Researches on the nature, pathology and treatment of emphysema of the lungs : and its relations with other diseases of the chest / by A.T.H. Waters.

Presented by the Author.

RESEARCHES

ON THE

NATURE, PATHOLOGY, AND TREATMENT

OF

EMPHYSEMA OF THE LUNGS,

AND ITS RELATIONS WITH OTHER

DISEASES OF THE CHEST.

BY

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PREFACE.

An extended inquiry into the anatomy of the human lungs naturally led me to a consideration of their pathological conditions. Amongst the subjects to which my attention has been directed for some years past is emphysema. The conclusions at which I have arrived, with reference to the morbid anatomy, pathology, and treatment of the disease, are the result, not only of a careful examination of the structural changes it presents after death, but also, of a close study of its symptoms and progress during life. Some of the facts detailed in the following pages have been already in part presented to the profession: first, in a paper communicated to the Royal Medical and Chirurgical Society of London; and, secondly, in one subsequently read before a section of the British Medical Association. The opportunities of further investigation and study of the disease, which I have had since these papers were written, have tended to confirm the opinions therein expressed.

Hope Street, Liverpool,
November, 1862.

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THE TREATMENT OF EMPHYSEMA, AND OF THE DISEASES CONNECTED WITH IT.

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FIG. 1.

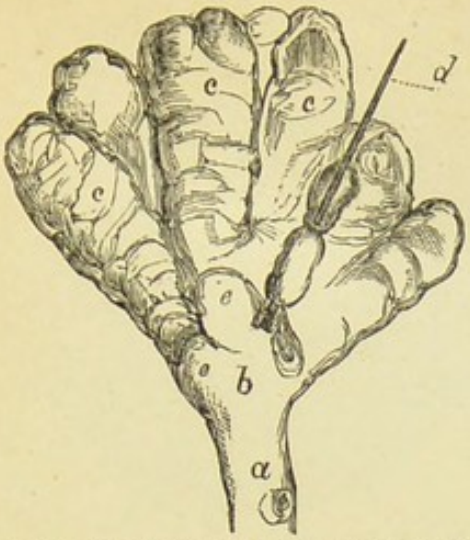


Fig. 1 is a drawing of a terminal bronchial tube, with a group of air-sacs, or lobulette, connected with it, divided transversely (human). *a*, the terminal bronchial tube; *b*, the dilated extremity of the terminal bronchial tube; *c c c*, individual air-sacs. At *d*, a bristle is seen passed into an air-sac; one end is seen opening into the common cavity in which the bronchial tube terminates. At *e e* are seen the openings of other sacs which lie beneath those which are exposed; six sacs are seen converging to the common centre. The markings in the air-sacs denote the boundaries of the alveoli.

FIG. 2.

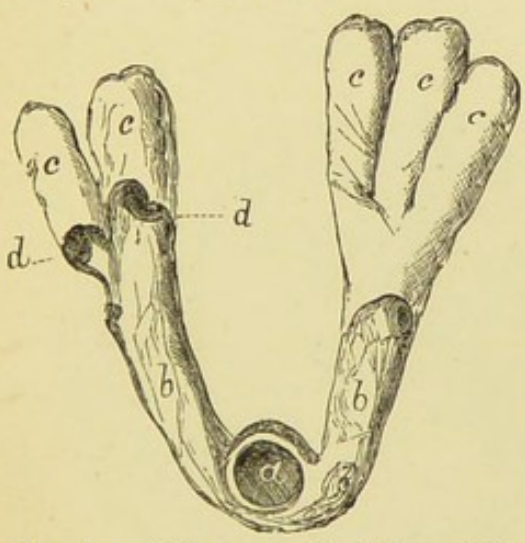


Fig. 2 is a drawing of two terminal bronchial tubes, with two groups of air-sacs, or lobulettes. The terminal tubes are seen to pass from a bronchial tube (*a*), and they terminate each in a group of air-sacs. *b*, terminal bronchial tube; *c c c*, air-sacs; *d d*, openings leading to air-sacs beneath those exposed. This diagram was taken from the lung of an infant under one year of age.

PLATE II.

FIG. 3

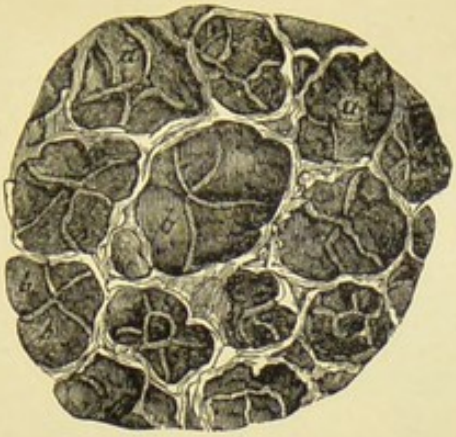


Fig. 3 represents a very thin slice of a cat's lung (injected, inflated, and dried), from the surface of the lung. The eye is looking upon the cut surface. The depressions, *a a*, are the bottoms of the air-sacs, resting on the pleura. The light coloured lines that surround them are their walls, and the small depressions seen within the walls, *b b*, marked off by less distinct lines, are the alveoli. The specimen from which this drawing was made was a very good one, and the drawing may be considered as fairly representing the appearance presented.

FIG. 4.

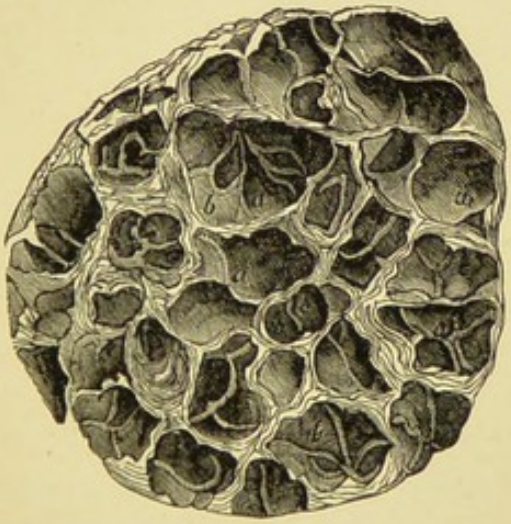
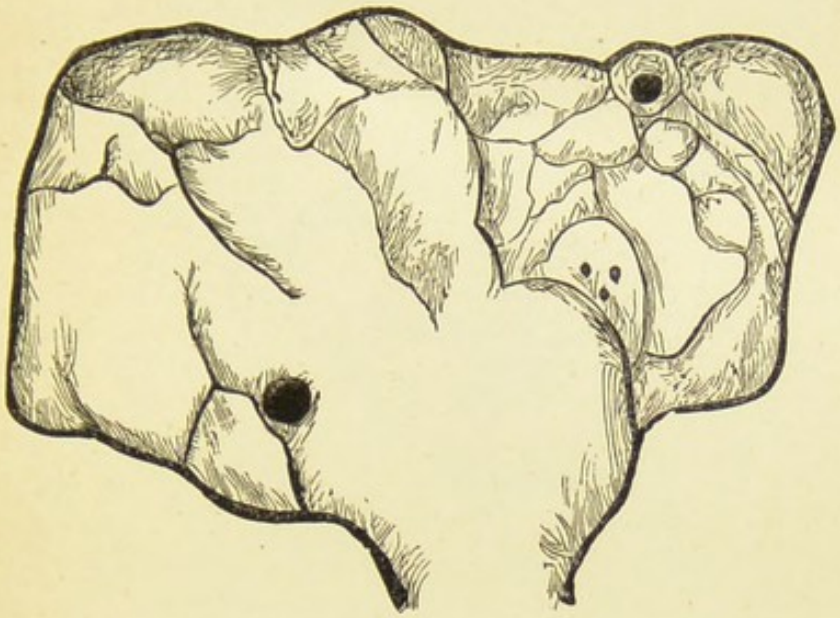


Fig. 4 is a drawing of a similar slice to that of Fig. 3, taken from a human lung. *a a a* are the air-sacs, *b b* the alveoli. The lighter portions represent the walls of the air-sacs and the alveoli.

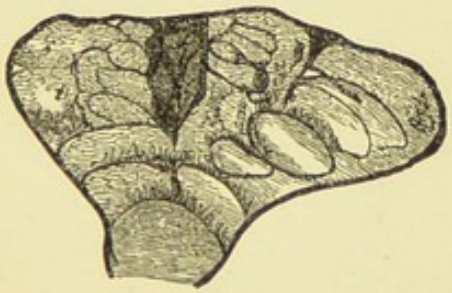
PLATE III.

FIG. 5.



Air-sacs of a piece of emphysematous lung, injected, inflated, and dried. The lung from which the piece was taken was emphysematous along its margins. The piece was taken from the margin It shews dilatation of the air-sacs, more or less obliteration of the alveoli, and slight perforation. The black spots represent the perforations. The air-sacs in the piece of lung examined varied in diameter from 1-20th to 1-30th of an inch — being more than double their ordinary size in health.

