricles. — Cautions. — Circumstances under which the Signs cease

Signs of diseased Valves. — “Bruit de Soufflet.” — “Frémissement Cataire.”

General Signs of Diseases of the Heart.

Treatment of Diseases of the Heart.

Carditis. — Morbid Anatomy. — Signs.

Ulcers of the Heart. — *Rupture of the Heart.* — *Hardness* of its Substance. — *Softness* of its Substance. — Signs. — *Surcharge of Fat.* — *Fatty Degeneration.* — *Cartilaginous* and *Ossific* Deposits. — Other accidental Deposits.

I now proceed, gentlemen, to the description of the signs of the diseases of the heart and its valves, after which I shall detail the treatment of these affections.

Simple Hypertrophy of the left Ventricle. — If the stethoscope be applied between the cartilages of the fifth and seventh ribs, a strong impulsion is communicated to the head of the observer. The force and duration of this shock is proportionate to the degree of hypertrophy of the ventricle. This impulsion is immediately followed by an apparent sudden recession of the heart from the parietes of the chest, which Dr. Hope denominates the “back stroke.” The sounds of the

heart now become very indistinct; that produced by the contraction of the ventricle, which is naturally slight, is almost entirely lost; and that caused by its dilatation is of much shorter duration, and less noisy, than in the healthy state.

The extent of surface on which the pulsations of the heart are heard becomes diminished; it is often confined to the space comprised between the cartilages of the fifth and seventh ribs, or to a surface over the ventricle equal only to a square inch.

The patient, although not subject to strong palpitations of the heart, except when under excitation, feels almost constantly, the sensation of the beatings of the organ. Irregularities or intermit- tences of the pulse are also rare in this case: Laënnec thought they sometimes occurred from partial hypertrophy of the walls of the cavity.

The pulse is often strong and developed, but occasionally it is reduced, so as to give the sensation of a mere thread feebly vibrating under the finger. Indeed, you should never rely upon the force of the pulse at the radial artery as indicating the force of the action of the heart; for often the pulse will be feeble, and the movements of the heart strong; and sometimes the reverse will take place; that is, the pulse will be strong and the heart feeble. The face is frequently of a bright red colour, and it is said that apoplexy supervenes upon this form of cardiac disease more frequently than upon any other.

Simple Hypertrophy of the right Ventricle.— When the stethoscope is applied to the inferior

half of the sternum, the impulsion of the right ventricle is very distinguishable: it is precisely similar in character to that given by hypertrophy of the left. This sign, when marked, may be considered as certainly indicating this disease.

According to Corvisart, the dyspnœa is greater in this affection than the preceding; this probably arises from disease of the lungs being usually its primitive cause. The face also assumes a more livid tint.

Lancisi was the first to announce that a swelling of the external jugular veins, accompanied by pulsations analogous and synchronous to those of the arteries, was a sign of hypertrophy of the right ventricle. Corvisart denies the sign to exist. The observations of others have, however, confirmed that of Lancisi, although the jugular pulsations are most common when the hypertrophy of the ventricle is combined with its dilatation. Bertin explains this, by saying, that where there is hypertrophy and dilatation of the right ventricle, the tricuspid orifice becomes larger, and consequently the valves do not meet at their edges so as to close it, and that, therefore, at each contraction of the ventricle, the blood regurgitates into the right auricle, and communicates its impulsion into that contained in the jugular veins. Dr. Hope asserts, that the valves always enlarge with the dilatation of the orifice sufficiently to close it, so that no regurgitation can take place, and that the ventricular contraction impels the blood against the closed valves, which communicate the shock of the blood contained in the right auricle and external jugular veins. I have twice seen the right

jugular vein pulsating, and not the left : this is an anomaly difficult of explanation.

Hypertrophy of both Ventricles.—When the two ventricles are affected, the signs consist of a reunion of those proper to each ventricle, but, according to Laënnec, with a constant predominance of those indicating hypertrophy of the right ventricle.

Simple Dilatation of the left Ventricle.—The impulsion given by the contraction of the ventricle is now entirely lost ; its dull sound is converted into a clear loud noise, similar to that of the ventricular dilatation. The heart's beatings are so feeble, that they are often not to be felt by the hand ; and the sound on percussion is dull over an extent of the chest proportionate to the degree of dilatation of the ventricle. The pulse is generally soft and weak, and there are feeble palpitations.

Simple Dilatation of the right Ventricle.—The pathognomonic signs of this disease are not only derivable from the absence of all impulsion at the inferior portion of the sternum, but from the presence of a clear noisy sound heard in that situation, and to a distance beyond, according to the extent of the dilatation. I refer you to the scale of progression of sound, which I gave in the first lecture upon diseases of the heart, and to the exceptions to the general rule I there announced.

It has been observed, also, in this disease, that the pulse offers nearly the same characters as when the left ventricle is similarly affected. Palpitations are felt, generally consisting of an increase of the frequency and noise of the beatings of the organ ; the impulsion, instead of being augmented,

is diminished. Irregularities of force and frequency of pulsations accompanying the palpitations rarely occur; still, however, they are more frequent than when the heart is in a state of hypertrophy. Compared with dilatation of the left ventricle, it is seen that, in that of the right, the dyspnœa is more intense, that there is a greater tendency to dropsy, that hæmoptysis is more frequent, and that the face is tinged with a deeper violet or lead colour, although sometimes it is remarkably pale.

Extended absence of sound on percussion is not always constant in simple dilatation, as Corvisart supposed, but it is rather found in cases of hypertrophy with dilatation.

These general signs may occur in other affections of the heart when arrived at a certain degree of intensity, and are therefore not to be depended upon, except when confirmed by the local signs.

Hypertrophy, with Dilatation of the Ventricles.—This, the active aneurism of Corvisart, is by far the most common affection of the heart; its local signs are composed of those proper to hypertrophy and dilatation. Thus the contractions of the ventricles give an impulse to the head of the observer by means of mediate or immediate auscultation, in proportion to the thickness of the parietes of the heart. This impulse seems to be formed, at first, by the application of only a small part of the heart to the corresponding internal parietes of the chest; immediately afterwards, it is felt as if produced by the whole mass of the heart, which then instantly recedes, giving the “back stroke.” In extreme cases, it produces the sensation of a large

mass of flesh rolling, or irregularly revolving, in the chest. I have seen the force of the ventricular contractions communicate their shocks to the whole person of the patient — nay, even to the bed on which he was placed, so that the pulse could easily be counted by the movements of the bed-furniture; this was the case in the individual from whom I obtained this enormous heart [presenting it.] The impulse of the ventricles can easily be felt also by the hand, which is, as it were, repelled by short and sudden blows, especially at the moment of palpitation of the viscus: to use the words of Corvisart, if the cardiac region be compressed, the heart “seems to be irritated by the pressure, and reacts stronger still.” The beatings of the temporal, radial, and other superficial arteries, are perfectly distinct. The pulse is often strong, hard, and vibrating, particularly when the disease attacks the left ventricle: this is, however, not constant, for I have met with cases in which, although the heart beat thus violently, yet the pulse could not be felt until after a slight bleeding and the administration of digitalis. Palpitations of the organ are frequent, although irregularities of its movements are rare, except towards the approach of death, or when there is great weakness. The dull sound of the ventricular systole now becomes much louder, and the clear and quick sound of the diastole is lost.

The distance to which the pulsations of the heart may be heard is now great; thus it may not only be distinguished in the precordial region, but, as the organ increases in size, all over the surface of the chest.

If the left ventricle be alone affected, the impulsion and noise will be great between the cartilages of the fifth and seventh ribs, and to the left side generally. If the right ventricle be hypertrophied and dilated, the same signs will be evident beneath the sternum, and towards the right side. If both ventricles be diseased, a combination of the signs proper to both affections will exist.

Signs of Hypertrophy and Dilatation of Auricles.

— The signs of these affections are yet extremely uncertain. The left auricle is usually thus diseased in consequence of a contracted mitral orifice ; and it may be inferred, when a “bruit de soufflet” exists produced by that contraction, that in all probability the auricle is diseased. The signs of hypertrophy and dilatation of the right auricle are still more obscure.

Laënnec makes the following important observations in relation to the local signs of diseases of the heart : —

“Great errors of diagnosis may be committed if you make but a single exploration of the condition of the organ ; it is necessary that you should repeat your examinations, taking also into consideration the general and functional signs which may coexist ; for nervous emotions, so commonly observed upon a first application of the stethoscope to the chest of a patient, may produce palpitations, which, to an inexperienced observer, may give the signs of hypertrophy or dilatation, when really neither of these states are present. Sometimes, too, in consequence of the carelessness or insufficiency of the investigation, these affections are overlooked. Examine, therefore, closely, carefully, and repeat-

edly, before you conclude as to the nature of the disease.

“ There are, perhaps, few individuals whose hearts are in the precise relation of volume or force to the rest of the organs of the body. A large-sized heart in one case produces no distressing effects, if the chest be ample ; while, if the organ be of a similar size in a person whose chest is contracted, death may soon be the consequence. It is therefore of great importance, in establishing our prognosis, to examine the capacity of the chest at the same time with the general volume of the heart.

“ In children the heart is usually larger, in proportion to the rest of the organs, than in the adult, and they often present the signs of hypertrophy with dilatation ; but as they arrive at the age of puberty, an equilibrium is commonly established.

“ Individuals, either at the youthful or the middle period of life, if possessed of good constitutions, may have the heart moderately hypertrophied or dilated, without any very perceptible inconvenience, perhaps being merely subject to occasional palpitations, or slight dyspnœa. In the lower order of people, the organ is doubtless often diseased for a long time, and they pay no attention to their symptoms until they are seriously incommoded by them.

“ But if any disease supervene, or as age advances, so that the body becomes thinned, and the powers of the individual diminished, the disproportion between the heart and the rest of the organs becomes more evident, and symptoms of cardiac disease will manifest themselves. A delicate female, or a man of sedentary occupations, will soon experience serious symptoms from this disproportion.

“The stethoscope may therefore exhibit proofs of hypertrophy and dilatation of the heart, and yet the general and functional signs of such states may not exist. The knowledge, however, that the organ is too thick or large is of great value, as it indicates the necessity of recommending means to diminish its too active nutrition and force; for it is much easier, especially in young persons, to prevent the occurrence of the general and functional signs of these diseases, than even to relieve a single symptom of them.

“All the local signs of hypertrophy and dilatation disappear under two circumstances: — 1. During the agonies of death, or the dyspnœa which usually precedes it for some days, or even weeks, the impulsion of the heart, and the sounds of its movements, cease almost entirely, whatever be the size of the organ; but the frequency of the pulse is often so great, that it cannot be counted. Corvisart observes, that the movements of the heart are then changed into a ‘*bruissement étendu, un tumulte obscur et profond, impossible à décrire.*’ 2. The coincidence of any affection capable of producing considerable dyspnœa, as peripneumonia, œdema pulmonum, hydrothorax, empyema, &c., tends to diminish the force of the impulsion of the organ very considerably.”

Signs of diseased Valves.—The general signs of obstruction caused by diseased valves are associated with those of the affections of the heart itself; I shall therefore describe them at the termination of this subject, and proceed at present with their special symptoms only.

Signs of diseased Mitral and Aortic Valves.—

When these orifices become contracted to a certain degree, the "bruit de soufflet" becomes apparent. This sound, as I have already mentioned, is similar to that produced by the blowing of a bellows whose valve makes no noise; it is sometimes like that heard from the sawing or rasping of wood at a distance. Dr. Hope states that the latter sound is produced by the blood rushing over a rough surface, as of that of an ossified valve, where the phosphatic matter is denuded in consequence of the membrane forming the valve being partly destroyed. Occasionally the "bruit" offers a musical intonation; but I have never heard this except in the arteries.

But as the bruit de soufflet occurs under circumstances of nervous agitation, as well as when the orifices are obstructed, it is of great importance to observe whether it be constant or not. If it be present for weeks or months together, you may be assured it depends upon organic lesion; if, however, it be fugacious, so that it may be heard one day and not another, then you may be equally assured that it is not caused by any permanent obstruction, but in all probability by some peculiar excitement of the nervous system. Sometimes, too, the bruit de soufflet is so slight, that it cannot be distinguished by a common observer. Occasionally, also, the heart, although manifestly diseased, will not produce this sound if the patient be in a perfectly quiescent state. Desire him to walk quickly up a flight of stairs, or as rapidly as he can along a plane surface, the circulation then becomes quicker and more energetic, the blood rushes through the impeded orifice with greater force, and the bruit de soufflet becomes louder where it was but slightly

audible, and often perfectly distinct where before it was not to be distinguished.

When the aortic orifice is obstructed, a distinct bruit de soufflet may be heard superficially under the centre of the sternum, coinciding with the contraction of the ventricle. When the mitral orifice is in a similar state, that sound is then heard towards the left edge of the sternum; it is then more obscure, evidently deeper seated, and coincides with the ventricular dilatation, the noise produced by that dilatation being entirely obscured by it. Another mode of detecting which orifice is affected, is by feeling the pulse at the radial artery whilst you are listening to the bruit de soufflet; for if that sound be synchronous with the elevation of the vessel, it is the aortic orifice which is affected; if with the depression, it is the mitral; for the contraction of the ventricle, the passage of the blood through the opening into the aorta, causing the bruit, and the pulsation of the arteries, occur at the same moment of time, or sufficiently nearly so for all practical purposes, as to be considered at the same instant; while the dilatation of the ventricle, the passage of the blood through the left auriculo-ventricular orifice, causing also the bruit, and the depression of the arteries, occur also at the same moment.

If the valves be so diseased that the orifice they should close remains always open, then a second and a slighter bellows sound will occur immediately after the first, in consequence of the reflux of the blood. The same double sound would also be produced if both the valvular orifices of the left side were affected, and therefore the diagnosis is rendered

difficult. If, however, the aortic orifice admitted the reflux, the second bellows sound would be heard nearer to the centre of the sternum, more superficially and louder than if it was caused by a reflux at the mitral opening: in the latter case, too, the bruit is heard towards the left edge of the sternum.

But, gentlemen, although the bruit de soufflet be always occasioned by obstruction to the circulation, caused by diseased valves, yet that sound may be caused also by other impediments to the current of the blood, especially in the aorta; thus I have met with this sign when that vessel has been contracted or dilated in its ascent, and where it forms its arch. I have before explained that these two opposite states are equally causes of obstruction to the current of the circulation. I think, therefore, we may be justified in saying that all organic causes presenting an obstacle to the passage of the blood from the auricles into the ventricles, and from the ventricles into the aorta or pulmonary artery, may produce the bruit de soufflet.

I have never had an opportunity of examining after death a case in which I had supposed the valves of the right side of the heart to be diseased. If the orifices of that side were rendered narrower, no doubt the bellows sound might be distinguished towards the centre of the sternum.