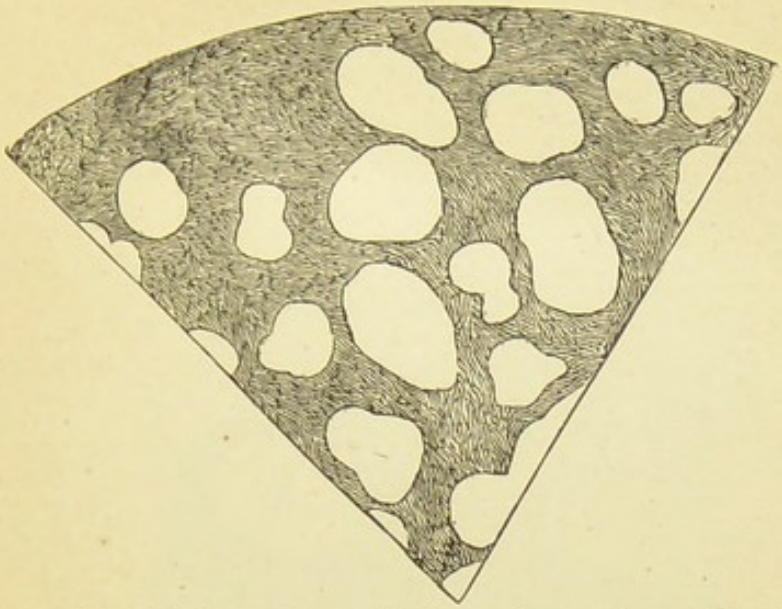
FIG. 6.



Air-sacs from the same lung as Fig. 5, but not emphysematous. The alveoli are shewn with their septa well marked. The diameter of the sacs in the part examined varied from 1-45th to 1-70th of an inch. Figs. 5 and 6 were both taken by means of the camera lucida, and the same magnifying power was used in both. They therefore shew the relative size of healthy and emphysematous air-sacs.

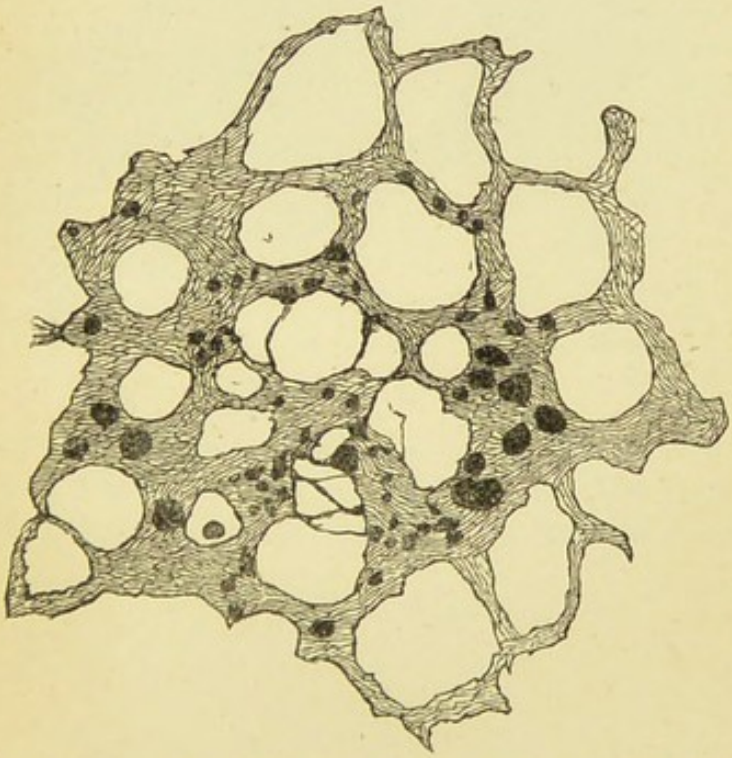
PLATE IV.

FIG. 7.



A slice of healthy lung-tissue. It shows the openings of the divided air-sacs, and the walls separating them, in a healthy condition. No perforations are seen. (Taken by means of the camera lucida.)

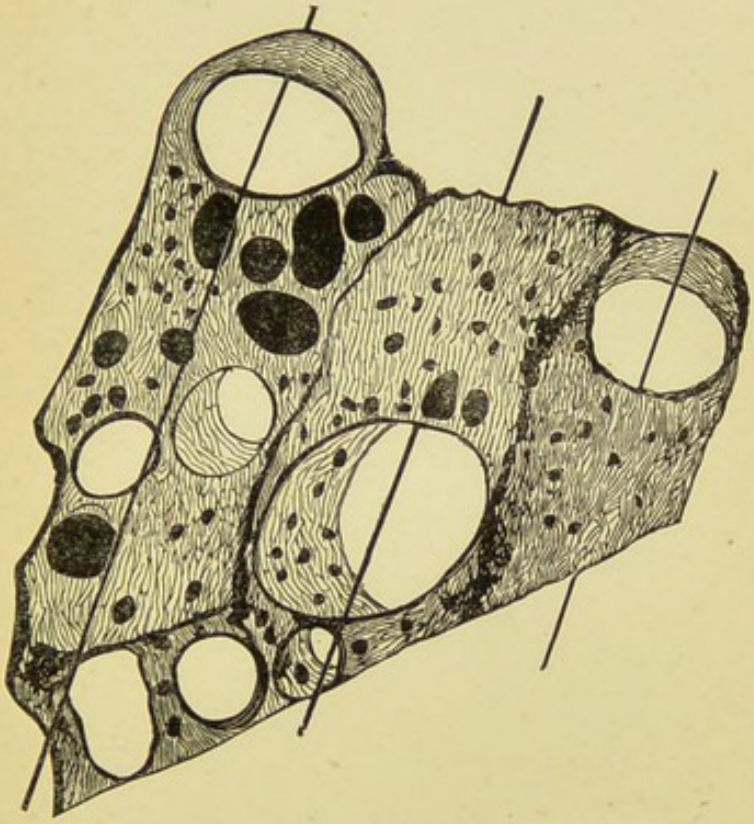
FIG. 8.



Slice of emphysematous lung-tissue in the earlier stage of the disease. The black spots show the perforations in the walls of the air-sacs. (Taken by means of the camera lucida.)

PLATE V.

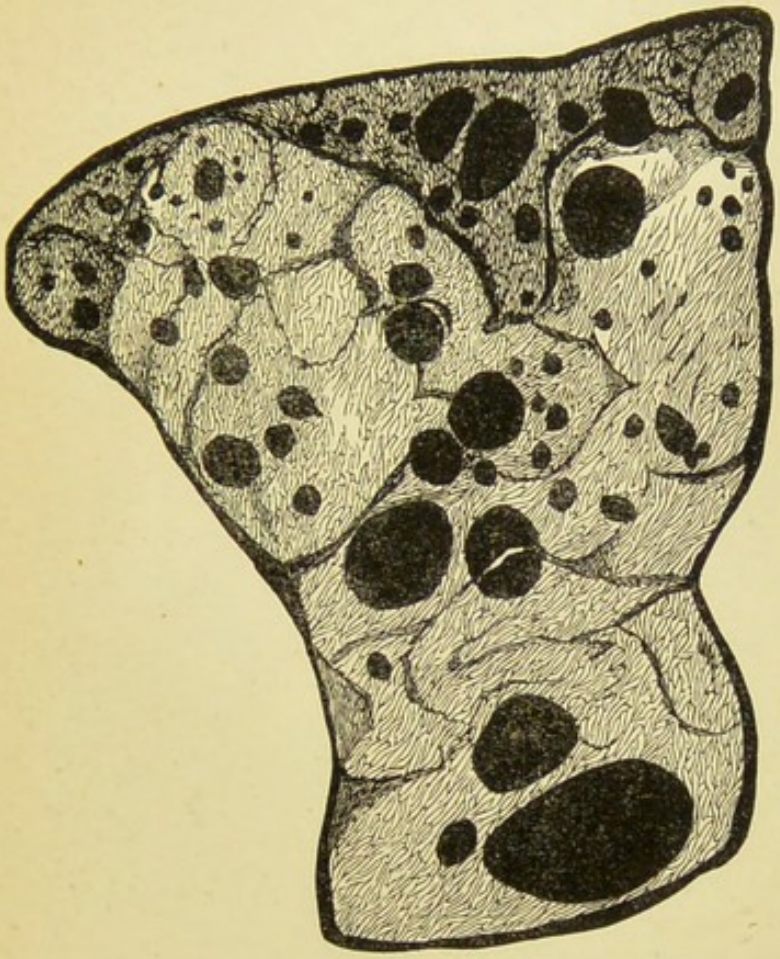
Fig. 9.



Air-sacs of emphysematous lung, as seen through dissecting microscope. The bristles are passed through the sacs. The upper walls of two, and the lower wall of one air-sac, are seen with their perforations.

PLATE VI.

FIG. 10.



Taken from a portion of lung very emphysematous. It shows the appearance of the air-sacs in a dilated condition, with their partitions much ruptured, and full of large perforations. (Drawn by means of the camera lucida.) The black spots shew the perforations. The cavities—the distended and broken-down air-sacs—in the piece of lung from which this was taken had a diameter of 1-16th, 1-20th, and 1-27th of an inch.

PLATE VII.

FIG. 11.

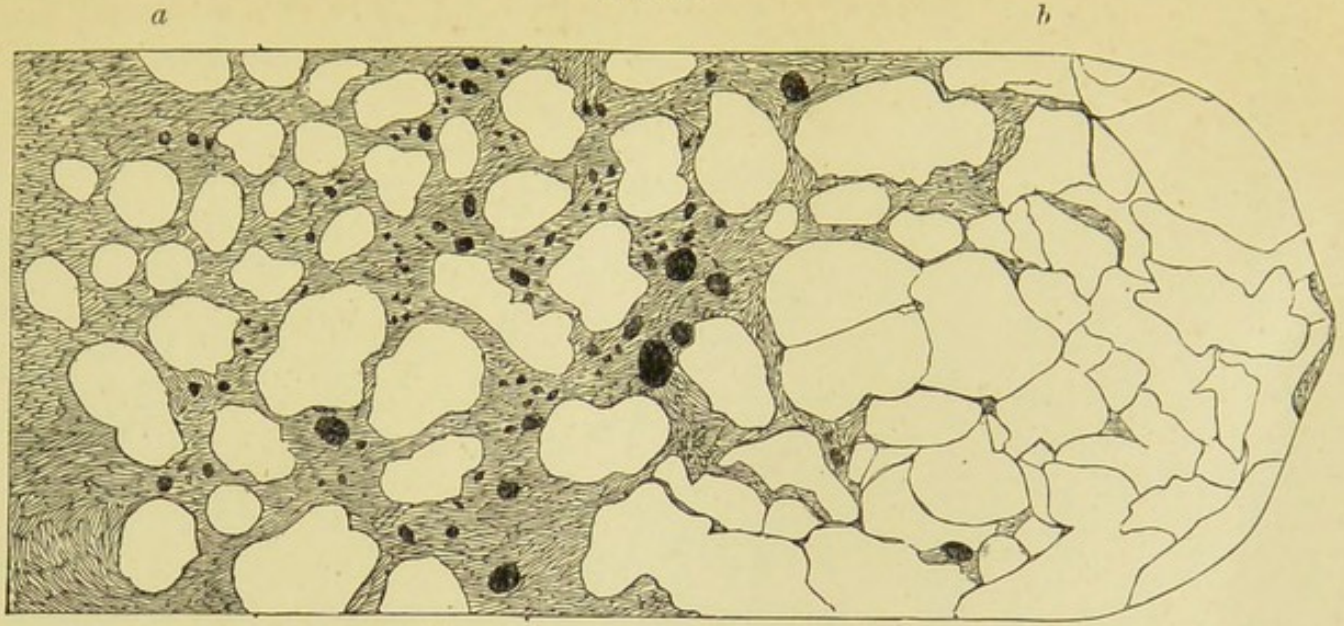


Fig. 11 shews the appearance of a piece of highly emphysematous lung, taken from the margin of the lung, where a distinct bulging or "vesicle" existed. The white spaces denote the openings of the divided air-sacs; the black spots, the perforations of their walls; and the lighter parts represent the walls themselves. At *a*, the lung-tissue is tolerably healthy; at *b*, the walls of the air-sacs are broken up, and the membranous shreds and septa alone remain. (Taken by means of the camera lucida, and magnified to one-half the linear dimensions of Figs. 5, 6, 7, 8, and 10.)

EMPHYSEMA OF THE LUNGS.

CHAPTER I.

THE MORBID ANATOMY OF EMPHYSEMA.

Opinions of earlier Pathologists. Views of Laennec. Frequency of the Disease. Importance of a knowledge of the Minute Structure of the Lungs. Ultimate Pulmonary Tissue; its Arrangement and Structure. The different kinds of Emphysema. Vesicular Emphysema; its various Forms. Modes of Preparation of Lung-Tissue for Examination. Changes which take place in the Air-Sacs in the different Stages of the Disease. Condition of the Blood Vessels; of the Bronchial Tubes. Interlobular Emphysema.

Previously to the time of Laennec, pulmonary emphysema was for the most part considered by pathologists as produced by an infiltration of air into the areolar tissue of the lungs.* An imperfect knowledge of the anatomy of these organs led to the supposition that a rupture of the air-cells would give rise to extravasation of air into the tissue by which they

* Descriptions of lungs which were large, and distended with air, and in some instances having dilated air-cells, will be found in the works of some of the earlier pathologists — Bonet, Morgagni, Floyer, Rusch, Valsalva, and Baillie; but no true definition of the disease was given before that of Laennec.

were connected with each other. An improved anatomical knowledge and further pathological inquiry have shown us that the quantity of areolar tissue in the lungs is very small; that extensive rupture of the air-cells may take place without infiltration of that tissue resulting; and that the only parts where such infiltration does occur are those in which the above-named tissue surrounds the various lobules.

Laennec was the first to draw the distinction between the two kinds of pulmonary emphysema; viz., vesicular and interlobular. The former he recognised as consisting in a dilatation or distension of the ultimate portions of the air-tubes. Subsequent researches have tended to prove the correctness of the French physician's observations in this respect; although the theory he advanced of the determining cause of the disease, as I shall have hereafter to show, has given way under an improved knowledge of the physiology of respiration, and more extended pathological inquiry.

Since the time when Laennec wrote, many able observers have directed their attention to this disease. Nevertheless, the information we possess with reference to it is insufficient to enable us to generalise with certainty on its essential nature; and there are numerous points in connection with its pathology and determining causes, on which investigations are necessary, in consequence of the diversity of opinion which exists thereon.

Of the great frequency of the affection in varying degrees of severity, no one who has been engaged in

extensive public or private practice can entertain any doubt;* and, considering the gravity of the symptoms the disease produces in its progress, and the tendency which exists for other organs to become secondarily involved, we cannot but admit the great importance of an early recognition of its symptoms, and a more accurate knowledge of its essential nature.

In speaking of the disease, Lebert has the following remarks in his *Pathological Anatomy* :—"Few diseases have given rise in the last twenty years to so many theories, so many explanations, more or less conjectural; and, as no one of them appears to us satisfactory, in the present state of science, it is the more essential to describe in a rigorously exact manner everything which relates to this affection, in order that for the future these materials may serve to establish doctrines more generally satisfactory on the subject."

Again, writing on the same subject, Rokitansky observes:—"The conditions giving rise to the production of emphysema, and its pathogeny in general, although much labour has been devoted to the affection, are still far from clear." Further, Hasse, in speaking of the affection, and whilst admitting the influence of catarrh and hooping-cough as occasional causes of the disease, remarks:—"The character and real import of emphysema must, however, be

* In fifty-three cases of cholera examined by Louis, twenty-three of the individuals were found to be more or less affected with emphysema of the lungs, in various stages; nor was there any ground for the assumption that it had become developed during the fatal malady.

admitted to depend on other causes, as yet not thoroughly made out."

These quotations will be sufficient to indicate the uncertainty which at present exists with reference to our knowledge of the nature and pathology of this affection.

To the study of morbid anatomy and pathology, a knowledge of minute structure is essential; hence the rapid strides which these sciences have made since the microscope has revealed to us the intimate arrangement of the various organs of the body. Important, however, as such knowledge is to the full appreciation of all diseased structure and function, its value becomes doubly manifest with reference to the disease now being considered; for there is probably no affection of the body, the symptoms and physical signs of which may be so easily explained, as may those of pulmonary emphysema, when once we are well acquainted with the structural changes and modifications by which the disease is accompanied.

Strongly impressed with the force of Lebert's remark, that it is "essential to describe in a rigorously exact manner everything which relates to the affection," and therefore to detail minutely the progressive changes which take place from the earliest periods of the disease, to the time of its full development, it becomes necessary for me to describe briefly the arrangement and structure of the ultimate pulmonary tissue, in order that the changes above

alluded to, of which it is the seat, may be the more readily understood.

The bronchial tubes of the lungs, after several divisions and sub-divisions, which, for the most part, are of a dichotomous nature, terminate in a dilatation, into which open a number of cavities, which constitute the ultimate expressions of the air-tubes. These cavities, to which various names have been given, I have elsewhere* called *air-sacs*, as being, in my opinion, more appropriate to their shape and arrangement than any term previously used; and the series of air-sacs connected with the extremity of each bronchial twig, with its system of blood-vessels, &c., I have called a *lobulette*.

Each *lobe* of a lung is composed of a number of *lobules*, the outlines of which can be seen on the surface of the lung. A *lobule* is made up of a series of *lobulettes*, and thus the description of a single *lobulette* will suffice for that of the entire *lobule*.

Each *lobulette* consists of a collection of air-sacs, which vary in number from six to twelve. The air-sacs are somewhat elongated cavities, communicating with the dilated extremity of a bronchial tube by a circular opening, which is usually smaller than the sac itself, and has sometimes the appearance of a circular hole in a diaphragm. When this is the case, the sac dilates suddenly beyond the opening. The sacs of the lobulette are placed side by side, and are separated from each other by thin membranous walls.

* "The Anatomy of the Human Lung," page 135.

Their shape, when properly inflated, or when distended by some material which has set in them, as gelatine, or a mixture of wax and turpentine, is polygonal. They approach the circular in form; but, in consequence of their mutual pressure, the parietes become somewhat flattened. The sacs increase slightly in size as they pass from the bronchial tube to their fundus, the latter being usually the broadest part of the sacs; but they often have an almost uniform diameter throughout. All the sacs pass from the extremity of the bronchial tube *towards* the circumference of the lobule of which the lobulette forms a part; they consequently radiate from the tip of the bronchial twig. The sacs connected with one lobulette do not communicate with those of another lobulette. As the sacs pass towards the boundary of the lobulette, they often bifurcate; and here and there circular orifices exist, leading to smaller air-sacs. If we trace the air-sacs from their fundus, we may say that, passing from the periphery of the lobulette, and diminishing somewhat in size, they all terminate in the dilated extremity of the bronchial tube; as they thus proceed, they often join, two or three together, and these terminate in a single mouth. The tube which results from the union of two or more sacs is smaller in capacity than the sacs taken together, but greater than either of them individually. The dilated extremity of the bronchial tube, above alluded to, constitutes the *point de réunion* of all the air-sacs, and may be considered as the common centre of the lobulette. The air-sacs of the

adult human lung vary in diameter from 1-45th to 1-85th of an inch. (For illustrations of this paragraph, see Plate I., figs. 1 and 2.)