The *frémissement cataire*, or purring vibration, is felt when there is a considerable obstruction at the left auriculo-ventricular, or at the aortic orifice. I have always found the intensity of this vibration to be in proportion to the loudness of the bruit de soufflet, and, like it, it may be increased by accelerat-

ing the circulation. This is often produced by nervous causes, but then it is never permanent, as when it arises from organic disease.

General Signs of Diseases of the Heart.—When simple hypertrophy exists, the heart is thrown into frequent palpitations upon any mental or physical excitement. The pulse varies exceedingly ; sometimes it is strong and hard, corresponding with the vigorous and impulsive movements of the organ ; at others it is small and thready, and bears no relation to the force of the action of the heart. Active hæmoptysis, or apoplexy, are occasionally the results of this form of disease, from the too forcible impulsion of the blood into the pulmonary and cerebral arteries. The turgid arterial capillaries tinge the face with a florid red colour. Œdema of the subcutaneous cellular tissue sometimes also occurs.

When hypertrophy and dilatation of the left cavities of the heart take place, especially if combined with obstruction at the aortic or mitral orifices, a series of symptoms arise, dependent upon the retardation of the flow of blood through these openings ; by which retardation that fluid tends to accumulate in a direction retrograde to its current, and consequently too great a quantity is thrown into the venous system.

Let us suppose that the obstruction exists at the aortic orifice ; the blood cannot pass it freely, it consequently accumulates in the left cavities of the heart ; the movement of the organ is impeded, and it often struggles and palpitates in throwing forwards the fluid it contains. The pulse, therefore, varies in character ; it may be feeble, if the ventricle be simply dilated, or it may be full, strong,

and hard, as in hypertrophy with dilatations; although, in the latter case, it is sometimes exceedingly small. As the blood cannot readily flow through the left auricle and ventricle, congestions take place in the pulmonary veins and artery, their capillaries become gorged, and dyspnœa is the result. The distension of the pulmonary capillaries is occasionally so great, that apoplexia pulmonum and hæmoptysis frequently follow. This distension of the vessels causes also serous effusions, as I have already explained in speaking of dropsy; œdema pulmonum, or hydro-thorax, or both states occur, aggravating the dyspnœa which previously existed. The dyspnœa is also increased by the enlarged heart pressing upon the lungs and diminishing the space in which these organs have to perform their functions.

The venous blood now preternaturally distends the right side of the heart, in consequence of the obstructed circulation through the pulmonary artery and veins. The superior cava can, therefore, no longer pour its contents freely into the right auricle and ventricle, and the blood accumulates in the whole of its system. The external jugular, the veins of the face and neck become turgid, the countenance is coloured of a bluish or slaty tint; the lips swell and project; the face becomes tumefied from œdema of its subcutaneous cellular tissue. The deep-seated veins of the neck, the sinuses of the brain, and the cerebral capillary vessels, become engorged; the latter occasionally burst from their distension, and instantaneous apoplexy is the consequence; or, what is much more common, the increased quantity of blood accumulated in the

vessels of the brain presses upon the organ, and induces a slowly increasingly sleepiness, or coma, from which the patient can be aroused with difficulty. The pressure is also increased by the increase of serum in the ventricles, and he frequently dies in a state of apoplectic lethargy.

If the system of the superior cava be thus gorged with blood, there is still greater reason that that of the inferior cava should be in a similar state, since that fluid has to mount against its gravity. Thus we find that the *venæ cavæ hepaticæ*, the *vena porta*, and their capillaries, are always highly congested; the consequence of which is, that the biliary secretion is augmented (at least, the urine often assumes a deep yellow colour, as in jaundice), and the *conjunctivæ* and skin are similarly tinged, especially towards the termination of the disease. The mesenteric and splenic veins, to their minutest ramifications, participate in the general fulness; the intestines present a bluish tint; the capillaries of their veins are seen meandering and distended beneath their mucous surfaces; ecchymoses form in their sub-mucous cellular tissue, or blood is even extravasated upon the free surface of the mucous membranes; which fluid, in passing with the *fæcal* matter, gives the latter a black or melanotic appearance: sometimes even pure blood is discharged in coagula. Serum also accumulates in the cavity of the peritoneum, so as to form ascites to a greater or less degree.

If we trace the inferior cava downwards, we shall find that it and the veins supplying it are in a similar state of congestion: the *saphena* and its branches become varicose. I have seen the superficial veins of the belly even largely dilated. Œdema of the

inferior extremities is also a constant result of this state of the veins.

You thus see, gentlemen, that, in consequence of the obstruction to the current of the blood in the left side of the heart, the largest quantity of that fluid is thrown into the venous system. I have chosen for this demonstration an extreme case. You are not to suppose that these symptoms are always thus completely formed, nor that they always appear rapidly: it requires many months, nay, I have seen years elapse, before the above state of things becomes complete.

Treatment of Diseases of the Heart. — Simple hypertrophy, or hypertrophy with dilatation, when arising from diseased valves, or contraction or enlargement of the aorta at its commencement, are states which I believe to be incurable, because the organic derangements which cause them are permanent; but I believe, also, that these conditions of the heart, produced by the long-continued and habitual positions of the body I have described, or by plethora or nervous causes, may be relieved, or even cured, by a judicious and persevering mode of treatment. Those cases, however, which cannot be cured, often admit of great palliation.

Our indications of treatment of simple hypertrophy, or hypertrophy with dilatation, are, in the first case, to diminish the action of the heart by diminishing the thickness of its parietes; and in the second, not only to lessen the thickness of its walls, but to cause a contraction of its enlarged cavity. Our first object should be to remove the cause of the disease. If it arise from the occupation of the patient, let him at once alter the position he is ac-

customed to, or change the nature of his business altogether ; the latter is almost impossible to the lower classes, and the former often difficult. The shoemaker may, however, be made to work before an upright frame ; the clerk can assume a less inclining position at his desk ; but the weaver is unfortunately unable to change his position, as his loom is so constructed that he cannot work otherwise than in a bent posture. Persons of this class very often present themselves at the Lung Infirmary, or at the hospital, and it is surprising the relief they experience from changing the curved position they have been habituated to when at work, to an upright one. If the causes of the affection be diseased valves, or contracted or dilated aorta, we cannot hope to remove them by any means we possess in the present state of medical science.

To answer the indications laid down, the patient should be enjoined to take all the repose that is compatible with the continuation of his general health ; all violent emotions of the mind or body should be sedulously avoided. The treatment proposed by Valsalva and Albertini, for aneurism, should be adopted ; but such is its severity, that you will scarcely find any patient will submit to it for a long period. Their plan, however, although not carried to the extent they proposed, is always of advantage.

This treatment is most efficacious at the beginning of the disease. It consists in bleeding the patient copiously, although not to syncope, every two, four, or eight days, or more, until the palpitations and impulsions have ceased. The food should, at the same time, be diminished until his muscular

powers be greatly weakened, avoiding all stimulants. If, after continuing this plan for two months, he feels no palpitation, and the strong impulse of the heart has ceased, then he may be gradually placed upon a more liberal diet. It will be necessary, of course, to return to the plan of treatment if the symptoms recur.

When the secondary symptoms of hypertrophy, or hypertrophy with dilatation of the heart, present themselves,—as dyspnœa, anasarca, ascites, hydrothorax, or œdema pulmonum, manifesting a general dropsical state,—bleeding, either topical or local, may yet be had recourse to; but more caution should be now used: a smaller quantity should be abstracted each time, recollecting that your object is not to relieve any inflammatory state, but that it is to remove the venous congestions which cause the serous effusions.

When a partial or general serous deposit takes place, diuretics have often been used with much advantage; although it must be admitted they are frequently very uncertain remedies. According to my experience, they are rarely useful except administered in large doses, and for a longer time than they are usually given. The diuretics in use are the nitrate, super-tartrate, and acetate of potash, digitalis, and squills: the latter may be given alone, or combined with mercury. Digitalis appears to me to have an effect upon hypertrophy of the heart perfectly independent of its diuretic qualities. No means, excepting the abstraction of blood, diminishes the impulsion of the organ so completely and so certainly. I have been in the habit of using it for several years for these affections, and have rarely

seen it fail in producing at least temporary relief. I use the digitalis in the form of tincture, giving it in doses of ten drops three times a day ; I continue it for a considerable time, taking care to suspend its administration for a short time if vertigo or nausea be produced by it. The hydrocyanic acid has also been used with a similar intention ; but I have not seen it produce such decided effects.

If diuretics do not diminish the quantity of the serous effusion, some of the drastic purgatives may be combined with them ; of all that class, the elaterium stands pre-eminent. I have frequently seen a distressing dyspnœa, general anasarca, and ascites completely disappear under the influence of the repeated and copious discharges produced by this hydragogue purgative. It should be used with some caution, as no medicine varies so much in its activity ; a quarter of a grain from one specimen may produce the most violent effects, while two, three, or four grains from another will hardly operate. I usually combine the elaterium with calomel, and give it every other day, or twice a week, according to its effects upon the dropsical effusion, watching, however, that it does not distress or debilitate the patient too much. It often produces considerable nausea and vomiting before and during its operation upon the intestines ; but the patient usually submits willingly to these distressing sensations, in consequence of the relief he receives from the diminution of the dyspnœa and general anasarca.

Simple dilatation of the heart is not so easily remedied as simple hypertrophy, or hypertrophy with dilatation. Bleeding should here be avoided,

except when there is great congestion of the venous system, and then but a small quantity of blood should be taken for the purpose of relieving it, otherwise a fatal syncope might be induced. If dropsy supervene, then diuretics and purgatives should be had recourse to, although sparingly and with caution. Tonics should also be given in this disease, — as the bark, bitters, iron, and the carminative plants. When the pulse is very frequent, I often combine the digitalis with these means, and with good effect.

Such is the general method of treating these affections of the heart; but, gentlemen, you must rarely expect to cure them: palliation alone is usually effected; but to relieve is to do much. You can often protract the duration of life for years by perseverance, and sometimes, even when the disease does not depend upon a permanent obstruction to the circulation, a cure may be performed.

It were unnecessary to say a word as to the mode of treating diseased valves; for no means have yet been devised by which any effect can be produced upon them.

Carditis.

By carditis we mean an inflammation of the muscular substance of the heart: this disease is rare, and little is yet known either of its morbid anatomy or of its symptoms.

Paleness, redness, softening, and hardening of the muscular texture of the heart have each been considered proofs of inflammation of the organ:

these, however, are but very equivocal evidences of this disease. The only appearance which may be considered as certainly demonstrative of carditis is the presence of pus infiltrated among the muscular fibres.

I am aware of but two cases recorded of universal carditis; the one is mentioned by Meckel, in which pus was found infiltrated into the muscular substance of the heart: this case was, however, combined with pericarditis; the other occurred to Dr. Latham, who describes the whole heart as being tinged of a deep red colour, that its substance was softened, and that innumerable small points of pus oozed from the section of the muscular fibres.

Partial inflammation, characterised by abscesses or ulcerations, are more frequent. Benevenius was the first to describe an abscess of the heart; Bonetus, in his "*Sepulcretum*," mentions also several instances. Laënnec met with an abscess in the parietes of the left ventricle, near its base, of the size of a nut; pericarditis was also present in this case. The same author met with another instance, in which concrete pus was intermixed with the muscular fibres of the left ventricle: the patient had presented symptoms of acute inflammation of one of the thoracic viscera, although it was not possible to decide precisely its seat: an inexpressible anguish and orthopnea were the principal symptoms.

Ulcers of the Heart.—Ulcers of the heart have been seen more frequently than abscesses. Care must be taken, however, not to confound a rough and unequal false membrane, which may be

the result of pericarditis, with ulcerations of the muscular texture of the heart. Oläus Borrichius has described a case of ulcer of this organ. I here present you a specimen [showing it], in which you perceive an ulcer is situated at the upper part of the left ventricle ; it is about the third of an inch broad, and an inch in length : this heart is also ruptured. Laënnec describes a case almost precisely similar to this.

Rupture of the Heart. — This fatal lesion of the heart rarely occurs without previous ulceration of its muscular tissue. Haller and Morgagni have, however, detailed cases in which this accident has resulted from violent efforts. The heart occasionally becomes so thinned and softened, especially towards its point, that it is surprising it does not rupture in that situation frequently : such an accident is rare. Dr. Hope, however, mentions two instances of rupture from softening of the organ, one of which occurred to himself, the other to Dr. Williams.

These ruptures commonly take place at the upper portion of the posterior surface of the left ventricle, about an inch from its base. My friend, Mr. Langstaff, has two specimens of rupture in that situation ; I possess two also, which are before you, similarly situated. On examining them, you will perceive that there is evidently ulceration of the muscular substance of the heart, proceeding from within to the exterior, and that this lesion was the cause of the rupture.

Rupture of the auricles, with or without ulceration, is still more uncommon than a similar lesion of the ventricles. Bertin describes two instances

of this accident; the one was the consequence of a fall, the other had no appreciable cause. Portal met with an instance of rupture of the superior vena cava at its junction with the auricle, in a young female, who died suddenly in a cold-bath.

Corvisart has described three cases of the rupture of the cordæ tendinæ and carneæ columnæ, occurring from violent efforts. A sudden and intense feeling of suffocation was the effect of these accidents, and finally the general symptoms of diseases of the heart were developed. I present you with a specimen of rupture of one of the carneæ columnæ of the left ventricle; but this is evidently the result of ulceration.

I have met with but two cases of ruptured hearts, and instant death was the consequence in both. This fatal result is not always so immediate, for the blood accumulated in the pericardium occasionally forms a solid coagulum, preventing, for a time, a farther effusion. Cullerier mentions an instance of this kind in the *Journal de Médecine* for 1806.

Hardness of the Substance of the Heart. — The muscular substance of the heart sometimes becomes so hard in cases of hypertrophy, that it resounds, when struck, like horn; the scalpel cuts it with difficulty, and gives a creaking or crepitating sound in passing through it: the heart, nevertheless, retains its proper colour. The creaking or crepitation produced by the section is only hard when the firmness of the tissue is very great.

Softening of the substance of the heart. — The substance of the heart is now flaccid; it appears withered, and may be torn with the greatest faci-

lity; it is sometimes so soft, that the fingers easily penetrate into the ventricles by a slight pressure: the heart is rarely gorged with blood, but appears half filled, and is slightly flattened and collapsed. If an incision be made into the ventricles, they collapse equally, whatever may be their thickness. The colour of the organ is usually changed: sometimes it becomes intensely red, or of a violet aspect, particularly in fevers of a putrid character; more commonly it is of a yellow tint, like that of a pale dead leaf. This yellow-coloured softening may pervade the whole organ, or it may affect only a part; it is generally found where there is dilatation with moderate hypertrophy, or it may occur where there is simple dilatation only, although in the latter case the muscular substance of the heart is generally deeply red, or violet.

Signs of softening of the Substance of the Heart.