The walls of which the air-sacs are composed are exceedingly thin, and much sacculated; *i. e.*, they have in them a number of small, shallow, cup-like depressions, separated from each other by portions of membrane, which are more or less raised, and project into the interior of the sacs. The bottom of an air-sac presents the same appearance as its lateral walls; and the cup-like depressions, or *alveoli*, are there very numerous. The number of these alveoli varies very much; I have counted as many as ten at the fundus of an air-sac in a cat's lung; in the human being I have counted five and six, but the number is not usually quite so great. I have found the entire number in each air-sac varying from eight to twenty. (See Plate II., figs. 3 and 4.)

The air-sacs rest, externally, by their fundus, on the pleura; but within the substance of the lung they in part rest on, and are supported by, the bronchial tubes and blood-vessels.

The air-sacs are separated from each other by thin walls; the membrane composing which, in a lung inflated and dried, is very transparent. The projections of this membrane in the shape of thin processes, having a sharp margin, constitute the septa between the alveoli; and wherever an opening exists, leading into a smaller sac, this membrane projects in a similar way, and forms a circular orifice, which is much smaller than the cavity to which it leads: the

sac, in fact, dilates abruptly on the distal side of the opening. It is in the membrane composing these walls, and in the septa of the alveoli, that the capillaries of the pulmonary artery are spread out.

Each lobulette is separated from those by which it is surrounded by walls, which appear to resemble in every way the walls of the air-sacs; and in an adult inflated and dried lung, careful observation is necessary to make out the partitions. That perfect septa do exist, is proved by laying open, in a recent lung, a bronchial tube, to its ultimate division; when, by placing a fine blowpipe in it, and blowing down it, a single lobulette is alone inflated.

As I have before mentioned, a number of the lobulettes constitutes a lobule. These lobules are of various sizes; they are each surrounded by a membranous wall, which is dense and strong, allowing of no communication between the blood-vessels passing to adjoining lobules. The lobules are connected together by a small quantity of areolar tissue, in which the branches of the pulmonary veins take their course.

The walls of the air-sacs and alveoli are formed of yellow elastic fibrous tissue, a basement membrane, and an epithelium, together with the capillary vessels constituting the pulmonary plexus. The fibres of the elastic tissue are arranged in bundles, and singly. They are found surrounding the mouths of the air-sacs, running through their walls in various directions, and encircling the alveoli. The fibres traversing

the walls are placed at some distance from each other, so that spaces are left between them where the blood-vessels are uncovered, except by the basement membrane and epithelium. At the circumference of the alveoli, and at the margins of the sacs, the fibres are often gathered into bundles of considerable size.

It is to this tissue that the elasticity of the air-sacs, and of the whole lung, is due; and the changes which take place in it, in the disease under consideration, constitute one of the most important features of the affection.

The basement membrane is thin, homogeneous, and transparent. It is a prolongation of that lining the bronchial tubes; but it becomes finer and more delicate as it passes from the latter vessels into the air-sacs. It is covered by an epithelium, consisting of small flattened nucleated cells.

The capillaries of the pulmonary arteries, which form the pulmonary plexus, ramify in the membrane described above as forming the walls of the air-sacs and the septa of the alveoli. The plexus consists of a single layer of vessels placed between two layers of basement membrane. As the respiratory changes take place in the air-sacs, it is obviously of the greatest importance that the integrity of these latter should remain unimpaired, for any loss of structure they may undergo diminishes the amount of aerating surface over which the blood has to pass. The bearings of these points on the disease under consideration will be dwelt on hereafter.

Having premised these observations on the arrange-

ment and structure of the healthy lung-tissue, I pass on to examine the changes it undergoes in the progress of emphysema.

PULMONARY EMPHYSEMA

is of two kinds :—

I. Vesicular Emphysema.

II. Interlobular Emphysema.

The first is by far the most frequent and most important affection. I have rarely seen the second, except in advanced cases of the first kind.

I. *Pulmonary Vesicular Emphysema* exists in three forms, which, although they may differ as to their pathological causes, do not differ anatomically except as to the extent to which they involve the lung. We have, firstly, that which is the most partial, and confined to a few air-sacs, or to a single lobulette; secondly, that in which the whole of a lobule is affected; and thirdly, that in which the whole of one lobe of a lung, or, more frequently, the whole of the lung itself, is involved in the disease.

1. The first form of the disease, or *partial lobular emphysema*, is not often seen as an independent affection; but in lungs in which the second form exists, we occasionally meet with small patches of dilated air-sacs, especially along the margins of the lobes—patches which clearly only involve a few air-sacs, or, at most, a single lobulette. These portions resemble enlarged vesicles; and, as pointed out by M. Lombard, of Geneva, many years ago, have very much

the appearance of the vesicles of pemphigus. They push outwards the pleura, so as to raise it above the level of the surrounding lung-tissue. They are not simple elevations of the pleura, as may be distinctly seen in injected preparations, when the blood-vessels belonging to the air-sacs may be seen ramifying beneath the pleura. Sometimes these partial emphysematous patches are seen extending for some distance along the margin of the base of the lung, and they have then an appearance very like that of a row of beads.

2. The second form, or *lobular emphysema*, is that most frequently met with; in fact, in the majority of *post mortem* examinations I have seen, since my attention has been directed to this subject, I have found this form of emphysema more or less prevailing. It involves one or more lobules in different parts of the lung, and is more especially found along the margin of the base, at the anterior border, and apex of the organ. This is the kind of emphysema so frequently met with in cases of phthisis; I have also seen it in cases of pneumonia where the lung has become consolidated; and in one instance of this latter kind the emphysema had all the appearances of having been recently produced.

In this form of emphysema it is easy to trace the different divisions of the lung; the boundary wall of the lobules has not given way, and there is no interlobular emphysema, except in some cases, and to a partial extent. Again, it is by no means essential that all the air-sacs in an affected lobule shall be

equally dilated; the most superficial ones, those especially beneath the pleura, suffer most. At the same time, it must be remarked that this unequal dilatation is sometimes more apparent than real, resulting from the mode of preparation to which the tissue has been subjected, viz., inflation and desiccation. During the process of drying, the air-sacs farthest from the surface collapse most; and thus they have the appearance of being smaller than those which are more superficial, without any real difference having originally existed between the two sets.*

The emphysematous lobules are seen on the surface of the lung, protruding beyond the level of the surrounding tissue; and along the margins of the lobes they often form projections of considerable size, in some instances becoming developed into the so-called "appendages."

3. The third form of pulmonary vesicular emphy-

* Some anatomists have taught that the "vesicles" of the lungs are larger towards the surface than in the interior of the lungs. This statement seems to me altogether devoid of proof. It is quite true that, in a lung which has been inflated and dried, the "vesicles" which are situated at the surface, beneath the pleura, appear larger than those which are situated at a distance from it, and from the observation of this fact in all probability the statement has been made; but it must be borne in mind that this is the result of the collapse of the lung which takes place during desiccation. When a lung is inflated, and slowly dried, the surface is that which dries most quickly; and the *air-sacs* and *alveoli* there situated, being subjected to but little pressure, do not collapse to any very great extent, and thus they maintain a size approaching that they originally had; but with the interior of the lung the case is altogether different; the shrinking which necessarily takes place gives rise to considerable pressure on the air-sacs, and collapse of them results, producing an appearance of small "vesicles," from the

sema, or *lobar emphysema*, is by far the most important. As far as my observations go, it more frequently attacks both lungs than one, and as well the lower as the upper lobes. It constitutes a most formidable affection, and often destroys life at an early period. The cases in which I have had an opportunity of seeing the disease after death, have been those of persons of adult age. Its features are very characteristic, and it is easily recognised during life. It consists of an emphysema of the whole pulmonary tissue of a lobe, or lung. The lung-substance has a peculiar doughy feel, pits on pressure, is wanting in healthy crepitation, and has a colour very closely resembling that of a calf's lung. The whole organ is increased in bulk. No collapse of the lung-tissue takes place when the walls of the chest are removed, in making a *post mortem* examination; on the contrary, the lung often bulges forwards, as if its cavity were too small for it.

process which is adopted, and not from any structural arrangement. From the examination of the human lung under various modes of preparation, and of the lungs of other animals, I have been led to the conclusion that there is no real difference of size between the superficial and deep air-sacs and alveoli of the lungs, nor yet between these structures in different parts of the organ; nor is it easy to see on what principle such a condition would be probable, or on what theory it could be expected.

These observations, with reference to the healthy lung, are equally applicable to the emphysematous organ; and the facts alluded to should be borne in mind, when we wish to make a comparison between the effects produced by the disease on the superficial and on the deep-seated air-sacs. It is, I think, highly probable that the circumstances I have stated have been overlooked by authors who have written on emphysema, and who have dwelt on the greater liability to distension of the superficial, as compared with the more deep-seated, lung-tissue.

It is also seen to overlap the heart, unless adhesions have occurred which prevent this taking place. The surface of the lung is not unfrequently marked by the impressions of the ribs. If a lung presenting this form of the disease be carefully removed from the chest, and inflated through its bronchus, it will be found that it may be distended to a very large size, without apparently any rupture taking place from the inflation. When the inflation is stopped, the air is expelled from the lung very slowly, and the collapse of the tissue is often very incomplete. The lung presents, when inflated, for the most part, a perfectly smooth and level surface, except in those cases where collapsed portions exist; or where portions of lung extremely emphysematous form projections or appendages. Often the outlines of the lobules cannot be distinctly seen, in consequence of the rupture of their boundary walls having given rise to the production of interlobular emphysema. When this is the case, the air may be pressed laterally from one lobule to another.