— Laënnec, who was the first particularly to describe this affection, gives the following signs by which it may be determined: —

The sounds of the movements of the heart are duller and more obtuse, but never present the characters of bruit de soufflet. A total softening of the heart is, perhaps, always accompanied by a cachectic state. In persons thus affected, though they may possess a certain degree of health, the skin is yellowish, colourless, and withered. The organ may also be inferred to be softened, when an individual, attacked with dilatation, with or without hypertrophy, has had long and frequent fits of suffocation, when the sufferings preceding death have been of long continuance, and when the violet tint of the face, extremities, and other

parts of the surface of the body, has announced that a congestion of the capillary system has been formed for a considerable time.

When the heart gives equally moderate and dull sounds in its movements, without impulsion, it is probably softened, but of good proportions.

When softening exists without dilatation, the sound, although louder, is yet duller than in simple dilatation.

When softening coincides with hypertrophy, the sound of the ventricular contraction is no longer audible, especially in extreme cases; these contractions then become also slower. Sometimes, however, during palpitation, the heart beats with great energy, and its contractions are quick and short, and similar to the blows of a hammer. After this effort, which may last many days, the organ falls into its habitual state of languor.

Softening of the substance of the heart coexists with fevers of the adynamic or typhoid forms. Laënnec does not assert that this state exists in all fevers denominated essential, but believes it to be always most marked where the signs of an alteration of the fluids are most evident.

Bouillaud attributes the softening and the hardening of the substance of the heart, as well as the alterations of its colour, to inflammation. It is replied to this supposition, that, although the organ has been seen so soft that its fibres have broken readily between the fingers, yet pus has never been found among them; nor are there any local pains or general symptoms indicating an inflammatory condition, precedent or present; and finally, that the treatment which is efficacious for soften-

ing of the organ is exactly opposed to that for inflammation.

The treatment for softening of the substance of the heart should be similar to that recommended for dilatation with thinning of its parietes ; I refer you, therefore, to that subject.

Of Surcharge of Fat, and of Fatty Degeneration of the Heart.—The heart is occasionally found covered by a large quantity of fat, which is deposited between its muscular substance and the serous membrane immediately investing it. This fat adheres firmly, and is placed principally at the base, edges, and point of the organ, and along the course of the coronary arteries ; the posterior surface of the right ventricle is also sometimes covered by it, but it is very rare to find any upon the centre of the posterior surface of the left ventricle.

The parietes of the heart are generally diminished in thickness in proportion to the quantity of fat accumulated upon them : sometimes the thickness is reduced to almost nothing, particularly at the point, and at the posterior surface of the right ventricle.

When the heart is in this state, there is usually also a large accumulation of fat in the inferior part of the mediastinum, and particularly before the pericardium, between it and the pluræ : this fat is firm ; a number of fine blood-vessels ramify upon and through it, giving it a reddish tint ; it sometimes pushes the pleura before it, and, enveloped by that membrane, it projects into the thoracic cavity, and forms irregular fringes, something like the comb of a cock. The fat directly applied upon

the muscular substance of the heart is of a pale yellow colour, and of moderate consistence.

No signs have been found by which this state can be discovered.

Fatty Degeneration of the Heart consists in an infiltration of its muscular substance of a matter precisely similar to fat. This change is usually found in a small portion of the heart only, usually its point; but I present you, gentlemen, with a specimen, which I believe to be unique, and in which you perceive the whole heart to be in a state of fatty degeneration. The form of the muscular fibres is still very apparent, but their red colour has entirely disappeared; instead of which, they are of a pale yellow tint. If a scalpel be plunged into the organ, or paper be rubbed upon its surface, they become greased, as if by butter or lard. When, however, the degeneration is partial, it seems to commence from the external surface to proceed inwardly, so that near the cavity of the ventricles the muscular texture is very apparent; more externally it is less evident; and upon the surface it is often lost.

There are no symptoms yet known indicative of this condition of the heart.

Of Cartilaginous or Ossific Deposits in the Muscular Texture of the Heart.—The muscular substance of the heart has rarely been seen ossified. Haller relates the case of a child, in whom the inferior portion of the right ventricle, the most fleshy part of the left ventricle, the semi-lunar valves of the aorta, and pulmonary artery, and the aorta, were in that state. Renauldin published a most extraordinary case, of a student in medicine, of the age

of 33 years, in whom the heart was found extremely hard and heavy; its muscular substance was in a state of petrification, presenting in some parts a sandy appearance, in others that of a saline crystallisation. The *carneæ columnæ* were ossified without their form being changed, although they had increased in volume, some of them being as large as the extremity of the little finger, and appearing like stalactites, placed in different directions. The right ventricle was healthy; the temporal, maxillary, and a part of the radial arteries, were ossified on each side.

The symptoms of this case were, strong and frequent palpitations of the heart; the cardiac region gave a dull sound on percussion, and, when the hand was applied over the organ, a sort of separation of the ribs was felt; upon slight pressure, considerable acute pain was produced, which remained some time after the hand was removed.

Burns also mentions a case of ossification, in which some of the *carneæ columnæ* were transformed into bone. There are a few instances on record of ossification of the auricles.

Cartilaginous deposits are occasionally found in the muscular substance of the heart; but they are generally interposed between the internal membrane of the organ and its muscular fibres, and are rarely of considerable extent or thickness.

Laënnec believed that an extensive ossification or cartilaginous induration of the heart would produce a considerable increase of the sounds of its movements, and that these sounds would probably be heard at some distance from the patient.

Other accidental Deposits in the Substance of the

Heart.—Tubercles have been found in the substance of the heart. Recamier records an instance of a part of the heart being converted into a scirrhus mass. I have seen a deposit of medullary sarcoma between the serous membrane enveloping the organ and its muscular fibres [showing it]. Laënnec, Andral, and Bayle mention similar instances.

Serous cysts have also been observed between the serous membrane and the muscular fibres: Dupuytren met with one in the substance of the organ, projecting into the cavity of the right auricle.

LECTURE XXIV.

DISEASES OF THE PERICARDIUM.

*Classification.**Pericarditis* — acute — chronic.*Acute Pericarditis.* — *Morbid Anatomy.* — First, Redness. — Second, Effusion. — Third, Formation of False Membranes. — Fourth, their Conversion into Cellular Tissue, or Fibro-Cartilage.*Chronic Pericarditis.* — *Morbid Anatomy.* — White Stains on the Surface of the Heart.*Signs of acute Pericarditis* — their Uncertainty. — Pain — Palpitations — Dyspnœa — “Bruit de Soufflet.” — Probable Causes. — Questions proposed. — Signs of Union of False Membranes. — Diagnosis.*Treatment.**Hydro-Pericarditis.* — Characters. — Signs. — Treatment.*Pneumo-Pericarditis.**Accidental Productions* developed in the Pericardium — *Polypi* — formed after and before Death. — Signs.*Diseases of the large Blood-Vessels.* — Inflammation of the inner Membrane of the Heart and Arteries. — Redness of external Membrane. — *Pseudo-Membranous* Exudation upon the internal Surface of the Heart and Arteries. — Ulceration of the inner Membrane. — Osseous, calcareous, and cartilaginous Incrustations of the Aorta. — Signs.

THE pericardium is a fibrous membrane enclosing the heart; it is lined by a fine serous membrane, which is afterwards reflected over the surface of that viscus, and the origin of the great blood-vessels arising from it. This serous membrane secretes a limpid fluid for the purpose of lubricating the inner surface of the pericardium and the external surface of the heart, by which the movements of these parts upon each other are facilitated.

The diseases of the pericardium bear a great analogy to those of the pleura; for, like the latter,

they almost all have for their results an alteration in the nature of the serous fluid secreted. These diseases are as follows : —

Pericarditis { Acute.
 { Chronic.

Hydro-pericarditis.

Pneumo-pericarditis.

Adventitious deposits.

Pericarditis.

By pericarditis is meant an inflammation of the serous membrane lining the pericardium and covering the heart : it is divided into two forms — *acute* and *chronic*.

Acute Pericarditis.

Morbid Anatomy. — This part of our subject may, like pleuritis, be considered in reference to the following stages : — 1st, redness ; 2d, effusion ; 3d, formation of false membrane ; 4th, conversion of false membrane into cellular tissue, or fibro-cartilage.

1st Stage. — Redness. — The redness of the serous membrane is rarely considerable ; it generally appears irregularly disseminated in spots, points, or stains upon its surface ; but, as I have mentioned in speaking of the red stage of pleurisy, these forms are probably mere post-mortem results, depending upon the partial transudation of blood through the inflamed vessels, so that during life the red colour was more universally diffused. The serous membrane itself is not thickened.

2d Stage. — Effusion. — Soon, probably a few hours after the red stage has commenced, effusion

takes place from the inflamed surface. This effusion is limpid, of a slight fawn or straw colour; sometimes it is slightly reddened; occasionally a few fragments of half-concreted albumen float within it, but these are rarely sufficient, in the early period of the disease, to produce a milky or troubled appearance. The quantity of fluid varies; it may be but very small, or there may be a pint or more. Corvisart once met with as much as four pints.

3d, Formation of False Membranes. — A semi-concrete albuminous false membrane is soon deposited after the effusion has commenced. It usually covers the whole surface of the heart, the origin of the great blood-vessels, and the inner surface of the proper pericardium. This pseudo-membrane rarely presents a smooth appearance, although there are two or three preparations on the table which are of that character; but it is usually reticulated, irregular, unequal, or papillated.

The consistence of the albuminous deposit is slight at first—it is scarcely that of half-boiled white of egg; it afterwards becomes more concreted, and finally acquires a greater firmness and thickness than the false membranes which form upon an inflamed pleura; it adheres also with great tenacity to the serous membrane beneath. Its colour varies from a light whity-brown to that of pus.

The comparative quantities of serum and false membranes vary considerably; generally there is infinitely less serum, in proportion to the false membrane, in pericarditis than in pleurisy or peritonitis. Often in intense pericarditis there is scarcely any serous fluid, whilst there is a large quantity of thick and concreted albuminous matter

occupying the space between the heart and pericardium. In this case, it is probable that the thinnest portion of the serum is absorbed, and that the albuminous substance with which it is charged becomes deposited.

When pericarditis occurs, it generally attacks the whole surface of the serous membrane lining the proper pericardium, and covering the heart and its great blood-vessels. The disease is rarely partial; when it is so, the anatomical characters are nearly the same: the serous effusion is then occasionally as considerable as when the inflammation is universal; usually, however, it is less abundant. The false membrane generally covers the inflamed parts only.

4th, Conversion of False Membranes into Cellular Tissue, or Fibro-cartilage.—Nature frequently effects the cure of this disease in precisely the same manner as she does that of pleurisy. If the opposed membranes be thin, they gradually approximate as the serum between them becomes absorbed; films of concrete albuminous matter then pass from one false membrane to the other: these films gradually become vascular and organised, being at first soft and opaque, and finally firm and transparent, assuming altogether the anatomical and physiological characters of cellular tissue. If, on the other hand, the pseudo-membranes be thick and firm, then also, as the interposing secretion becomes slowly absorbed, their surfaces approach, unite, and form, in the same manner as in pleurisy, a single fibro-cartilaginous membrane, of a thickness proportionate to the elementary membranes which by their union have formed it.

Chronic Pericarditis.

Morbid Anatomy.—This form of the disease always affects the whole surface of the serous membrane, which is then highly reddened in the form of small spots, or stains, closely approximating to each other. Laënnec considers that chronic pericarditis is rarely accompanied by pseudo-membranous formations, or that, when they exist, they are softer, and similar to a layer of thick pus. There occurs a lactescent secretion, which ultimately becomes puriform. He believes, also, that the intimate adhesion of the pericardium to the heart is the result of chronic pericarditis, while the cellular union depends upon the acute disease.

But it would be, perhaps, more correct to consider the red stage of pericarditis, when accompanied by acute and sudden symptoms, as the *acute* form alone; and the disease would be more properly called chronic soon after the serous effusion has occurred, or, at least, when the albuminous matter separates to form the false membranes; for after that period, the changes in the colour, consistence, &c. of the serous fluid, and of the false membrane, and the mode of their union, vary exceedingly: sometimes they will present all the appearances described under the head of acute pericarditis, sometimes all those mentioned of the chronic disease. It appears to me that Laënnec has erred in considering the above characters as conclusive of pericarditis originating in the chronic form, when they are, perhaps, almost as frequently the consequences of acute inflammation. I have

already made some observations upon this subject, in describing acute and chronic pleuritis. I have there also detailed some opinions relative to the formation of false membranes upon the serous surfaces: the same observations apply here. I shall not recapitulate them, but refer you to the lecture on Pleurisy.

In many cases of pericarditis, particularly when it has arrived at the chronic form, the substance of the heart itself becomes discoloured and whitened, as if it had been macerated for some time in water. This appearance is often united with a softening of its tissue. The discolouration and softening has been attributed by some authors to inflammation of the muscular fibres; but pus has never been seen among them, and therefore this opinion remains problematical.

White Stains on the Surface of the Heart.—Those who are frequently in the habit of making post-mortem examinations often find upon the surface of the heart, not otherwise affected, substances of a whitish colour, opaque, and varying in size from a shilling to that of the palm of the hand; they are about a line in thickness, and have somewhat the consistence of soft cartilage. It appears they are placed upon the surface of the serous membrane investing the heart, for they may be removed without destroying it. It is a question whether this appearance depends upon a partial pericarditis forming a false membrane, afterwards converted into condensed cellular tissue, or not: Laënnec inclines to the former opinion; but why are not these bodies found also, subsequent to inflammation of the serous membrane, lining the

fibrous pericardium, or on the pleura and peritoneum?

Signs of Pericarditis. — Acute Pericarditis. — This disease is almost always accompanied at its commencement by severe inflammatory fever, but its local and functional signs are certainly more irregular and equivocal than those of any of the diseases of the heart; for sometimes they may present themselves in their exquisite form, and yet the pericardium be intact; in other cases, the disease may be perfectly formed, and yet produce no symptoms.

When the symptoms present themselves, they vary also exceedingly in their duration, intensity, and succession. Sometimes the disease runs a rapid course, and death is the consequence after the most agonising distress; at other times the symptoms may be slight and chronic. The symptoms also do not, as in pleuritis, indicate the particular anatomical stages of the affection with any certainty, nor are they the measure of the degree of lesion to which the pericardium is subjected. Let us, however, examine these signs.

Pain often takes place in the region of the heart, and it is increased by inspiration and coughing. Dr. Hope observes, that it is aggravated by pressure on the precordial ribs, and by forcing the epigastrium upwards on the left side: these latter circumstances occasionally may be produced, but certainly not invariably.

Palpitations most frequently occur, consisting of impulsive ventricular contractions, succeeded often at intervals by shorter and feebler pulsations: this

sign is often accompanied by syncope or lypothymia. The *pulse* usually bears a relation to the regularity or irregularity of the beatings of the heart, but frequently none to the force of its movements: thus it may be full, hard, and jerking, conveying a thrilling vibration to the finger, or it may be very feeble. Corvisart believed that the pulse gradually increased in irregularity from the commencement of the disease; but there is no doubt that, at the earliest period of the affection, it is often as intermittent, irregular, and thready, as towards the termination.

Dyspnœa is rarely absent; it is often extremely great, so that patients can scarcely breathe in a horizontal position, but you find them generally in the upright posture. I have seen them in this state of orthopnœa for many days, and even weeks. If this sign be severe, the uneasiness, distress, and jactitation become incessant; the face assumes a livid hue, and swells; the extremities and trunk even become œdematous; the cerebral functions are also disturbed; slight delirium or constant sleepiness takes place, although positive repose is rarely obtained. A slight dry *cough* occasionally coexists with the dyspnœa.

The patient sometimes complains of heat and weight about the heart, but these signs are frequently absent.

Upon the application of the stethoscope to the region of the heart, the impulse of the movements of the organ are almost always felt to be increased: that impulse is not regular, for after them feebler movements frequently follow, to be succeeded by stronger. The *bruit de soufflet* is also frequently

heard during the contractions or dilatations of the ventricles.

When I first met with the bruit de soufflet in a case of pericarditis, I confess I was much surprised; for I saw no relation between the causes which usually produce that sound and inflammation of the pericardium. The known causes of the *bruit* are obstructions at the orifices of the heart to the current of the blood, and what are called nervous causes: the first being distinguishable from the second by organic lesions rendering the bellows sound permanent, and the nervous of temporary duration.

Finding, however, that in many cases of pericarditis there was no bruit de soufflet, I was led to suppose that in those cases of the disease in which it was permanently formed, it was probable that organic lesions of the valves, or dilatations and contractions of the aorta, with their consequences upon the structure of the heart, might have existed before the pericarditis; and, upon a most minute enquiry into the history of the patients thus affected, I found that the greater number of them, particularly children, were never considered by those around as perfectly healthy individuals, but that they had always been subject to dyspnœa or palpitations upon that degree of active exertion or movement which upon healthy persons of the same age would produce no such effects. It is probable, therefore, that, if these individuals had been examined before the pericarditis had occurred, the bruit de soufflet would have been found.

I examined not only all my own preparations of inflammation of the pericardium, but also, by the

permission of my excellent friend Mr. Langstaff, all those, and they are numerous and complete, contained in his museum. I divided them into two series; in the first I placed all the cases of pericarditis in which the heart was of its normal proportions: in these the valves were not diseased, and I presume there had been no bellows sound; in the second series I arranged all the cases of inflammation of the pericardium in which the hearts were of large proportions; and in all these there were obstructions at the mitral orifice, or diseases of the aortic valves, or enlargement or diminution of the ascending aorta. In these cases a permanent bruit de soufflet must incontestably have been present.

I would consequently infer, that, when a bellows sound exists throughout the progress of pericarditis, it arises from the organic causes which usually produce that sound, and not from inflammation of the pericardium. I may add, also, that it is extremely probable that enlargements of the heart, by compressing the pericardium, must strongly predispose that membrane to inflammation.

To elucidate the cause or causes of the bruit de soufflet, when it coexists with pericarditis, I propose to you the investigation of the following points:—

1. To ascertain, *post mortem*, when the bruit has been permanent through the course of the pericarditis, whether the heart or its orifices are of the normal proportions: because, if they are, then the sound must have depended upon the inflammation of the pericardium; if they are not, it was the consequence of disproportion.

2. To determine, in those cases of pericarditis in which the bellows sound is not heard, whether the heart and its orifices are of their natural size; because, if they are, the absence of the bruit is explained by the absence of its usual organic causes.

3. To examine whether, after all the signs of pericarditis have disappeared, the bruit de soufflet continues: if it does not, we must infer that it was caused by that disease only; if it does, that it depends upon obstructed cardiac orifices.

4. The bruit de soufflet sometimes coincides with the diastole of the ventricle, and sometimes with the systole. There is no reason at present known why this irregularity should occur, if it depended merely upon pericarditis; examine, therefore, post mortem, whether these variations are not found to correspond with the lesion of a particular set of valves only.

When the union of the false membranes has been so effected as to cause, by their intermedium, adhesions between the pericardium and the heart, it has been supposed by Meckel that the pulse is then habitually small, and by Laënnec and Viuessens that palpitations are the constant results. Dr. Sanders supposed, also, that a depression or hollow was formed at the epigastrium under these circumstances. None of these signs are to be depended upon as indicative of adhesions. I have seen the pericardium united with the heart by a thick and cartilaginous false membrane, without any lesion of the functions of the organ.

There is no single sign which can be considered as pathognomonic of any stage of pericarditis; nay, the union of them all is insufficient to express

the certain presence of the disease ; yet, by the examination of the abdominal organs and the lungs, certain negative circumstances tend to render the diagnosis more certain.

Thus the most certain symptom of pericarditis is dyspnœa. Now, dyspnœa can only arise from diseases of the lungs and pleura, from affections of the heart and pericardium, or from lesions of the organs contained in the abdominal cavity preventing the free play of the diaphragm. Examine, then, the abdomen, and you will readily detect any intumescence sufficiently great to prevent the descent of that muscle ; next, examine the lungs most carefully, and note whether there is sufficient disease there to account for the dyspnœa. If there be no abdominal or pulmonary disease, or the latter be so slight as to bear no proportion to the dyspnœa, then it must be inferred that the cause of the difficulty of breathing arises from some disease either of the heart or pericardium. The question will now be to determine whether the heart be diseased, or the pericardium inflamed : the latter affection may be affirmed to be present when the attack is sudden, when there is inflammatory fever, when the pain and burning sensations occur in the cardiac region, when there are sudden palpitations of the heart, and especially when these signs have supervened upon a general attack of acute rheumatism. After all, gentlemen, you must not consider this accumulation of symptoms as *perfectly* diagnostic, as affections even of the head have been seen to stimulate this disease ; yet I have most commonly found that they have been sufficient to establish a correct diagnosis.

Treatment of Pericarditis.—During the first few days, when inflammatory fever and pain exist, the disease should be treated precisely after the plan proposed in the first stage of pleurisy; bleeding, leeches, and cupping should be employed proportionate to the intensity of the disease and the power of the patient. Mercury is also a most important remedy, and should be freely given from the commencement; and the antiphlogistic plan is to be strictly enforced. I refer you to the treatment of acute pleurisy, as it is unnecessary to recapitulate it here.

When, however, some days or weeks have passed, so that the disease assumes the chronic form, your indication is to cause absorption of the effused fluid; but that is rendered difficult, from the inorganic nature of the false membrane by which that function has to be performed. Mercury, carefully administered in this stage, so as not to excite salivation, but rather to keep the mouth tender for two or three weeks, is the most efficacious remedy. I would recommend you to use the same method, with the precautions I have given in using mercury, for the treatment of chronic hepatisation of the lungs. You will find, also, that counter-stimulants and exutories are often of great service; as blisters, setons, &c.

Hydro-Pericarditis.

By hydro-pericarditis we mean an accumulation of serous fluid, in greater or less quantity, in the bag of the pericardium. This disease, like hydrothorax, may be symptomatic or idiopathic: the latter form is, however, rare; the preceding very

common. Slight serous effusion is often a mere cadaveric result, or it is a consequence of the agonies of death. Symptomatic hydro-pericarditis is usually combined with general infiltration in the serous cavities and cellular tissue of the rest of the body. In the idiopathic form, the effusion is confined to the pericardium.

The effused fluid is sometimes colourless, but most frequently it is of a slight fawn, straw, or ruddy tint; it rarely presents a reddish aspect. Its quantity varies from an ounce or two to two or three pints: Corvisart had seen as many as eight pints. The heart and pericardium are usually free from any alteration in this disease.

Signs of Hydro-Pericarditis.—Various signs have been given of hydro-pericarditis, although none of them are pathognomonic: thus, according to Lancisi, there is an enormous weight at the precordial region; others have stated that the patient has felt his heart as if swimming in a fluid; Senac mentioned that he had seen the movements of the fluid in the intercostal spaces between the third, fourth, and fifth ribs; and Corvisart asserted that he had felt its fluctuation. The last author gives the following signs of this disease:—"Sensation of weight at the precordial region, which region gives a dull sound on percussion. The beatings of the heart are felt to a considerable extent; they are to be distinguished, at certain moments, better at one point than at another, and this point varies every instant, being sometimes at the right side, sometimes at the left. These movements are also tumultuous and obscure, and seem to arrive at the hand through an interposed soft body; the pulse

is frequent, small, and irregular; the extremities, and the integuments over the region of the heart and trunk, are infiltrated with serum; the patient cannot bear the upright posture for an instant; he experiences frequent syncope, but rarely palpitations."

These signs are as uncertain as those of pericarditis. Much assistance may be given in determining this disease by observing the state of the lungs, to ascertain whether they present sufficient disease to account for the great dyspnoea.

Treatment of Hydro-Pericarditis. — The treatment of this disease should be precisely the same as that for hydro-thorax and œdema pulmonum; I refer you, therefore, to those subjects. It has also been proposed to puncture the pericardium, for the purpose of evacuating the fluid; but I have so little confidence in the signs of this affection, that I have never ventured to recommend that operation.

Pneumo-Pericarditis.

By this expression is meant an aëriform effusion developed in the pericardium. This effusion is often found at the examination of bodies in whom the process of putrefaction has commenced; but it is occasionally also found when it must have existed during life. In the latter case, it is accompanied by liquid secretions, although sometimes the pericardium is found distended by air alone. Laënnec asserts, that air and liquids may be developed in the pericardium a short time before death, and that he has been enabled to

announce that event by the sound on percussion being clearer and louder over the cardiac region, or by a fluctuation corresponding to the beatings of the heart, or to a strong inspiration. He believes, also, that, when the pulsations of the heart are heard at a distance from the patient, that it depends upon a sudden deposition of air in the pericardium, which is as suddenly absorbed, producing no distressing symptoms. I have met with several instances in which these pulsations were heard at a distance, but the patients presented no signs of disease either of the heart or pericardium.

Accidental Productions developed in the Thickness of the Parietes of the Pericardium. — Bonetus, in his *Sepulchretum*, mentions instances of the development of tubercles, cysts, and cancerous tumours, in the pericardium. Mr. Langstaff has a preparation showing a number of tumours presenting the characters of medullary sarcoma, placed upon the serous membrane covering the heart. Ossific deposits have also been seen formed between the membranes of the pericardium: these ossifications vary considerably in thickness and extent.

Polypi.

Before we proceed to the consideration of the diseases of the large blood-vessels, let us first study the characters of the sanguineous concretions that are frequently found in the cavities of the heart, and which have been called polypi.

Upon making an anatomical examination of the heart, you will often find that its cavities contain

coagula or concretions, varying in size, colour, and density. These coagula were supposed to give rise to symptoms which we now know to depend upon hypertrophy or dilatation of the organ. Similar concretions are occasionally found also in the large blood-vessels. These polypiform substances form either during life, or during the last moments, or immediately after death.

The left carotid artery, and the left internal jugular vein, have been seen obstructed by coagula by Haller. I have seen coagula extending considerably up both carotids. Haller had seen the inferior cava similarly obstructed, between the giving off of the renal and iliac veins. Laënnec saw the inferior cava contracted and obliterated to the breadth of four fingers, by a whitish, fibrinous concretion, completely filling the vein; its external layer, strongly adherent to the internal parietes of the vessel, was exactly similar to the inflammatory crust of the blood, but of a firmer consistence. The internal layers were of a yellowish colour, perfectly opaque, and like the decomposed fibrine sometimes found in large aneurismal sacs. Other concretions have been observed more or less red coloured, especially in their interior; and these have not so completely filled the vessel but that the blood has been enabled to flow, although with difficulty.

These coagula must have formed during life; and it is extremely probable that the same thing takes place in the heart, especially in individuals who are struggling with death for a considerable period, and in whom the circulation is carried on with irregularity. Many authors have noticed

these facts, and have recorded them"; as Bertin, Bouillaud, Laënnec, Burns, Velpeau, &c. It is now generally admitted that some of these concretions are formed before, and some after, death.

The polypus which forms in the heart after death, and is consequently the most recent, consists of a slightly whitish, or yellowish, opaque and semi-transparent layer, like the inflammatory crust of the blood, enveloping a coagulum of blood; this layer is never complete, but only partially surrounds the clot. It does not adhere to the parietes of the heart, or to the vessel in which it may be enclosed. This concretion is sometimes firmer and thicker, and forms an isolated mass. When the blood is very thin, it is semi-transparent, less firm, and trembles like jelly; it has no fibrous texture, and appears infiltrated with serosity.

The polypi which form during life are much firmer than the preceding, having nearly the consistence of muscular fibre. They adhere more or less to the parietes of the heart; and that adhesion is often considerable when they form in the sinuses of the auricles, or in the ventricles, in consequence of their interlacement with the *carneæ columnæ*; which latter are often even flattened by their pressure. These concretions are opaque; their texture is fibrinous; they are of a pale, fleshy, or violet colour, different shades of which often exist in different parts of the same concretion. Sometimes an isolated clot of blood is found in their centre, and stains of that fluid upon its surface are seen plunging more or less deeply within. Laënnec believes that these polypi may become organised, and that the vegetations I have

already described are the forms which they then assume. These polypi are most commonly found in the right auricle and ventricle.

There are other concretions occasionally found, which must have formed some time before death. They are adherent to the parietes of the heart, and can be generally detached only by scraping them with a scalpel. Their consistence is less than that of the preceding species; they are not fibrinous, but are somewhat similar to a dry paste, or to the decomposed layers found in aneurismal sacs. These have been discovered only in the auricles and their sinuses.

Signs of Polypi. — The signs of polypi of the heart are very obscure. Laënnec believed that, when they had arrived at a certain volume, they would be indicated by the following symptoms:—

When, in an individual whose heart has hitherto pulsated regularly, its beatings become suddenly anomalous, confused, and obscure, so that they no longer admitted of analysis, a polypus may be suspected. If this derangement occurs only on one side of the heart, its presence may be considered as almost certain. Thus, when in exploring the heart at the inferior part of the sternum, its beatings are found to be confused and tumultuous, whilst they were regular a few hours before, a concretion may be suspected to have formed in the right cavities, especially if the movements of the left side of the heart are still distinctly heard.

DISEASES OF THE LARGE BLOOD-VESSELS.

1. *Inflammation of the inner Membrane of the Heart and Arteries.*

I shall, in the first instance, describe the various appearances which occur on the inner surfaces of the heart and arteries, and which have been supposed by some authors to arise from inflammation of their lining membrane, although denied by others.

1. *Redness of the internal Membrane of the Heart and Arteries.*—The interior of the aorta and pulmonary artery is often found uniformly reddened, as if tinted by the blood they contained: this redness may be of a scarlet, or brown, or violet shade.