In examining the emphysematous lung-tissue, to ascertain the anatomical changes of which it is the seat, I have adopted similar methods of preparation to those which I have made use of for the examination of the healthy lung; viz., first, inflation through a bronchial tube, and subsequent desiccation: and secondly, injection of the blood-vessels with a coloured solution of gelatine, followed by inflation and desiccation. If we examine pieces of lung which have been pre-

pared in either of these ways, under different degrees of development of emphysema, we are enabled to ascertain the exact condition of the pulmonary tissue in the progressive stages of the affection, and the extent to which it has departed from the normal type.

In the early stages of the disease we recognise a simple dilatation of the air-sacs, an increase in the size of the alveoli, and a diminution in the height of the alveolar walls, which, yielding with the distending cavities, become partially obliterated. As the disease progresses, the air-sacs become still more distended, and the alveolar walls in some instances completely obliterated, so as to give a regular and smooth appearance to the inner surface of the air-sacs, instead of the honey-combed appearance characteristic of their normal state. (See Plate III., figs. 5 and 6.) This distension of the air-sacs is necessarily attended with a divergence of the elastic fibres which enter into their composition, and with a general thinning of their walls—a condition which prepares the way for the next stage in the progress of the disease, viz., a perforation of the walls themselves. This, at first, is but slight; here and there a circular or oval opening may be seen in the membrane; as the disease progresses, these openings become more numerous, and larger; in some instances, the whole of the walls of the air-sacs and the septa of the alveoli being perfectly riddled with small openings, so that a horizontal section of the lung-substance has a general cribriform appearance. These openings are for the

most part either circular or slightly oval. They exist in all parts of the walls, and are often seen in the septa between the alveoli, before the air-sacs are sufficiently distended to obliterate the septa. (See Plate IV., figs. 7 and 8, and Plate V., fig. 9.)

The subsequent steps in the progress of the disease consist in a further distension of the air-sacs, an enlargement of their perforations, and a rupture of the fibres of which their walls are composed. As these results take place, the walls become more and more imperfect, and the openings in them coalesce. A further breaking down of the walls then occurs, so as to leave but very partial partitions between the cavities; and in the most advanced stages of the disease these partitions undergo other changes, and are reduced to mere membranous shreds, or thin fibrous cords, passing in various directions, traversing, in fact, the distended sacs, two or more of which, by the destruction of their walls, have united to form a single cavity. These cavities occasionally assume a large size, and project from the margin of the lung; they sometimes also form appendages, being connected with the body of the lung merely by stalk-like processes. (See Plate VI., fig. 10, and Plate VII., fig. 11.)

These appendages differ much in form and volume. In the latter they vary from the size of a nut to that of a pigeon's egg; and occasionally they are even larger. As has been observed by Louis, their elongated and rounded form sometimes makes them resemble the swimming bladder of a fish. They

will sometimes empty themselves from a single puncture; at others, two or more punctures are required before they will completely collapse. They present in their interior, cavities varying in size, and traversed at all angles by thin membranous shreds. At the distal extremity of the appendages these cavities are larger than elsewhere, and they diminish towards the end where the appendages join the lung.

If we examine the inner surface of the appendages I have just described, or of a portion of emphysematous lung-tissue which has reached an ordinary degree of development, we find abundant evidence that the cavities are formed by the dilated air-sacs. We recognise at once, under the microscope, the same appearance of the lining membrane—viz., its epithelial covering—as we find in the air-sacs in a condition of health.

I have observed an anatomical difference in preparations of different lungs, which I think is important in its pathological bearing. In some lungs, in which the emphysema has been of the *lobular* kind, I have found the air-sacs distended to a very considerable extent, but I have observed little or no perforation; whilst in other lungs, and especially in those where the disease has been of the *lobar* character, I have found extensive perforation, with certainly not more, and in some instances less, dilatation than in those alluded to above. In all the cases of lobar emphysema which have come under my notice, I have found the walls of the air-sacs extensively perforated; whilst, in some cases of lobular emphy-

sema, this condition has not existed. These facts would seem to indicate that, in the cases where rupture takes place under a dilatation which in others does not produce such rupture, there must be some degeneration of the lung-tissue which renders it more liable to give way.

If we examine the condition of the blood-vessels in an emphysematous lung, we have evidence of the cause of its anæmic appearance. We find in the earlier stages of the disease, when there is simply a dilatation of the lung-tissue, that the capillaries of the pulmonary plexus are wider apart than in a state of health; the meshes formed by them are larger. As the walls become perforated, and the sacs further distended and broken, the capillaries become ruptured; and hence we have one cause of the hæmoptysis which occasionally attends the progress of the disease, although this symptom does not necessarily follow from the pathological condition just alluded to.

If we examine a piece of lung in a condition of extreme emphysema, we observe a number of small blood-vessels taking their course in the membranous septa and shreds, which I have previously described as traversing the cavities. The vascularity of these parts is exceedingly slight; and but little or no respiratory function can be performed by them.

In examining the condition of the bronchial tubes in emphysematous lungs, I have found them occasionally dilated, and more especially in old standing cases. Where recent acute or long-continued chronic bronchitis has existed, the mucous membrane of the

large tubes has been red, injected, and somewhat thickened; but I have usually observed that the smaller tubes were pale, and ex-sanguine, although in some instances I have found them filled with muco-purulent matter. I have not seen ulceration present in any case.

If the smaller bronchial tubes of two portions of the same lung be examined, the one portion being healthy, and the other emphysematous, the tubes of the former will be found of a darker colour than those of the latter. This results in part from the diminished vascularity of the pulmonary tissue in the emphysematous portion, as compared with that of the other, and in part from a similar condition of the bronchial mucous membrane.

An alteration of tissue, I have frequently observed, in old cases of lobar emphysema, is increased development of the circular muscular fibres of the bronchial tubes. In these cases, the fibres become much more apparent in the smaller tubes than they are in the healthy lung.

Interlobular Emphysema. This consists of an infiltration of air into the areolar tissue which exists between the various lobules. I have never seen it as an independent affection; but, in almost every case where there has been extensive vesicular emphysema, I have found the interlobular kind existing to a greater or less extent. It is often very partial, and seems to have little tendency to spread. At other times (especially in cases of lobar vesicular emphy-

sema) it is quite easy to pass the air from one lobule to another, throughout a great part of the lung, so that it must traverse extensively the interlobular areolar tissue. From the communication of this tissue with that beneath the pleura, it is easy to see that the latter may become stripped from the lung by air which has first found its way between the lobules; and from its connection with the tissue surrounding the bronchial tubes and blood-vessels, and thus with that in the mediastinum, we can explain the occurrence of those cases in which emphysema of the cellular tissue of the neck has been produced by violent and long-continued expiratory efforts.

Lebert says that interlobular emphysema is an early result of the vesicular form. With this opinion I cannot concur. I have rarely seen the former affection, except in cases where vesicular emphysema was extensively developed. One remarkable instance, forming an exception to this statement, in which the air made its way into the interlobular tissue, and thence into the mediastinum and cellular tissue of the trunk and neck, I shall refer to hereafter.

The pleura is occasionally raised from the surface of the lung by infiltration of air beneath it. I have, however, only met with one instance where this has occurred to any considerable extent. In the case I allude to, a cavity existed as large as a small orange. The substance of the lung from which the pleura was thus stripped was highly emphysematous; and an opening existed at one spot, through which

the air had escaped, and found its way beneath the pleura.

Instances of extensive sub-pleural emphysema are, I believe, rare in adults. M. Guillot, in a paper published in the *Archives Générales de Médecine* for 1853, has detailed a number of such cases in children, who had been for the most part the subjects of long-continued hooping-cough. In these cases the pleura was found, after death, raised from the surface in numerous parts of the lung; and in some instances there was emphysema of the cellular tissue of the mediastinum, and even of the neck. M. Guillot sems to me to have clearly shown that the pathological condition just described was a result of the disease from which the patients suffered, and was produced by the severe fits of coughing under which they laboured.

CHAPTER II.

THE PATHOLOGY OF EMPHYSEMA.

Importance of a Correct View of the Pathology of the Disease, in reference to Treatment. The Question of Degeneration of Tissue Considered. All Cases of Emphysema not to be included under the same Head. Distinction between Partial and General Emphysema. Mr. Rainey's View of Fatty Degeneration. Views of Dr. Williams and Dr. Jenner. Investigations of the Author. Constitutional Nature of the Disease; its Hereditary Character; etc.

I HAVE in the previous chapter traced out the various anatomical changes which take place in the lung-tissue in emphysema. These changes may be briefly described as distension, perforation, atrophy, and breaking up of the walls of the air-sacs. In different parts of one and the same lung, which may present the disease in different stages, the various conditions enumerated may frequently be seen; for instance, towards the margins or borders of the lung, and at its apex—parts which have a greater tendency than others to become affected—the disease may be seen in its most developed form, and "appendages" may exist; whilst away from these spots, and in the deeper parts of the lung, the stages of perforation or simple distension may not have been passed.

The next point which presents itself in connection

with our subject, is the consideration of the pathology of the disease, or the nature of the morbid action which results in the structural changes I have previously described. An examination of this question lies at the very root of the therapeutics of the affection, for it is obviously impossible that we can scientifically direct our measures towards checking its progress, so long as our knowledge of its pathology is imperfect. Undoubtedly the subject is still involved in much obscurity; and no good can possibly result from any attempt to dogmatise on it.