The scarlet colour is usually confined to the internal membrane of the heart, aorta, and pulmonary artery, but sometimes it penetrates through the fibrous even to the cellular coat of the arteries. This tint is uniform, some points being only deeper coloured than others; there is no arborescent appearance of injected capillaries. Sometimes the colour gradually diminishes in its intensity, from the origin of the vessel to where the tint is lost; at others it ceases suddenly, forming irregular, although strictly defined, edges. Occasionally, in the midst of a red surface, a circumscribed white spot is seen, as if the blood had been pressed out by the point of the finger. When there is but little blood in the aorta, the red colour exists only where that fluid is in contact with the vessel. The origin and arch of the aorta are most frequently reddened; the whole of the arteries are rarely so.

The mitral and aortic valves appear as if they had been plunged into a red tincture, and their colour is generally deeper than that of the arteries.

When the pulmonary artery is thus affected, its valves and the tricuspid are generally in the same state.

The valves are occasionally coloured when the internal surfaces of the heart are not; but when the latter are also affected, the aspect of the valves presents a still deeper hue. Sometimes these surfaces are tinted of a deep red, whilst the valves remain of their natural appearance: in that case the heart has always been filled with blood, and the arteries emptied.

Does this redness depend upon inflammatory action? Frank, Bertin and Kreysig, and Bouillaud, have adopted this notion; but the colour is never attended by thickening of the membrane lining the heart and arteries, and its sudden, abrupt, and irregular termination, would seem to indicate that it was caused by the irregular flowing of a coloured liquid upon the surface, and which, by the smallness of its quantity, could not touch all its points. Finally, this state of the heart and vessels has not only not been found to correspond with any specific symptoms, but it occurs in diseases of a very opposite character.

The second species of redness, or that which has a brown or violet tint, is also found in the aorta, pulmonary artery, valves, auricles, and ventricles; and sometimes it appears in them all at the same time.

This colour is often of unequal intensity, but it is always particularly marked where the blood has

been most in contact with the parts : it generally penetrates through the internal membranous lining of the heart and arteries, and more or less into the textures beneath it. This redness is commonly found in those who have died of typhoid fevers, of emphysema of the lungs, of diseases of the heart, or whenever the agonies of death have been of long continuance, and accompanied by sensations of suffocation ; it has been found, especially when the blood is very liquid, and when the putrefactive process has commenced : thus it is seen most in the summer time, and when the body has not been opened before twenty-four hours after death. The redness is frequently attended by a certain degree of softening of the tissue of the heart, and humidity of the parietes of the arteries. Does this colour depend upon inflammation ? It appears not, but that it is caused by the blood tinting the parts with which it comes in contact. The redness may also be produced artificially in a day or two, by introducing thin blood into a portion of an artery separated from the body.

Inflammation of the internal coats of an artery may be suspected, if, in addition to the red colouration, they are swelled, and their capillary vessels injected.

Pseudo-membranous Exudation from the internal Surface of the Heart and Arteries. — The formation of a false membrane may be considered as an incontestable proof of the pre-existence of inflammation of the lining membrane of the heart and arteries. Several instances of this plastic formation have been observed : Bayle had seen the tricuspid valves inflamed and covered by

lymph; Dr. Farre saw the aorta lined by it; Burns, Bouillaud, Bertin, and Laënnec have described similar instances of this deposit adhering to the internal surfaces of the heart and arteries.

Ulceration of the Membrane lining the Heart and Arteries. — The internal membrane of the heart and arteries is so thin, that it is difficult to conceive its ulceration without that also of the tissues beneath it; yet there are many evident proofs of it, as may be seen in the works of Hodgson and Kreysig. Small pustules are also occasionally found projecting into the cavity of the aorta. These are probably formed upon the surface of the fibrous coat of the artery, or in the fine cellular tissue uniting that coat with the internal membrane.

Osseous, calcareous, and cartilaginous Incrustations of the Aorta. — Osseous and cretaceous deposits are always placed, when present, between the internal and middle coats of an artery. These substances are usually irregularly flattened, and of unequal thickness. Their internal surface is generally rough, and imbedded in the fibrous coat of the artery, upon which they not only leave their impression, but gradually destroy its texture, so that when these concretions are removed, the vessel is semi-transparent where they had been deposited. Their internal surface is usually smooth, and covered by the internal membrane of the artery; sometimes, however, they are rough, and destroy that membrane, so that the phosphatic matter is exposed to, and bathed by, the blood. These incrustations form at first in small points, which gradually increase and unite, until they have

been seen so completely to surround the artery, as to form an osseous coat around it.

Cartilaginous deposits often precede, and may be considered as the rudiments of, the ossific formations. These cartilages are softer than their analogous tissues. The phosphatic matter is at first deposited in them in small points, which increase in number, unite, until the whole of the cartilage is converted into bony matter.

The phosphate of lime is sometimes secreted in a pulverulent form; it is then found between the internal and middle tunics of the artery, mixed with a certain quantity of fluid, so that it presents the appearance of mortar, or putty, of greater or less fineness. The phosphate is occasionally found beneath the cartilaginous deposit, of the same putty-like appearance.

These osseous incrustations often break through the internal membrane of the artery, and separate it to a certain extent at their edges, forming projections into the cavity of the vessel. The blood then insinuates itself behind these projections, which fluid being placed out of the direct current of the circulation, coagulates, and still further destroys the fibrous coat, so as to become the most common cause of false consecutive aneurism. The fibrine thus formed has been called atheromatous matter, and the parts from whence the osseous deposits have been separated have been considered as ulcers, although they are really not so; it is true that the edges of the internal membrane which has been ruptured are seen red and swollen; but these are the effects of the separation, and not the causes.

It is still a contested point whether the deposits I have just described are the consequences of inflammation or not. My time does not permit me to enter into the various hypotheses of different authors upon this subject; but I believe they are no more the results of inflammation than the calcareous deposits in the lungs.

You thus, gentlemen, perceive that the redness of the internal coats of the arteries depends most commonly upon mechanical causes, and not upon inflammation; and that the latter cause can only be admitted when the colour assumes an arborescent form, combined with a certain degree of swelling of the membrane, or is followed by an exudation of plastic lymph, or by ulceration.

Have we any signs of this inflammation when it really exists? None that are certain; but the following have been mentioned:—Pulse hard, quick, and full; pain and heat in the course of the aorta. Frank and Pinel have supposed arteritis to be the organic cause of synocha, from whence the latter author has denominated that affection “fièvre angioténique.”

LECTURE XXV.

DISEASES OF THE LARGE BLOOD VESSELS—*continued.*

ANEURISM OF THE AORTA.

- Definition.*—*True Aneurism.*—*Morbid Anatomy.*—Ascending Aorta.
—Descending Aorta.—Other Arteries.—Dilatation of the Capillaries.—State of internal Membrane of the Aorta.—Layers of Fibrine.
—*False consecutive Aneurism.*—Sac—its Structure—Situation.
- Causes of Aneurisms.*—*True.*—Impulse of Blood against the Parietes of a diseased Artery.—*False.*—Various Opinions—Dubois, Dupuytren, Scarpa.—Sudden Rupture of internal and fibrous Coats.—Cases.
- Concretions of Blood in Aneurisms*—rare in True, common in False Aneurisms—why.—Character of Concretions.—Rupture of Sac.
- Effects of Aneurism upon surrounding Organs.*—Compression upon Pillars of Diaphragm.—Lung.—Trachea.—Œsophagus.—Heart.—Pulmonary Artery.—Thoracic Duct.—Superior Cava.—Bones.—Vertebrae.—Sternum.
- General Signs*—equivocal.
- Local Signs.*—Impulsion.—Bruit de Soufflet.—Frémissement Cataire.—Signs according to Situation of Aneurism.
- Treatment.*—Tumour on an Artery—its Description.—Corvisart's Theory.—Mr. Hodgson's Explanation.—Indications.—Means.
- Diseases of the Pulmonary Artery and Veins, and of the Coronary Artery and Vessels.*—*Pulmonary Artery.*—Ossific Deposits—rare.—Dilatation.—Signs.—*Pulmonary Veins.*—Coronary Artery and Veins.

WE now arrive, gentlemen, at the consideration of one of the most important diseases which affect the animal economy; I mean aneurism of the aorta. Such is the admirable description of this affection given by Laënnec, that I can do no better than follow it; exemplifying it, as I shall be enabled to do, by a series of preparations, illustrative of almost every point of the morbid anatomy of the disease described by that great pathologist.

By aneurism, we mean the dilatation of an artery, or its communication by an opening, more or less large, with a sac formed by its external coat, and occasionally also by the organs immediately surrounding the diseased vessel. The first species is called *true aneurism*; the second, *false consecutive aneurism*. A combination of the two species often also exists; that is, a true aneurism may be surmounted by a false consecutive one.

True Aneurism.

Morbid Anatomy. — True aneurism of the aorta is not an unfrequent disease, particularly in the ascending portion of the arch of that vessel. The disease commonly extends from the origin of the aorta to its descending part: the centre of its ascending portion often presenting the breadth of three or four fingers, is usually the most dilated. The convexity of the arch and the anterior part of the artery are generally more enlarged than its posterior parietes. When the dilatation occurs in the descending aorta, it forms an ovoid or fusiform tumour, and sometimes there are several of these tumours in the course of the same vessel. When the dilatation occurs at the space from whence the innominata arises, that vessel, and often also its primary branches, are similarly affected: the left subclavian artery, however extensive the aneurism may be, generally preserves its natural caliber. Occasionally, especially in old persons, the descending aorta, to its iliac divisions, becomes double its ordinary breadth; the ascending aorta and its arch are then also somewhat dilated.

Other arteries are subject to this species of simple dilatation: thus the carotid, at its emergence from its canal in the temporal bone, and the temporal artery to its minutest ramifications, have been seen in this state. Sometimes the arterial capillaries are also found enlarged, forming the principal part of erectile tumours: these have received various denominations, — as *nævus*, *fungus hæmatodes*, *varicose tumours*, and *aneurisms by anastomosis*.

Slight dilatations of the aorta have rarely received the name of aneurisms, that expression being reserved for larger dilatations: yet the disease in both cases is the same, the difference being only in degree. The largest true aneurisms form at the ascending portion of the aorta and its arch: they have been seen of various sizes, up to that of the head of a full-grown *fœtus*. When a true aneurism becomes large, a false consecutive aneurism often forms upon it, thereby augmenting its volume.

The internal membrane of the aorta is often affected in this disease; red points and slight cracks are seen upon it, and numerous incrustations are often placed between the internal and fibrous coats; these cracks, or fissures, correspond with the edges of the incrustations, and the internal surface of the vessel becomes therefore rough and unequal. On examining the parietes of the artery by a transmitted light, it will be seen to show various degrees of transparency, from the thinning of the fibrous coat produced at different points, in consequence of the action (probably mechanical) of the phosphate of lime upon it. In some cases these rough surfaces entangle portions of the blood, which then concrete: this matter accumulates, and occasionally

forms a series of layers of fibrine, similar to those we shall describe as contained in the sacs of false aneurisms.

False consecutive Aneurism

Is defined to be a tumour placed along the external surface of an artery, and communicating with it by an orifice of greater or less diameter.

The sac of a false is generally more unequal in thickness than that of a true aneurism. It is formed by the cellular coat of the artery, and is occasionally increased in its strength at different points, by cellular tissue, and by the organs around it. In other cases, it is almost as thin as a sheet of paper. Its internal surface is very rough, but the orifice of communication is almost always smooth.

False aneurisms are formed most commonly in the descending aorta. When they are placed in the ascending part of that vessel, or its curve, they are generally superadded or attached to a true aneurism. The descending aorta, however, is rarely dilated at the point from which the false aneurism springs: it is often, indeed, contracted.

Causes of Aneurisms.—The simple dilatation of an artery, or true aneurism, is no doubt occasioned by the impulsion of the blood against the parietes of the vessel, already weakened by disease, as by osseous or cartilaginous deposits; and we find that the dilatation is always greatest where the force of the impulsion is more particularly directed. Thus, the ascending aorta and the anterior part of its arch are always most enlarged; whilst the posterior parietes of the arch rarely enter into the

aneurismatic dilatation, in consequence of the greater resistance they afford to the impetus of the blood, from their being placed against the unyielding dorsal vertebræ.

Although the formation of a true aneurism can easily be explained in this manner, yet many opinions have been entertained as to the modes in which a false membrane originates. Thus some have supposed, in consequence of the smoothness of the orifice of communication, that the internal membrane of the artery formed a hernia through a rupture of the fibrous coat of the vessel, and lined the whole of the aneurismal sac as it gradually extended and increased in volume; so that the sac was formed of the external cellular investment of the artery and of its internal tunic. Such was the opinion of MM. Dubois, Dupuytren, and many others: but it is impossible to trace the smooth internal membrane beyond a few lines within the sac, except the aneurism be very small — no larger than a nut, for instance. In all probability, as the aneurism enlarges, the internal membrane breaks at a short distance from the orifice of communication; the internal part of the sac then becomes rough and irregular. An aneurism supposed to be formed in this manner was called *aneurisma herniosum*, *aneurisma herniam arteriæ sistens*, or *mixed aneurism*.

Scarpa believed that no aneurism took place without the rupture of both the middle and internal tunics of the artery, and that the sac was formed of the cellular coat of the vessel alone. He went so far as to say that the true aneurism of authors had no existence — that the dilatation of the aorta near

the heart could not be called an aneurism, and that that dilatation was never to be found in the remaining part of the course of the vessel. It is singular that the latter opinion should be held by so great a pathologist. You see before you, on the table, preparations of dilatations of the descending aorta and of the primitive iliacs — dilatations containing layers of fibrine as distinct as those we shall presently find existing so commonly in false aneurisms.

But that the opinion of Scarpa is correct, as to the mode in which false aneurisms originate, is most probable: that is, that it is caused by a rupture of the middle and internal coats of an artery, through which the blood insinuates, and gradually, by its accumulation, dilates the distensible cellular coat of the vessel so as to form a sac.

The rupture of these two coats may, though very rarely, be distinctly seen. M. Ambroise Laënnec has detailed a singular instance of this state. The arch of the aorta was encrusted with osseous deposits, and was also dilated to the volume of a moderate-sized apple. The descending aorta, at about two inches from its origin, presented a transversal slit through its internal and fibrous coat, occupying about two thirds of the contour of its cylinder; the edges of this slit were thin, unequal, and torn; the cellular coat was healthy, but separated from the fibrous, from the point of rupture to the primitive iliacs, so that the cavity of the artery was divided into two parts by an intermediate partition. This separation of the fibrous coat was not complete; it occupied about one half or two thirds of the surface of the vessel, and turned in

some parts around its cylinder. The space between the separated fibrous and cellular coats was filled by clots of blood, or by fibrinous or polypiform concretions. Mr. Guthrie has met with two cases analogous to this, and Mr. Hodgson mentions a similar instance.

I present you, gentlemen, with two beautiful specimens of this sudden rupture of the internal and fibrous coats of the aorta; for the one I am indebted to Dr. Langmore, of Finsbury Square; for the other, to Mr. Bennett, of the Commercial Road. The first occurred in a woman, who, during a slow convalescence from typhus fever, became from some cause suddenly alarmed; she instantly fell into a state of syncope, from which she shortly rallied, but died in about twenty hours after.