For the elucidation of the nature of many pathological processes, a knowledge of morbid anatomy alone is often insufficient; and although this latter may afford indications of the most important character, confirmatory evidence may frequently be derived from other sources, and perhaps from no one to so great an extent as from an examination of the manner in which a disease is influenced by certain modes of treatment. To this point, and to the important indications which may be derived with reference to the pathology of emphysema, from the effect produced on it by certain remedial agents, I shall have to recur in speaking of the treatment of the disease.

The great question for consideration in connection with the pathology of emphysema is, whether there is any degeneration of tissue either preceding or attending the affection; whether, in fact, there is any local or general condition which so interferes with the normal nutrition of the walls of the air-sacs,

as to cause their perforation, rupture, and even total destruction. If it could be shown that, either in the elastic fibres, which are so numerous in the walls of the air-sacs, in the basement membrane, or in the capillary blood-vessels, certain changes take place of a degenerative character, a very important step would have been made in reference to our knowledge of this disease.

The first appreciable anatomical change which occurs in the lung-tissue is an increase in the size of the air-sacs. This must necessarily be attended with a loss of elasticity on the part of the elastic fibres, which, on being stretched by mechanical power, are unable to recover themselves. When we find emphysema existing only as a partial affection, and in conjunction with some old-standing disease of the lung, we can readily imagine that it may have been produced by mechanical violence, whilst the tissue affected was in a healthy state; but when we see the disease, even in moderately young persons, creeping insidiously on, and attacking the whole of one or both lungs, without the previous existence of long-continued or violent cough, we can scarcely imagine that changes of so extensive a character can result from mechanical violence, without the previous existence of some morbid condition of the lung-tissue.

My own observations have led me to conclude that all cases of emphysema cannot be included, either with reference to their pathology or determining cause, under the same head. Where the disease

is partial, and situated, as is then usually the case, along the margins and at the apex of the lung, and is associated with or has followed some other pulmonary affection,—as, for instance, chronic bronchitis, or, in fact, any disease which has been attended with long-standing or violent cough,—I believe the morbid changes may have been brought about mainly by mechanical violence, without there having been any pre-existing affection of the lung-tissue. The general appearance of the lung-substance, in these cases, is very different from that which characterises the disease to which the name of lobar emphysema has been given; the tissue has more the appearance of that of the healthy lung, but it is paler and more anæmic than the latter. It is quite true that in these cases the same anatomical changes — dilatation, perforation, etc.—take place in the progress of the disease, as in the larger and more formidable affection; but these changes necessarily ensue, not only from a rupture of the elastic fibres and basement membrane, but also from the giving way and absorption of the capillary blood-vessels—circumstances which lead to imperfect nutrition and consequent atrophy.

Lobar emphysema is a disease which has been but imperfectly treated of by systematic writers, and has not received that attention which its importance deserves. It is true that some of our great pathologists have recognised it as a substantive affection; but practically it has not been sufficiently distinguished from the more partial and less formidable kind of the disease. As I have before remarked, it

is occasionally seen in early life; but the most numerous and most marked instances of it that have fallen under my notice have occurred in adults. When fully established, it presents symptoms easily recognised, and which will form the subject of future consideration.

That this form of the disease differs very materially in its pathology from the partial emphysema which I have spoken of in a former paragraph, will, I think, be admitted by all who will give a careful attention to the subject. The insidious manner in which the disease sometimes comes on; the almost entire absence of cough frequently observed, as well as of all other symptoms except a gradually increasing dyspnœa, and, as the patients constantly describe it, a "smothering in the chest;" the occasional rapidity with which the affection progresses; the secondary consequences which ensue; and the general cachexia which often supervenes;—all point to the grave character of the malady, and I believe to its constitutional origin in some degeneration of the pulmonary tissue.

But if emphysema be the result of some degeneration of the lung-tissue, it behoves us to inquire what the nature of that degeneration is. On this point a good deal of obscurity still exists. It does not appear that much attention has been given to this part of the subject; and its important bearings on the treatment of the disease have been but little dwelt on.

In a paper which was presented to the Royal Medical and Chirurgical Society of London, and

subsequently published in their "Transactions," Mr. Rainey has described the condition of an emphysematous lung, which he seems to have examined with great care. This lung was taken from a subject forty years of age, and the emphysema seems to have been only partial; for the general aspect of the lung, especially in the vicinity of the emphysematous parts, is described as being healthy. A few tubercles, however, existed in some spots. Mr. Rainey found the pulmonary membrane in the emphysematous portions more or less studded with fatty matter; and he has expressed an opinion that this deposit of fat is the precursor of the perforations and subsequent changes which take place in the disease.* As it does not appear that Mr. Rainey has observed this condition of the lung-substance as a general accompaniment of emphysema, but only in the specimen from which he has drawn the chief conclusions referred to in his paper, and as his observations have not been confirmed by subsequent inquiries, the pathological view he has sought to establish—viz., that the disease is the result of fatty degeneration—cannot be considered as settled.

In his recent lectures, delivered before the Royal College of Physicians of London, Dr. C. J. B. Williams, in speaking of emphysema of the lungs, says: "It is fatty degeneration of the lung-tissue which aids in bringing about the atrophy and rupture of the cells."†

* Medico-Chirurgical Transactions, vol. xxxi.

† Lumleian Lectures: *The Lancet*, 1862.

With the exception of the two authors quoted above, no pathologists, as far as I am aware, have supported the opinion that emphysema is produced by, or attended with, fatty degeneration. On the contrary, one of our most accurate observers in connection with this disease, Dr. Jenner, to whom we are indebted for an able paper on it, has expressed his opinion that "the most frequent anatomical change in the lung," producing loss of its elasticity, "is fibrous degeneration—the consequence of the exudation of that variety of lymph which escapes from the capillaries, when they are the seat of slight but long-continued congestion."*

With the view of ascertaining whether emphysema is preceded by, or has associated with it, fatty degeneration of the pulmonary tissue, I have made a careful examination of a large number of specimens of lungs which were the seat of the disease. These specimens were taken from lungs which presented the affection in all its varieties, whether partial or general. I have not only submitted to examination the diseased portions, but (where the disease was partial) pieces taken from contiguous parts, and where the lung-tissue was apparently healthy. The general results of my investigations may be briefly stated as follows: In the large majority of cases I have found no indications whatever of fatty matter; in some few instances, however, I have seen deposits of fat in the walls of the air-sacs.

My examinations have been conducted with the

* Medico-Chirurgical Transactions, vol. xl.

microscope on recent, and dried specimens; and also by heating the lung-tissue between pieces of glass, so as to dissolve out the fat, if present, and thus get indications of its existence.

In examining this question, I have viewed it in several lights; and, considering that the disease might possibly be due to some affection originally commencing in the capillary blood-vessels, and producing mal-nutrition in the pulmonary tissue, I have made a careful examination of the branches of the pulmonary artery, from their commencement to their termination in the pulmonary plexus, in order to ascertain if they were the seat of anything like fatty or atheromatous degeneration. The results are that I have found in some cases atheroma existing in the branches of the pulmonary arteries and in their capillaries, whilst in others I have found no indications of it whatever. In the cases where the atheroma existed in the pulmonary arteries, I have always found it in the aorta as well; so that the affection of the former vessels must be considered as simply the result of the general tendency to arterial degeneration, and not as possessing any specific bearings on the emphysematous condition of the lung-tissue.

I have further endeavoured to ascertain, by careful microscopical examination, whether any appreciable difference could be traced between the elastic fibres of the emphysematous lung and those of the healthy organ. Here, again, I am not able to say that any marked distinction existed between the diseased and healthy fibres, except that the former sometimes

appeared less regular in their outline, and had less tendency to curl up at their ends; but as to any structural change in the fibres, I have not been able to satisfy myself that any such had taken place.

Considering, therefore, the facts and statements I have adduced in connection with this question, I cannot agree with the view that has been expressed as to the dependence of emphysema on fatty degeneration; for, when we find that this condition is only an occasional, and not a constant, accompaniment of the disease, we cannot look upon it as its essential and predisposing cause. Nor can I, on the other hand, agree with Dr. Jenner in his view of the nature of the degeneration — at least as regards the lobar form of emphysema. It is quite possible that, in cases of chronic bronchitis, there may be congestion of the pulmonary plexus, which may give rise to a weakening of the walls of the air-sacs, and that under this condition the sacs may become distended and ruptured by the mechanical act of coughing; but this will not apply to those cases where the emphysema is of the primary kind, coming on without any pre-existent affection of the bronchial tubes. In such cases the degenerative process is the first step in the disease; and any congestion which may occur is but a secondary consequence.

But although microscopical examination does not enable us to detect any structural alteration in the ultimate tissues of the air-sacs, and the application of other means furnishes us with no proof of the presence in them of any morbid condition;

yet that degeneration does not exist is by no means determined by the failure of our present methods of investigation to demonstrate it. It is quite possible that the elasticity of the yellow fibres may become impaired, or even destroyed, without any such structural alteration resulting as could be appreciated even with the highest powers of the microscope; and it is also equally possible that changes may occur in the blood-vessels giving rise to malnutrition of the pulmonary tissue, and yet that we shall be unable to distinguish them. We see in this disease, as in many others, the secondary changes, as I have described them in the previous chapter; but the primary and essential ones we cannot recognise.