On examining the aorta, you perceive that at about an inch from its origin, its internal and middle coats, to nearly one half of the circumference of the vessel, are ruptured transversely, the edges of the torn membranes being thin and unequal: the blood passed through the rupture, and completely separated the cellular from the fibrous tunic, and formed a continuous clot between them; the clot, you see, is preserved, and forms a fourth coat completely round the vessel. The artery must have remained in this state for many hours: at last, the blood, by continually distending the cellular coat, ruptured it also nearer to the origin of the aorta, so that that fluid poured into the pericardium, filled it, and prevented the further movements of the heart. The second specimen was from a man who suddenly fell in the street in a state of syncope; he recovered for a short time,

but died five hours afterwards. The pericardium was found filled with blood, and the same rupture was found as in the preceding case; it was, however, not in a transversal direction, but extremely irregular, and the bursting into the pericardium was by a number of small orifices. You observe also in both these preparations the internal surfaces of the aorta are diseased, in consequence of cartilaginous deposits between their internal and fibrous coats.

In these cases the ruptures were too large, and the effusion of blood too sudden, to dilate the cellular coat into an aneurismal sac; but solutions of continuity are effected slowly in the internal membrane of the artery, either by small pustules, slight ulcerations, and, above all, by the partial separations of osseous or cartilaginous incrustations from the fibrous coat. These separations form cracks on the internal surface of the vessel; small coagula are lodged within them, by which the fibrous membrane becomes still more diseased, and finally perforated; the coagula increase, gradually press upon the cellular coat and distend it; the pressure of the circulating blood increases that distension; the cellular coat still farther enlarges, and finally, though very slowly, forms a sac, the original diseased part of the vessel being the point at which the orifice of communication ultimately forms.

This is the mode in which the false consecutive aneurism is commonly produced although it is probable that when the sac is very small, and its internal surface is smooth, the internal membrane may have perforated the fibrous coat, distended with and lined its cavity.

Concretions of Blood in Aneurismal Sacs. — In all false, consecutive, and in many true aneurisms, the internal parietes of the aneurismal sac are lined by a series of layers of coagulated blood, evidently deposited at successive periods, and presenting different appearances, according to the date of their formation ; thus, those which are nearest to the orifice of communication with the artery are formed of blood more or less coagulated ; a little farther, the clots are drier, and mixed with a large proportion of fibrine ; deeper still, the layers are of a purer fibrine ; they are then white or yellowish, firmer, less humid, and more opaque ; below these, layers of a similar colour are often found, which have a certain degree of brittleness, are of the consistence of dried paste, and may easily be broken down by the fingers ; the last described layers are in contact with and adhere to the parietes of the sac. Sometimes they are softened to the consistence of "bouillie," and they are formed of fibrine, more or less advanced into a state of decomposition.

These are the characters of the fibrinous layers usually found in false consecutive aneurisms ; but there are also occasionally found others, which, when cut in fine slices, are semi-transparent : they are of a greyish-brown colour, having whitish veins ramifying within them. This substance is like horn softened by heat ; it is very compact, easily cut, and leaves no moisture upon the scalpel ; its thickness is frequently very considerable, especially in large aneurisms.

The blood often insinuates itself between these

fibrinous layers, and separates them from each other, so that fresh coagula are frequently found between deposits of older formation. The blood often also separates the external layers from the parietes of the sac, penetrates it at its weakest point, and occasionally destroys life in a moment by the quantity that is poured out.

These layers are generally numerous, in proportion to the size of the sac. False consecutive aneurisms are usually entirely filled by them, but that nearest to the opening into the artery often consists of a mere clot of blood, probably formed after death. In moderate dilatations of the aorta these concretions are rare, their formation being prevented by the rapid current of blood passing through the vessel. I have occasionally, however, seen the aneurismal dilatation so nearly filled with coagulated layers of blood, that the passage of that fluid was considerably obstructed. You perceive, gentlemen, that that was the case in this preparation, in which there is a true aneurism of the descending aorta, and one smaller one in each of the primitive iliacs.

The Effects of Aneurisms upon the surrounding Organs.—When the aorta is simply dilated, it rarely produces any serious effects upon the surrounding organs, except the dilatation be very considerable; but the smallest false consecutive aneurism, or even a true one, in which the dilatation is confined to one side of the vessel, forming a projecting tumour, may occasion the most serious consequences.

The effects of the aneurismal tumour, as it increases in volume, are to compress the organs in

contact with it. When the tumour is very large, or when, by its position, it becomes a cause of very considerable pressure, it often changes the position of the surrounding parts, which then apply themselves to it so as to form its external parietes. Thus, in an aneurism placed at the extremity of the thoracic portion of the aorta, or at the origin of the cæliac artery, the distended and flattened pillars of the diaphragm cover the lateral and even the anterior part of the tumour; the vessels, nerves, and cellular tissue, spread over the sac, as well also as the pleura or peritoneum.

The next effect of the compression of the aneurismal swelling is to attack the structure of the organ with which it may be placed in contact; thus, it may alter and destroy a portion of the pulmonary tissue, so that the aneurism may burst into the air-vesicles.

Aneurisms of the ascending aorta, or of its arch, often compress the trachea, or one of the bronchial tubes, and flatten them; the cartilaginous rings of these vessels become finally destroyed, and aneurisms, by bursting into them, cause a fatal hæmoptysis.

The œsophagus has been similarly compressed and perforated, producing a fatal vomiting of blood.

The heart may alter its position, if the aneurism be large; thus, if the disease be situate above it, that organ is pushed downwards; if below it, upwards; or it may be forced to the right or to the left, according to the position of the compressing cause. Aneurisms have been seen to burst into the pericardium; but these cases are rare.

I present you with a rare specimen of aneurism

of the ascending aorta, destroying the coats of the pulmonary artery, into which vessel it ultimately burst.

When an aneurismal sac ruptures into the pleuritic cavity, it is generally into that of the left side; it is very rare to find it open into the right.

Aneurisms have been seen to compress the thoracic duct, and occasion engorgement of the lacteals. Corvisart saw an aneurism of the ascending aorta compress the superior cava, so as to prevent the free return of blood from the head. The patient died in a sub-apoplectic state.

Aneurisms produce the most singular effects upon the bones,—they seem to destroy them as if by attrition; thus the false consecutive aneurisms of the aorta destroy the bodies of the dorsal vertebræ, leaving the intervertebral cartilages intact, and forming incomplete partitions traversing the back part of the aneurismal sac. It is needless to state, that the posterior portion of the sac has in this case been previously destroyed. True aneurisms have also occasionally produced the same effects upon the bones.

Aneurisms of the abdominal aorta rarely affect the bodies of the lumbar vertebræ, probably because of the greater freedom with which the sac can develope anteriorly.

True or false consecutive aneurisms often also corrode the sternum, perforate it, and form tumours, projecting beyond the surface of the chest. These tumours sometimes acquire a volume equal to that of the head of a full-grown fœtus, as you observe in these casts and preparations.

Aneurisms of the arch of the aorta, or the in-

nominata, sometimes project above the sternum, or force their way under the cartilages of the ribs on the right side. The bones in these cases are destroyed, although their cartilages are scarcely at all affected.

It is singular that it is not the largest aneurisms which thus destroy the sternum and form external tumours. Sometimes those of the size of an egg will produce this effect, and very large ones remain hidden in the cavity of the chest, although the anterior face of their sacs be closely compressed against the posterior surface of the sternum.

General Signs of Aneurism of the Aorta.—Perhaps there is no certain sign of thoracic aneurism except the pulsating tumour which it occasionally forms when it penetrates through the parietes of the chest. The signs of this disease are generally derivable from the effects it produces upon other organs by compressing them. Sometimes the very first evidence we have of the disease is a fatal hæmorrhage from its rupture. The general signs I am about to detail may be considered as equivocal.

There is not infrequently a difference in the state of the pulse in the radial arteries: this takes place when the aneurism compresses the left subclavian artery or the innominata, or when coagula fill the opening of either vessel, or when the tumour, by its pressure, changes the angle at which these vessels arise, and renders it more acute. When an aneurism compresses the trachea, a peculiar sibilating sound is heard, and the larynx and trachea are felt by the patient to be drawn downwards; there is also a sense of oppression

and dyspnœa, which diminishes when the tumour projects outwardly. Occasionally, also, when an aneurism of the descending thoracic aorta is destroying the bodies of the dorsal vertebræ, a sensation of terebration is produced in the back, as if a gimlet were perforating the bones.

All these symptoms form, however, but doubtful signs ; for inequality of the force of the two radial arteries is frequent in many healthy persons ; and the difference of the rhythm in these vessels might depend upon obstructions caused by clots of blood in the origin of the subclavians, as well as from an aneurism. The signs from compression may be derived from a variety of other causes : even the terebrating pains may be mistaken for rheumatic or neuralgic sensations. Laënnec has seen the pulsating tumour itself confounded with an elongated cerebriform swelling, which, after destroying the first bone of the sternum, projected through it.

Local Signs. — The most certain stethoscopic sign we possess of this disease is that of the strong pulsation of the aneurism, a pulsation which is isochronous with that of the ventricular contraction, but stronger than it. This impulsion is frequently coincident with the *bruit de soufflet*, and the *frémissement cataire*. The sound on percussion is dull also over that part of the chest which corresponds to the situation of the aneurism.

Laënnec lays much stress upon the aneurismatic pulsation being simple ; by which he means that it is not accompanied by the second sound, as in the beatings of the heart ; but it has occurred to me frequently to meet with the second sound in

aneurisms of the ascending aorta and its arch, although not so loudly as at the heart itself. No doubt this arises from the noise caused by the ventricular dilatation being communicated through the sac, which is often placed, and reposes, upon the heart itself. I have never heard the second sound in aneurism of the abdominal aorta.

Laënnec has stated that, when the ascending aorta, or its arch, are dilated, a pulsation is felt isochronous with the beatings of the pulse, and stronger than that of the heart, beneath the sternum, or below the right clavicle. Dr. Hope observes, that then there is a constant pulsation above both clavicles, at their sternal extremities, which is stronger on the right side if the ascending aorta be the seat of dilatation; and that this impulse is never communicated to the sternum except the aneurism be very large. He states also, that there is a hoarse and short bellows sound above the clavicles, if the dilatation be confined to the ascending aorta, and that it is also hissing and superficial upon the sternum; and that, finally, the harshness of the "bruit" is always proportionate to the roughness of the osseous inequalities formed upon the inner surface of the aorta.

Dr. Hope describes the signs of a false consecutive aneurism of the ascending aorta and its arch to consist in a pulsation both above and below the clavicles, but that it is the least impulsive in the latter situation. If the ascending aorta be the seat of the disease, these pulsations are most distinct on the right side of the sternum; if the arch, or the beginning of the descending aorta, they are

felt on the left side of that bone, and even on the shoulders and back. The sounds are not so loud as when the vessel is dilated. In large aneurisms the noise produced by the pulsations is dull and remote, and sometimes louder on the side of the neck opposed to the tumor than on that on which the tumor is situated. It is often loudest on the back; and when the disease affects the descending thoracic aorta, it is generally more evident posteriorly than anteriorly. If a strong rasping sound be heard also on the back, the disease may be considered as almost certainly present. The "fremissement cataire" is occasionally felt above the clavicles, but never above, except the ribs are perforated by the tumor. When the aneurism is large this sign often disappears. Dr. Hope has, with great discrimination, described the circumstances which rendered these signs fallacious, and the means by which these fallacies may be detected.

Thus, tumors in the anterior mediastinum may give the aneurismal impulse, but not the sound. Hydro-pericarditis may also occasion impulsion, but the shocks vary in force, and are not exactly synchronous with the beatings of the heart. The heaving movements of an aneurism are always felt in the same place; that from the fluid in the pericardium constantly varies. Hypertrophy and dilatation of the heart may be confounded with aneurism; but in the latter disease there are evidently two points from which the implusions proceed — the one from the aneurism, the other from the heart; in the former affection there is but one — the heart itself. The heart, also, when in a state of hypertrophy and dilatation, produces

a loud noise upon its contraction, but an aneurism a dull sound, like that of concentric hypertrophy. Enlarged glands situated above the clavicles may also cause impulsion, and simulate aneurism, but it is rarely accompanied by sound.

Aneurisms of the abdominal aorta are usually distinguishable by the stethoscope. The impulse is often enormous, always simple, and isochronous with the contractions of the ventricles. The noise accompanying these beatings is clear, sonorous, and often combined with the "bruit de râpe." Tumors lying upon the aorta frequently receive an impulse from the pulsations of that vessel, but then it is always infinitely less than in aneurisms. The aorta sometimes beats violently under the influence of nervous causes; but in these cases the pulsations are never permanent; and upon pressing the aorta with the stethoscope, they are found not to extend beyond the natural diameter of that vessel.

Treatment of Aneurism. — Corvisart attributed the occasional formation of aneurisms to certain tumors attached to the arteries, of the following characters: — Speaking of one of them, he states, "That this tumor was formed of a fibrous cyst, whose parietes were two lines thick; that it inclosed a substance of a less consistence than suet, and of a deep red colour, similar to clots of blood which had long formed, and adhere to aneurismal sacs. The external coats of the aorta were destroyed at the point corresponding to the cavity of the cyst; and the thickness of the parietes of the vessel was, in that part only, infinitely less than at any other point. Although he believed,

from the colour of the matter contained in the cyst, that it communicated with the cavity of the aorta, yet he could perceive no orifice of communication; he saw only a slight gray or livid stain, corresponding to the base of the cyst." He had observed two or three tumors bearing these appearances.

From these facts Corvisart inferred, that if the patient had lived a sufficient time, these tumors would ultimately have so destroyed the coats of the artery, that the blood would have freely passed into the cavity of the cyst, which then would be transformed into an aneurismal sac, which would gradually become larger as the blood dilated its cavity.

Mr. Hodgson has, however, attributed the formation of these tumors to a very different cause. An aneurism of the extremities is occasionally spontaneously cured by coagula completely filling its sac, and obliterating also the canal of the artery communicating with it up to the collateral vessel nearest the aneurism, after which these clots become converted into a fibrous tissue, which, with the vessel and the aneurismal sac containing them, gradually diminish in volume by the process of absorption. Mr. Hodgson believes, and his opinion is now generally received, that the tumors thus described by Corvisart are the remains of false consecutive aneurisms, spontaneously cured by a similar process.

Our indications of cure, then, should be to occasion the complete coagulation of the blood contained in the sac, so as to prevent any more of

that fluid from passing into it. We should, therefore, diminish the quantity of the blood, and the force by which it is circulated. These effects can sometimes be produced by the method proposed by Albertini and Valsalva; a method I have described in speaking of the treatment of hypertrophy, and hypertrophy with dilatation of the heart, to which subjects I refer you. The acetate of lead has also been recommended. I have used it, but cannot say with much good effect. The coagulation of the blood in the aneurism may also be attempted by the application of ice upon the tumour. I think it often retards its increase; but it unfortunately happens, that, when the disease has arrived at the state to form an external swelling, little can be effected by any remedy.

If the morbid anatomy of this disease has been well understood, I need hardly state that the cure of aneurisms caused by simple dilatations of the arteries can never be effected, inasmuch as the current of the blood passing through them must tend to prevent the formation of coagula. It is true that they occasionally, although very rarely, do form; but it is scarcely possible that they can remain in such a state of repose as is necessary for their organisation and permanent attachment to the parietes of the aorta, by which the natural caliber of that vessel can be restored. It is only in false aneurisms, where the sac is placed external to the artery, that such a cure can be hoped; for then the blood in the sac is thrown out of the general circulation; it has time, therefore, to coagulate and become organised: and the whole may ultimately be considerably, if not com-

pletely, absorbed, without affecting the flow of blood through the artery itself.

DISEASES OF THE PULMONARY ARTERY AND VEINS,
AND OF THE CORONARY VESSELS.

Pulmonary Artery. — Diseases of this vessel are of rare occurrence; they consist of malconformations, — of which I shall hereafter speak, — of osseous incrustations, and of dilatation of the parietes of the vessel.

Ossific deposits are very seldom found in the parietes of the pulmonary artery; but dilatations of this vessel are not uncommon in persons subject to chronic diseases of the lungs. The artery has been seen at its origin of the breadth of three fingers. Ambrose Paré describes a case in which it was dilated to the size of the fist. The vessel in the latter case was also incrustated with ossific matter. No well recorded instance of false consecutive aneurism of the pulmonary artery is known.

Dr. Hope describes the signs of a case of dilatation of the pulmonary artery, which occurred to him, to have been as follow: — Pulsation, with *frémissement cataire*, between the cartilages of the second and third ribs on the left side, decreasing downwards, and not appreciable above the clavicles. This was accompanied by a loud, superficial, harsh, and sawing sound, which was heard above the clavicles, and upon the precordial region: the sound was loudest upon a prominence found between the second and third ribs. This state of the artery was combined with hypertrophy and dilatation of the heart.

Pulmonary Veins. — Chaussier describes the case of a young woman in whom the left auricle was largely dilated, in consequence of obstruction of the mitral valve. The pulmonary veins were greatly enlarged; and one of those from the left lung was ruptured to three quarters of an inch of its extent, at its emergence from the lung.

Affections of the Coronary Vessels. — The coronary arteries occasionally are observed ossified, and sometimes so completely, as to obliterate them at several points.

In hypertrophy and dilatation of the heart, these vessels are often dilated in the whole of their extent.

The coronary veins occasionally dilate: their distention is not unequal, like that of varicose veins of the limbs, so as to form a knotted appearance, but their bendings are considerably increased, so that their length is really augmented, as well as their diameters. The veins are commonly found in this state when the heart is hypertrophied and dilated.

LECTURE XXVI.

NERVOUS DISEASES OF THE HEART AND ARTERIES.

Angina Pectoris. — Symptoms — Frequency. — *Theory* of the Disease. — *Treatment.* — Case.

Nervous Palpitations of the Heart. — Symptoms. — Difference from those arising from organic Disease of the Heart. — *Treatment.*

Spasm of the Heart — accompanied by “Bruit de Soufflet” and “Frémissement Cataire.” — Symptoms. — *Neuralgia* of the Arteries. — *Augmentation* of arterial Pulsations. — Extent and Symptoms. — *Spasm* of the Arteries, combined with Bruit de Soufflet.

Treatment of Neuralgia of the Arteries.

Displacements of the Heart. — *Malconformations of the Heart and great Blood-Vessels.* — Necessity that the Blood should be sent into the Lung during its Circulation. — *Single Heart*, with no pulmonary communication. — Proposition, that the arterial and venous Blood should not intermingle in their Vessels on the Heart. — *Single Hearts.* — *Imperfect double Hearts.* — Imperfect Septum of Auricles. — Singular Case. — Imperfect Septum of Ventricles. — Transposition of Vessels. — Signs. — Theory of the Coldness and Blueness of Skin.

By nervous disorders of the heart and arteries, we mean the affections of those organs which do not depend upon organic changes of their structure. The symptoms of these disorders are not of permanent duration, and they appear at indeterminate periods. We shall describe them in the following order: —

1. Angina pectoris, or neuralgia of the heart.
2. Nervous palpitations.
3. Spasm of the heart, with bruit de soufflet and frémissement cataire.
4. Nervous affections of the arteries.

ANGINA PECTORIS.

This disease usually commences with a sensation of pain, oppression, weight, or constriction, at the cardiac region. In severe cases, the patient feels as if the heart were violently squeezed; and he then supports himself by firmly embracing any fixed object; at the same time he retains his breath, for the purpose of relieving himself from this sensation. As the fit continues, the dyspnœa often becomes extreme; sanguineous congestions are formed towards the head and face; occasionally syncope or convulsions supervene, and the patient manifests the most unceasing jactitation and apprehension of imminent suffocation. I have seen instances of sudden death occurring in the midst of the most agonising distress. The access, after lasting for an uncertain time, usually ceases, to recur at an indeterminate time. Certain circumstances often invariably bring it on in the same individual at any period. Thus, if he endeavour to walk a little quicker than usual, or ascend an inclined plane, the sense of obstruction, or weight, or squeezing, and the dyspnœa, immediately come on, and he is obliged to rest a few moments, and these feelings then generally suddenly disappear. Emotions of the mind will also occasionally reproduce the same symptoms.

Previous to and during the access, a painful numbness is felt in the inside of the left arm: it is rarely experienced in both arms, or on the whole of the left side of the body; and more rarely still in the right arm only, or in the four extremities. This sensation is usually felt along the inside of the

left arm, from the axilla to the elbow ; sometimes it continues in the course of the ulnar nerve to the little finger, and to the ulnar side of the ring finger. These pains often follow, also, the course of the left anterior thoracic nerves, and are therefore felt upon the left anterior parietes of the chest ; they vary not only in situation, but also in character ; occasionally there is a sensation of mere numbness without pain ; sometimes the pain and numbness are combined. The increase of the sensibility of the skin and mammæ is often so great, that the slightest pressure is excessively painful. I have met with patients whose torments have been so great, that they described their flesh to feel as if torn by the talons of an animal. These signs finally disappear for a time, leaving only a slight numbness or uneasiness of the parietes of the chest and of the inside of the left arm.

I have often abserved, that these neuralgic sensations attacking the parietes of the chest occurred alone, especially in a slight degree, without any feeling of obstruction, weight, or squeezing in the region of the heart ; the anterior thoracic nerves being then only the seat of the neuralgia. Sometimes the cardiac symptoms are present, unaccompanied by any pain on the surface of the chest or in the arm. When the junction of both sets of symptoms obtains, than the *angina pectoris* may be said to be completely formed.

Angina pectoris, in a moderate degree, is by no means an uncommon affection ; but the disease, in its most violent form, is very rare. In ten years, I have seen but two instances of death occurring during the violence of the paroxysm.

When the anterior parietes of the chest and the arm are affected, there can be no doubt that the disease depends upon some peculiar condition of the nerves supplying these parts, — a condition similar to that which occurs in the facial nerve in *tic douloureux*, and to which the term *neuralgia* has been applied, — an expression simply denoting the fact of pain or aching of the nerves, without implying any notion of their specific lesion. But when the heart is also affected, various opinions have been entertained as to the cause of the symptoms.

Heberden and Parry, Bertin and Kreysig, believed the disease to depend upon ossification of the coronary arteries. The Germans and Italians are of opinion that it is consequent upon some organic disease of the heart; but the only means by which such questions can be decided — the scalpel — has distinctly shown, that, although *angina pectoris* may coincide with every organic change of the structure of the heart, yet that it exists often also independently of any apparent lesion of that organ. The cause of the disease must therefore be referred to some inexplicable state of innervation. It is probable that, when the cardiac symptoms exist alone, the disease depends upon a lesion of the eighth pair of nerves, and of the filaments sent to the heart by the great sympathetic. When the anterior parietes of the chest and the inside of the arm are only affected, the anterior thoracic, the internal cutaneous, and the ulnar nerves, are the seat of the disorder; and finally, in those cases when the heart, lungs, surface of the chest, and inner side of the arm, are thus affected, these combined disorders are the result of a simultaneous

neuralgic state of all the nerves supplying these parts.

Treatment of Angina Pectoris. — Angina often affects individuals in whom the digestive functions are in a state of derangement. It is necessary, in such cases, to pay great attention to the stomach and bowels, obviating accumulations by gentle emetics and purgatives, correcting acrid secretions by antacids, — as the soda, chalk, and magnesia, — and giving tone by the employment of tonics, as iron, quinine, &c., combined with carminatives.

When the attack has arrived, the patient should be placed in a state of repose, — at least as much as his irritability permits; for sometimes his extreme distress forces him to quick movements, or violent strainings, which no effort of volition can control. Bleeding, at this moment, has been recommended, if the patient be plethoric; but it is a remedy of suspicious value in most cases. Opium, the diffusible stimuli, — as æther, camphor, &c., — the foetid gases, — as asafoetida, are often highly advantageous during the access.

During all periods of this disease, I have long been in the habit of employing the belladonna to the surface of the chest, and generally with excellent effect, especially if the angina be not complicated with organic lesions of the heart; although even then it affords temporary relief. I prescribe a plaister, of the size of the hand, formed of one part of extract of belladonna, mixed with four parts of soap plaister, and place it upon the region of the heart. I once had an opportunity of seeing the full effects of this medicament in a most unexpected manner.

I was requested, about three years since, to see a gentleman who had just arrived in London from Dublin : I found him labouring under a severe form of angina pectoris, — so severe that he could bear no movement whatever, except upon the water, without its producing a violent access ; he had therefore lodged himself in a street close to the Thames, for the purpose of readily obtaining the only exercise he could enjoy. Upon a most careful examination of his heart, I could find no organic disease, nor could an eminent physician, Dr. Billing, who is a practised auscultator. The usual belladonna plaister was applied to the chest of the patient about the middle of the day ; towards evening alarming symptoms arose : he became slightly amaurotic, his head was confused and giddy, and he articulated his words with great difficulty. The plaister was immediately taken off, and the surface of the skin beneath it was found covered by ulcerations of various sizes, produced by the action of the tartar emetic ointment ; a circumstance which, of course, we had been totally unacquainted with. He recovered the integrity of his functions in a few hours, but the symptoms of angina had totally disappeared. The patient returned home in the mail coach, the movement of which, for a long time before, he could not have borne, and from that time he has remained perfectly free from his disease.

Laënnec had great confidence in the magnet in angina pectoris. He applied a plate of steel, of a line in thickness, and strongly magnetised, upon the left precordial region, and a similar one exactly opposite, on the back, so that their poles were

opposed to each other, and the magnetic current traversed the parts affected. He frequently applied a blister under the anterior plate. I have tried this plan, and have thought, with good effect.

Blisters, tepid shower-baths, and change of air, are often of utility also in this affection.

Nervous Palpitations of the Heart.

Purely nervous palpitations of the heart are often more distressing than those which arise from organic causes; for, instead of diminishing by repose, they are often aggravated by it. They usually commence at the beginning of the night, and the patient, for many hours, obtains no sleep. A moderate exercise often diminishes these palpitations, or, at least, distracts the attention from them.

Palpitations consist in the beatings of the heart being sensible to the patient; the impulsion, noise, and frequency of the pulsations of the organ are then increased; a sensation of internal agitation, particularly in the head and abdomen, is consequent upon this state. The urine is generally clear and limpid during the access. The duration of these palpitations varies; when they arise from a sudden emotion, they may pass off immediately: in young, plethoric, nervous persons, they may last for years, with but little intermission.

It has been supposed, that purely nervous palpitations must ultimately induce hypertrophy, or hypertrophy with dilatation of the heart: this fact, although probable, has not yet been proved.

Nervous palpitations differ from those produced

by hypertrophy, or hypertrophy and dilatation of the heart, inasmuch as in the former the application of the stethoscope shows that the organ is not really of considerable size, the noise of the pulsations is not heard to a great extent upon the surface of the chest, and, above all, the shocks communicated by them have but little impulsion, for they never sensibly move the head of the observer. Sanguine congestions rarely form, except in old persons.

These palpitations are very frequently found in hypochondriacs and hysterical women. Their treatment merges into that recommended for these diseases. Warm or cold baths, according to the season, may be used. Digitalis, combined with antispasmodics and tonics, I have often seen produce advantageous effects. The hydrocyanic acid has been also employed with some success. Bleeding is rarely beneficial, but is often hurtful in nervous persons, except they be plethoric. A change of air, combined with constant pleasing occupations, is, perhaps, the most useful means of combating this disease.

Spasm of the Heart, accompanied by Bruit de Soufflet and Frémissement Cataire. —The bruit de soufflet of the heart, although frequently connected with organic lesions of the organ, may exist independently of them. It then depends upon some peculiar state of innervation. This sound is found particularly in hypochondriacs of sanguine and nervous constitutions. Frequently it is present in some artery at the same time. Often the bruit alternates from the artery to the heart, and from the heart to the artery. It is sometimes con-

tinued, occasionally intermittent; in the latter case it is produced by the slightest mental emotions: even coughing, or breathing deeply, may be sufficient to cause it. The symptoms are usually serious in proportion to the intensity of the sound, to its duration, and to its extent in the heart and arteries. When the bruit is marked, and continued in the heart only, there is always more or less dyspnœa and general weakness: these symptoms are aggravated if the *frémissement cataire* be present also. There is usually but little nervous agitation, especially if the patient be in a state of repose; but in walking quickly, dyspnœa is soon produced, and, in severe cases, the head becomes affected by exercise as well as the lungs.

Nervous Affections of the Arteries. — These disorders may be considered in three points of view: — 1st, arterial neuralgia; 2dly, augmentation of arterial impulse; 3dly, spasm of arteries, with *bruit de soufflet* and *frémissement cataire*.

1. *Neuralgia of the Arteries.* — Pains, more or less violent, continued or intermittent, sometimes follow the course of the arteries, and appear to be seated in the nervous net-work furnished by the ganglionic system surrounding these vessels. These pains are commonly found in hypochondriacs and hysterical women. The treatment recommended for angina may be employed here. A blister applied over the affected artery is very often efficacious.

2. *Augmentation of Arterial Impulsion.* — It occasionally happens that the pulsations of one of the carotids, temporal or radial arteries, are

stronger than those of the other. In many persons in health there is a perceptible difference, the right pulse being stronger than the left. Sometimes the radial arteries will pulsate alternately, with a different force.

The abdominal artery is often the seat of this augmented impulsion: the vessel then always appears increased in its fulness also.

When the impulsion exists only in a small or middle-sized artery, it scarcely affects the general health, except it occurs in consequence of inflammation of the parts which that artery supplies: thus, when the hand or fingers become inflamed, the digital, radial, cubital, or even the brachial artery, beat more strongly than those of the opposite or healthy side. Anormal pulsations of the carotids often accompany nervous affections.