Notwithstanding, however, that my investigations do not enable me to say what is the exact nature of the degeneration which leads to the production of emphysema, nor yet whether it commences as an affection of the capillary blood-vessels, or of the elastic fibres and basement membrane, I do not entertain the slightest doubt that *the disease in its severer forms is of a constitutional nature; that one of its most important features, and perhaps the primary step in it, is a mal-nutrition of the pulmonary tissue, causing its degeneration, and giving rise to all the structural changes I have previously described.* That fatty deposit occasionally exists, I have already stated; but the question arises whether this is a primary cause of the anatomical changes which take place, or whether it may not be the result of the imperfect nutrition which necessarily ensues in the progress of the disease.

The view I have taken of the constitutional nature of emphysema receives support from the facts which have been brought forward of its hereditary character. On this point I quote the observations of Jackson, which furnish us with very important results. He found that, of twenty-eight persons affected with emphysema of the lungs, eighteen were the offspring of parents (father or mother) affected with the same disease, and that several of these had died in its course. In some instances the brothers and sisters of these persons were also emphysematous. On the other hand, of fifty persons not affected with emphysema of the lungs, three only were the offspring of emphysematous parents.

Facts of this kind tend to throw great doubt on the opinion of emphysema being solely produced by mechanical dilatation of the healthy air-sacs, and to favour the view that some deep-seated pathological cause for it frequently exists in connection with the lung-tissue.*

Some pathologists have supposed that there is hypertrophy of the pulmonary tissue in emphysema, and M. Louis has endeavoured to account for this condition, on the principle that all membranous structures become thickened in proportion as they are dilated; and he quotes as instances the effects

* Dr. Walshe, in his last edition of "Diseases of the Lungs," adopts a view of a similar character to that which I have advanced as a possible cause of emphysema. He says: "The dilatation may thus be the resultant of *primary* nutritive change in the actual walls of the enlarged vesicles, affecting both their statical and dynamic properties."

produced on the œsophagus by cancer of the cardiac orifice of the stomach—on the stomach by a like disease of the pylorus—and on the walls of the heart by dilatation of that organ in consequence of obstruction. My own researches do not enable me to agree with the view that any real hypertrophy takes place in emphysema; and with reference to the explanation which has been given by Louis of the causes producing hypertrophy, it appears to me that no analogy whatever exists between the cases he has brought forward and the disease under consideration. It is true that in emphysema there is a retention of air in the air-sacs, but there is no obstruction to its exit from them; and even if such obstruction did exist, there would be no parallelism between the condition thus produced in the lung, and that produced in the organs alluded to by M. Louis. The air is not driven from the air-sacs—as the blood is driven from the heart, or the food from the stomach—by forcible contraction of their walls, but by their elastic reaction. Now, in emphysema this elasticity is impaired or lost; the air is consequently retained passively, and not because it cannot be forced through an obstructed orifice. The air-sacs are not called upon to perform extra work; and no element of hypertrophy, such as M. Louis supposes, can consequently exist.

In considering the pathology of a disease, as I have before remarked, we often derive material aid from observing the manner in which it is influenced by certain remedial agents; and with reference to emphysema, I think we may gather from this source

very important indications. From a close study of the disease for some years past, and a careful observation of the results of treatment in numerous cases, I am convinced that, setting aside the bronchial and asthmatic symptoms which are so constantly associated with the affection, the main principles on which emphysema should be treated are precisely those which guide us in the treatment of diseases attended with degeneration—such, for instance, as Bright's disease of the kidneys, and fatty degeneration of the heart. The principles must be modified in reference to the peculiarities of emphysema; but, as all these points will form the subject of consideration in the chapter devoted to the question of treatment, they are merely alluded to here in connection with certain inferences which will be found below.

In concluding this portion of my subject, I shall state briefly the circumstances which induce me to believe that emphysema is the result of some degenerative process; and although it would be more satisfactory to be able to speak positively as to the exact nature of the degeneration, yet, for all practical purposes, it is sufficient to point out the general pathology of the malady, and to indicate the principles on which it should be treated.

1st. The high degree of development which the disease often reaches, without any previous history of violent or long-standing cough, either in connection with bronchitis, hooping-cough, or any similar affection.

2nd. The frequency with which the disease attacks the whole of both lungs; and the uniformly equal character of the morbid changes often observed throughout all parts of the lungs.

3rd. The hereditary nature of the disease, as shown by the observations I have alluded to.

4th. The manner in which the disease is influenced by certain remedial measures which are known to act beneficially on other diseases attended with degeneration of tissue.

CHAPTER III.

THE DETERMINING CAUSES AND MECHANISM OF EMPHYSEMA.

Two Principal Theories of the Determining Causes of the Disease. Views of Laennec. Theory of Dr. Gairdner. The Mechanism of Respiration. Effects produced by Collapse of the Lung. The Expiratory Theory. The Modus Operandi of Expiratory Efforts. The Anatomical Arrangement of the Walls of the Chest, and the Disposition of the Lungs. Effects of Forced Expiration. The Case of M. Groux. Results of the Author's Observations. Infrequency of the Disease as a Sequel of Pleurisy and Pneumonia; its frequent Occurrence in Tubercular Lungs. Cases recorded by M. Guillot.

I HAVE endeavoured to show, in speaking of the pathology of emphysema, that all cases cannot be included under the same head, and that some, in all probability, arise spontaneously; whilst others may be dependent upon a pre-existent affection of the bronchial tubes, and may be caused by mechanical violence. The connection of the disease with bronchitis has long been recognised by pathologists; and, with few exceptions, the general opinion seems to have been that this alone was its determining cause. It is true that Lebert admits the spontaneous origin of emphysema; and that Hasse, whilst agreeing with the general opinion that catarrh is the principal occasional cause of the disease, states that its character and real import depend on other causes, not thoroughly made out. Louis also, in his "Researches on Emphysema,"

denies the dependence of the disease in all cases on pulmonary catarrh; and admits the possibility of its development without any such previous affection. The observations of Louis are so important on this point, and the statement he makes, as to the existence of dyspnœa and oppression long before the occurrence of cough, so entirely accord with the results of my own experience, that I quote the following remarks from his paper:—"Setting aside the cases where dyspnœa could be traced back to very early youth, and in which cough did not generally supervene till much later, the oppression was not nearly always preceded by pulmonary catarrh; and in many patients catarrh did not come on till one or more years after the commencement of the oppression; whence this necessary conclusion—that emphysema can develop itself, and in fact does tolerably frequently develop itself, without pulmonary catarrh. This conclusion is again, in some measure, confirmed by this further fact, that dyspnœa often appears not to have increased in an appreciable degree after a severe acute pulmonary catarrh; and when we recollect that the maximum of emphysema usually has its seat along the thin borders of the lungs and their adjacent parts, whilst acute pulmonary catarrh has its seat behind and below, we shall be forced to conclude that, if catarrh has any influence whatever on the development of emphysema, that influence is slight, and, without doubt, but rarely exercised."*

* Recherches sur l'Emphysème des Poumons. Mémoires de la Société Médicale d'Observation. Paris, 1837. Tome i., p. 160.

I think Louis has underestimated the influence of bronchial affections in giving rise to emphysema; but of the correctness of his observations with reference to the early existence of dyspnœa I have had abundant proof.

Although there is now, for the most part, a general agreement amongst pathologists, as to the dependence of emphysema on bronchitis — and as far as regards the partial forms of the disease I entirely agree with the view — and further, that its mechanical production is in some way associated with bronchial inflammation, yet the exact manner, in which the dilatation and rupture of the pulmonary tissue are brought about, has been the subject of much discussion, and has given rise to great diversity of opinion. As the question is one not only of great interest, but of much practical importance, it is necessary to consider it at some length.

Speaking generally with reference to the determining causes of emphysema, we may say that two principal theories have been entertained: these are respectively called the inspiratory, and the expiratory, theory.

By those who hold the former view, it is supposed that dilatation and subsequent rupture of the air-sacs of the lung take place as the result of their over-distension during an inspiratory act; by those who entertain the latter view, that these results are brought about by expiratory efforts, more especially by the act of coughing.

Laennec, recognising the frequency of the disease as a sequence of pulmonary catarrh, supposed that it was occasioned by an over-distension of the air-cells, from an accumulation of air taking place in them, in consequence of the obstructed condition of the bronchial tubes. He says:—"The small bronchial tubes are distended by the viscid mucus, or by the swelling of the mucous membrane. Now, as the muscles which act in inspiration are strong and numerous, and as expiration, on the contrary, is only produced by the elasticity of the parts and the feeble contraction of the intercostal muscles, it must often happen that in inspiration, the air, after having overcome the resistance which was opposed to it by the mucus, or by the tumefaction of the mucous membrane, cannot overcome it during expiration, and remains imprisoned. The following inspirations add further to the dilatation of the cells to which the obliterated tube leads. Lastly, the distension, by the heat of the lungs, of the air introduced cold into the chest, must contribute to this dilatation."

The theory thus advanced by Laennec is based on a view of the respiratory function which has been proved to be essentially incorrect; viz., that the inspiratory power is greater than the expiratory. The researches of Hutchinson and others have shown that the power of forced expiration considerably exceeds that of inspiration. This important physiological fact cannot be too constantly borne in mind, in considering the nature of emphysema.