Nervous palpitations are sometimes combined with increased impulsion of the arteries of the whole body: the beatings of these vessels are then universally felt, and very small arterial branches often become perfectly visible under these circumstances.

These palpitations, when they affect the aorta, or any portion of it, always produce sensations which are more less painful. When the ascending aorta is thus disordered, it causes a certain degree of dyspnœa, anxiety, and disposition to syncope; its beatings are heard above the middle part of the sternum, stronger and more noisy than at the heart itself. If the descending aorta be the seat of the palpitations, the symptoms are nearly the same: the pulsations can then also be heard with greater distinctness on the back, and particularly on the

left side, near the vertebræ, than in the precordial region.

Nervous palpitations are more commonly found in the abdominal aorta than in the rest of the course of that vessel. Gaseous accumulations, forming tumours, often arise over the artery, and, as the pulsations are communicated through them, they frequently simulate aneurisms; but these swellings and pulsations rarely remain above a few days, and upon examination with the stethoscope, the impulse is never strong, bounding, and extensive, like that of aneurism. The pulsations also are confined to the natural caliber of the artery.

Spasm of the Arteries, combined with Bruit de Soufflet. — When the bruit de soufflet exists in a moderate-sized artery, and is found in only a small part of its course, and particularly if it be intermittent, it is usually accompanied by slight nervous agitation and an acceleration of the pulse. This state is especially found in young hypochondriacs of sanguine or sanguine-lymphatic temperaments, under the slightest exercise or mental emotion. The bruit is usually distinguishable in the subclavian arteries — more rarely in the carotids — more frequently on the right side than on the left. It commonly occurs in persons attacked by disease of the heart, or nervous palpitations.

When the bellows sound is situated in the aorta, and particularly in its abdominal portion, there is great anxiety, and a tendency to lypothymia, from the slightest causes: the pulse is then always quickened.

When the two carotids are affected, especially if the *frémissement cardiaque* be felt also, the same

symptoms obtain, although in a less degree. When the sound exists at once in the heart, the aorta, the carotids, subclavians, brachial and femoral arteries, the anxiety of the patient becomes extreme, the respiration is oppressed, the pulse is frequent, and sometimes he experiences the sensation of great internal heat, although the skin, &c. indicates no sign of fever. This state is attended with considerable danger, and death may even be the consequence.

If the *bruit de soufflet* be very intense in a number of arteries at once, the *frémissement cataire* is usually perceptible in some of them; the latter phenomenon is not, however, always found to be in proportion to the intensity of the bellows sound, nor to its extent, for it sometimes occurs in one of the carotids when the *bruit* is very feeble.

In many cases where the *bruit de soufflet* is tolerably evident in the arteries, the pulse of the radial arteries produces a trembling or trilling vibration, similar to that which a tight metallic string, slightly struck, would give to the end of the finger. This is probably but a minor degree of the *frémissement cataire*, yet it is often found where the *bruit de soufflet* is present in some arteries, and the *frémissement cataire* in none. It is sometimes also present when there is neither the bellows sound nor *frémissement cataire*; but then the former sound can often be produced by making the patient walk gently, or inspire or cough forcibly.

This trembling, or trilling vibration, can often be produced by a certain degree of pressure upon the artery with the finger; it is then sometimes felt for a moment, and then lost. If the pressure

be increased, it instantly re-appears; but it is extremely difficult to seize and continue the precise degree of compression necessary to render the sensation, under these circumstances, permanent, or to produce it at will.

The bellows sound may exist in the highest degree with or without the *frémissement cataire*, either in the heart or arteries, without any increase of their impulsion. When these circumstances are united, the above described symptoms become infinitely more marked.

Treatment of the Neuralgia of the Arteries.—When the arterial impulsion is greatly increased, bleeding is indicated; and it is often by that means alone, frequently repeated, that any relief can be obtained; but abstraction of blood should be very cautiously effected if the *bruit de soufflet* exist without impulsion. Tepid warm and shower baths, of that degree of temperature that the patient may feel ultimately a slight sensation of cold, are commonly very useful. Laënnec asserts, that he has occasionally found success by the use of the magnet, when the *bruit* existed only in the heart and aorta. If the *bruit de soufflet* is found without increased impulsion in pale and cachectic subjects, then iron, the foetid gums, and tonics, often produce good effects. A moderate diet, and abstinence from all kinds of stimulus, should be recommended.

Displacements of the Heart.

The heart is retained in its natural position by the diaphragm, the mediastinum, and the habitual

plenitude of the chest ; but the following circumstances may force it to deviate in various directions : —

The organ may be thrown to the right side by effusion of fluid or air, or by large tumours placed in the left pleuritic cavity.

It may deviate from its position farther to the left side, by the above causes operating from the right pleuritic cavity.

It may be forced *upwards* by aneurisms of the abdominal aorta, by tumours, and by ascites.

It may be forced *downwards* by the compression of an aneurism of the ascending aorta, or its arch, or by any other voluminous tumour situated above it. The heart, also, has been seen in this situation without any obvious cause : this is the *prolapsus cordis* of the older authors.

Malconformation of the Heart and great Blood-Vessels.

A circumstance essentially necessary to the healthy state of the circulation is, that the blood should have free access to the lung during a portion of its course round the system. If, by any derangement or alteration of the heart or its vessels, that fluid is prevented from entering into the pulmonary organs, death is the immediate result. The case of malconformation I shall first speak of, is that in which there is no communication whatever between the heart and lungs : it is what is called a single heart, and its parts are arranged in the following manner : —

The two venæ cavæ enter into the auricle in the

usual way ; the auricle communicates with a ventricle, from which the aorta arises ; there is but one auricle and one ventricle, an aorta, but no pulmonary artery or veins. The blood, therefore, in this case, can never be renovated by the contact of the atmospheric air ; it is therefore venous in the systems of the arteries and veins, and the subject can live but a very short time after birth.

It is necessary also, for the healthy state of the circulation, that the arterial and venous bloods should be kept separate and distinct from each other ; thus it is that they flow in different sets of vessels, the arteries and veins ; thus it is they are prevented from intermingling in the heart itself, by the septa of the auricles and ventricles. If by any alteration of the structure of the vessels, or of the heart, these two fluids can intermix, then disorder will be the result. If the mixture occur from a lesion of the arteries and veins, the affection is generally local, and but of little consequence ; if it occur in the heart, it always occasions, sooner or later, a fatal result.

A vein and artery are sometimes seen to communicate, so that the blood flows directly from one into the other. This constitutes a varicose aneurism ; but the disease is confined to the lesed parts only ; the quantity of blood intermingling is insufficient to disturb the general circulation. Occasionally the smaller arteries and veins open and communicate with each other, as in certain *nævi* ; but the consequences here again are only local, the system remains undisturbed.

But when the admixture of arterial and venous blood takes place in the central organ of the circu-

lation, then the most serious results occur, in consequence of the quantity of these different fluids which become mixed, and by which union each of them is deteriorated, and unfitted for the specific functions which it has to perform. All the malconformations I have to describe have for their consequence the mixture of the arterial and venous bloods in greater or less proportion, as well as the difficulty of transmission to the pulmonary organs.

Malconformation of the heart may be divided into two classes — 1. *Single hearts*; 2. *Imperfect double hearts*.

1. *Single Hearts*. — The following is the usual structure of a single heart: — The two venæ cavæ and the four pulmonary veins enter into an auricle; the auricle opens into a ventricle, from which arises an aorta; the aorta soon gives off two pulmonary arteries, which proceed to the lungs, and then the vessel continues in its usual course.

In this case there is a perfect mixture of the arterial and venous bloods. The black blood passes into the auricles from the two cavæ, and mixes with the red blood flowing from the four pulmonary veins into the same auricle; the united fluids are propelled into the single ventricle; the aorta then receives them, and transmits these fluids all over the arterial system, and to the lungs also, by the two pulmonary arteries arising from it.

Two or three modifications of this form of single heart have been observed, but they are but slight. Thus, in the above case, two pulmonary arteries arise from the aorta; sometimes, however, there is only one, which afterwards divides into two branches; and occasionally the aorta and pulmo-

nary artery originate in the single ventricle by a common trunk.

2. *Imperfect double Hearts.*—These imperfections consist in the incompleteness of the septum of the auricles, or of the ventricles. In either case the arterial and venous blood must intermix.

Species 1.—Imperfect Septum of Auricles.—The foramen ovale is often found a little open; but from the equal pressure of the fluids in the two auricles, and from the slightness and obliquity of the opening, rarely any intermingling takes place. I shall state the following case of a patescent foramen ovale, which I believe to be unique; at least, I know of no such case on record.

I was requested to see a young man of about eighteen years of age. I found him labouring under the most severe dyspnœa; his face, body, and limbs were œdematous, cold, and of a dark slaty purple tint; the beatings of the heart were strongly impulsive, and extended over the greater part of the parietes of the chest; the lungs were infiltrated with serum; the “rhoncus crepitans” was distinct; his expectoration was pituitous, and often tinged with blood. Upon enquiring into his history, I was told that up to the age of twelve years he enjoyed the most perfect health; that at that period, on using violent exertion at cricket, his breathing suddenly became very difficult, and he coughed up a large quantity of blood. He had gradually become worse up to the time I saw him; he had suffered therefore during a period of about six years from the original attack. He lived but a very short time longer. On a post-mortem examination, the heart was found large and hyper-

trophied; the edges of the foramen ovale were smooth, and so widely opened, that the thumb could easily pass through, as you perceive in this preparation.

The violent exercise which this lad had made at the age of twelve years, must have caused the rupture of the partition between the auricles, and admitted the passage of the blood from one to the other: the disease could not have been congenital, for the individual was in perfect health for so many years after his birth.

The next case of imperfect auricular septum is more complex, at least, in the explanation of the alteration of the current of the circulation through the heart. It consists in the foramen ovale not only being open, but the ductus arteriosus also. Under these circumstances, the blood must have passed in the following way:—A large proportion, let us say half of the blood, was transmitted into the right ventricle, and from thence into the pulmonary artery into the lungs: a diminished quantity was therefore sent into the latter organs, and we shall see how a compensation was afforded. The other half of the auricular blood traversed the foramen ovale into the left auricle, by which the quantity of blood was there preternaturally increased; the mixed bloods then passed into the left ventricle and into the aorta; and as the ductus arteriosus was open, the superfluous proportion of mingled aortic blood was sent through it into the pulmonary artery, by which the deficiency was made up of that transmitted into the latter vessel by the right ventricle. A consequence of this state of things is as follows:—If there be a due relation between

the diameter of the open foramen ovale and the caliber of the ductus arteriosus, so that a sufficient supply of blood be conveyed to the lungs, life may be sustained for a considerable period.

I present you a preparation of a singular malconformation, in which the foramen ovale is open, the pulmonary artery impervious at its origin only, and the ductus arteriosus widely patescent: the right ventricle is so completely in a state of concentric hypertrophy, that its cavity would scarcely contain a small pea.

In this case the whole of the venous blood must have passed from the right auricle into the left, and the mixed fluid from thence into the aorta. The circulation then divided into two currents; the one through the ductus arteriosus into the open part of the pulmonary artery, the other continued its course along the aorta. You will notice that the blood which had been transmitted through the ductus arteriosus in these cases flows in a different direction to that which occurs in the foetal circulation; for in this malconformation the current must have been from the aorta into the ductus arteriosus. In the foetus, its course is from the pulmonary artery into the aorta.

Species 2.—Imperfect Septum of Ventricles.—A malconformation of this species has been observed in which a trunk common to both the aorta and pulmonary arteries arose from both ventricles, in consequence of the septum being incomplete at its upper part. The aorta formed a sudden curve, and descended to supply the body and lower extremities only. A second aorta proceeded from its ordinary situation, ascended and formed its

curve giving off the innominate, the left sub-clavian, and carotid arteries; then becoming impervious, and terminating in the form of a ligament in the parietes of the thoracic portion of the descending aorta previously described. In this case the septum of the ventricles being imperfect, the arterial and venous bloods must have passed into both the aorta and pulmonary artery.

The septum of the ventricles is sometimes imperfect in its centre, so that a round and smooth orifice of communication has existed between the two cavities.

The preparation I hold in my hand beautifully shows a singular transposition of the vessels and cavities of the heart. The two cavæ, you perceive, enter into the right auricle as usual; the foramen ovale and the ductus arteriosus are widely open; the aorta arises from the *right* ventricle, whose parietes are as thick as those of the left generally; the pulmonary artery arises from the *left* ventricle, the walls of which cavity are thin, and similar to the ordinary state of the right ventricle: so that in this case the blood contained in the right auricle must have divided into two currents, one of which passed into the right ventricle, and from thence into the aorta; the second into the left auricle, ventricle, and pulmonary artery. When arrived at the latter vessel, that fluid again divided into two parts; one proceeded to the lungs, the other through the ductus arteriosus into the aorta; by which a compensation was effected for the diminished quantity of blood sent into the latter vessel by the right ventricle.

The symptoms of these malconformations con-

sist in violent palpitations, irregular pulse, dyspncea occurring in paroxysms, panting, coughing, screaming. Serous effusions and hæmorrhages frequently take place. The cerebral functions become disturbed; torpor, coma, convulsions, or paralysis, may supervene. The digestive functions become also disturbed, and the little patients finally arrive at a state of extreme marasmus. To these symptoms are frequently superadded an appearance of blueness or lividity over the body, and a general coldness over its whole surface.

The theory of this unusual coldness and livid colour of the body, is as follows:—When two substances enter into chemical combination, the specific heat of the resulting compound is different from that of its constituents. The respiratory and circulatory movements bring together two fluids, air and venous blood, each containing a certain quantity of specific heat. By the chemical action of these fluids upon each other, two compounds result, carbonic acid and arterial blood; the first having less specific heat than air, the second more than venous blood,—the sum of their specific heats being less than that of the air and of the venous blood. A portion of this heat becomes sensible, and is expended partly on the air, partly on the lungs themselves, and partly on raising the sensible heat of the arterial beyond the venous blood. When the arterial is converted into venous blood in the capillaries, a portion of its specific heat is also evolved, becomes sensible, and gives rise to what is called animal heat.

The lungs may be considered as a stove placed nearly in the centre of the body; the air and the

venous blood are the fuel which supplies it. If either of these fail, the body necessarily becomes cold. The air is almost always present in due quantity; for malconformations rarely affect the trachea, or the bronchial tubes. The venous blood occasionally either does not arrive at all at the lungs, or it is not carried there in sufficient quantity, in consequence of some of the malconformations I have just described; that fuel then fails, and the body becomes cold; so that the coldness of the body becomes a measure of the diminution of the quantity of the venous blood sent into the pulmonary organs.

The greater part of the blood, and, indeed, occasionally the whole, becomes finally venous, in consequence of its insufficient exposure to the air. The rosy hue of health is then lost, and the skin is tinted blue, dark purple, or livid, from the transmission of the colour of the venous blood through the cutaneous capillaries.

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