But further, it has been shown by the researches

of Gairdner, and others, that an accumulation of mucus in the bronchial tubes, such as Laennec thought would lead to a distension of the air-sacs, has an exactly opposite effect; and is, in fact, followed by a collapse, and not a dilatation, of the pulmonary tissue. Dr. Gairdner has shown that the pathological condition of the lung, which has been known under the name of lobular pneumonia, is nothing more than a collapse of the lung-tissue, which — unless the affection have existed for a long time, and have produced a truly atrophic condition— may be readily distended by inflation through the bronchial tube leading to it. He has found this state of the lung following bronchitis, and constantly associated with an obstructed condition of the bronchial tubes leading to the affected part. He accounts for the production of the collapse in the following manner:—

"The bronchi are a series of gradually diminishing cylinders, dividing for the most part dichotomously. If a plug of any kind, but especially one closely adapted to the form of the tube, and possessing considerable tenacity, be lodged in any portion of such cylinder, it will move with much more difficulty towards the smaller end, and in doing so will close up the tapering tube much more tightly against the passage of air, than when moved, in the opposite direction, into a wider space. If such a plug be placed over a bifurcation, it will, even if freely moving in the larger space in which it lies, be of sufficient bulk to fall back upon one or other of the

subdivisions during inspiration, in the manner of a ball-valve upon the orifice of a syringe, and thus completely occlude it. The consequence of this mechanical arrangement must inevitably be, that at every expiration a portion of air will be expelled, which, in inspiration, is not restored, partly owing to the comparative weakness of the inspiratory force, and in part to the valvular action of the plug. If cough supervene, the plug may be entirely dislodged from its position, or expectorated, the air, of course, returning freely into the obstructed part; but if the expiratory force is only sufficient slightly to displace the plug, so as to allow of the outward passage of air, the inspiration will again bring it back to its former position; and the repetition of this process must, after a time, end in a perfect collapse of the portion of lung usually fed with air by the obstructed bronchus."*

In confirmation of the view, that obstruction of the bronchial tubes leads to collapse of the pulmonary tissue, we have the results of experiments performed by Mendelsohn and Traube. These experimenters introduced into the trachea of certain animals, small hard bodies, which they pushed down into the bronchial tubes as far as possible. The general result found, on examining the lungs of these animals after death, was, that the portion of lung connected with the tube which was obstructed by the introduced plug was red and void of air; in fact, in a state of collapse. In addition to these facts and observations, which may be adduced against the theory advanced by

* The Monthly Journal of Medical Science, vol. xi., p. 242.

Laennec, it may be stated that the seat of pulmonary emphysema, when it exists as a partial affection, and that of obstructed bronchial tubes, is not the same; in fact, the two affections have altogether different localities, bronchial inflammation and collapse being most frequent in the lower and back parts of the lungs, and emphysema in the upper and anterior parts. It has been shown, by the researches of Gairdner, Lebert, and others, that pulmonary collapse and emphysema are frequently found existing together in the same lung; and the former author has so constantly seen the two affections associated together, that he has looked upon them as having the relation to each other of cause and effect. Hence he has sought for an explanation of the production of emphysema, in the altered relation which the collapsed lung bears to the cavity in which it is placed, as compared with its relations in a state of health. His opinions may be summed up as follows:— Adopting the view that emphysema is produced by the force of the inspired air acting on the walls of the air-sacs, he considers the disease in the light of a complementary lesion, depending upon the fact that a portion of the lung has become *diminished in bulk and incapable of distension.* In consequence of this condition of the lung, which is found in pulmonary collapse, those portions of the organ which remain in a sound state receive into them a larger quantity of air than usual, in order to fill the space previously occupied by the portion now collapsed: hence over-distension of the air-sacs and rupture.

Notwithstanding the very able manner in which the author of the above theory has supported his views, it appears to me that there is an error in the position he has assumed, which tends to invalidate his conclusions; and that the exclusive doctrine of the production of emphysema, which he has advocated, will not bear the test of strict clinical investigation. That, when one portion of a lung is collapsed, it necessarily follows that the sound portions of the organ will expand beyond their usual size, so as to fill the space previously occupied by the collapsed lung, and dilate the thoracic cavity to the same extent as before, appears to me to be opposed to what we know of the mechanism of respiration. During respiration the chest expands, to make room for the dilating lungs, and it will only expand to the extent required by the amount of air which enters the lungs. The air is drawn equally to all parts of the lungs; neither the muscles of the chest, nor the lungs themselves, have any power to *determine* the air to one part of the organs more than to another. No external force exists which can accomplish such a result; and therefore it seems difficult to understand how a diversity of currents could be produced in different portions of the lungs, so great as to lead to an over-distension of some parts, whilst others remained normally dilated. That, when collapse of a portion of the lung takes place, so that no air can be received into it, the same quantity of air as previously entered finds its way into the chest, so as to dilate

it to the same extent as before, appears to me extremely doubtful; and such a view is, in my opinion, opposed to other pathological facts we witness in connection with the lungs. But if we admit that this view is correct, and that in proportion as some parts of the lung collapse, others become more than normally dilated, so that the chest reaches its previous state of expansion, then the air would become diffused throughout the whole of the lung remaining sound, and not be driven, or drawn, to any particular locality. The consequence of this would be, that an increased small dilatation of every part would compensate for the want of action of the small collapsed portion. There would, under these circumstances, be no special strain on any particular part of the pulmonary tissue — no rush of air to one part more than to another. But further; if, as the result of pulmonary collapse, any portion of the lung becomes abnormally distended, so as to lead to the production of emphysema, it appears to me that it ought to be those parts which lie in contiguity to the collapsed tissue. But we do not find this to be the case; on the contrary, the collapsed portions are most frequent in the posterior and lower parts of the lungs, the emphysematous at the apex and along the margins. It is true that Dr. Gairdner has found in some cases patches of emphysema lying side by side with the collapsed tissue, and I have seen the same thing myself; but, as a rule, the two affections have, as I before stated, different seats.

From the views I have expressed above, it appears

to me very difficult to account for the production of emphysema by a forcible distension of the lung as the result of an inspiratory act; and especially when we consider the amount of distension which the lungs will bear, when in a healthy condition, without any rupture of the air-sacs taking place; a distension, probably, far greater than they undergo in those cases of disease where one lung, or part of one lung, takes on increased action, to compensate for the want of action of a disabled portion.

I pass now to consider briefly the expiratory theory of the disease. Are there any circumstances in ordinary or forced expiration which have a tendency to produce a distension of any parts of the lungs, and consequently to lead to the production of emphysema? With reference to the act of ordinary expiration, we may safely say there are none; but in regard to forced expiration, it appears to me that such circumstances do exist.

It has been urged, as an objection to the theory we are now considering, that the expiratory act is mechanically incapable of producing distension of any part of the lung; and that the air-sacs are emptied by an uniform pressure of the thoracic walls upon the whole pulmonary surface. This objection only applies to the ordinary act of expiration; and it is undoubtedly true that the lungs then undergo equable pressure on all parts, and that there is no tendency for the air to be forced towards, or retained in, any particular part of the pulmonary substance;

but the argument loses its weight when we come to apply it to forced expiration, as, for instance, to the act of coughing. The effect which is produced on the lung by coughing has been pointed out by Dr. Jenner, in a paper read before the Royal Medical and Chirurgical Society of London, and already referred to; and the remarks he has there made entirely accord with what I have myself observed. If we examine a person whose chest is exposed during the act of coughing, we see a distinct bulging produced above the level of the clavicle; in fact, it is clearly shown that the air is forced upwards by this expiratory act, and that it forcibly distends the upper parts of the lungs. Percussion of the tumour, formed as I have stated, yields a resonant sound, which becomes almost tympanitic if the lung be in an emphysematous state. Now, if we examine the anatomical arrangements of the walls of the chest, we have a ready explanation of the phenomena to which I have just alluded. The lateral and inferior walls of the thoracic cavity are strong and resisting, and by their muscular action and elasticity they assist *actively* in expiration; further, the contraction of the abdominal muscles forces upwards the diaphragm, and this more especially in violent expiration. The part of the thoracic walls which is the weakest, and which offers the least resistance, is that which separates the cavity of the chest from the region of the neck. We there find a fibrous structure, a strong fascia, in fact, connected externally with the first rib, and internally blending with the cervical

fascia as it passes down into the chest. This plays no active part in the expiratory process, and offers no active resistance to the distension of the lung. From a knowledge of this peculiar arrangement of the walls of the chest, and a consideration of the action of those muscles which are concerned in expiration, it appears to me that, during violent expiratory efforts, the lungs *must* be unequally compressed, and that air must be driven, first, to those parts of the lungs where the walls are least resisting, and, secondly, to those portions which contain the least volume of air.

I have shown above that the apices of the lungs are the parts covered by the least resisting walls ; and it will at once occur to all, that the parts which contain the least volume of air are the anterior borders and the margins of each base. These parts are not only the thinnest, but they are also out of the direct line of strongest pressure which the lungs undergo in expiration. Violent expiratory efforts are chiefly made with the abdominal muscles, and the most powerful agents are the recti ; the contraction of these muscles, forcing upwards the abdominal viscera and the diaphragm, produces the greatest amount of compression at the base of each lung; the air is consequently driven upwards in a strong current. There being no corresponding force acting at the upper part of the chest, on the apex of the lung, this latter is not emptied ; on the contrary, it becomes forcibly distended by the upward current. Further, the strong currents of air from the central and basic portions of the lungs overcome those from the thin

portions; and thus these latter, instead of being emptied, become, like the apex, forcibly distended. Dr. Jenner supposes that the cartilaginous portions of the thoracic walls are somewhat yielding, and thus accounts for the production of emphysema along the borders of the lung. This explanation seems to me doubtful, and the one I have given as far more probable.

Again, I may refer to the phenomena which were witnessed in the case of M. Groux, who was over in this country some years ago, and made a tour of many of the metropolitan and provincial towns. In this case a fissure of the sternum existed, which allowed of some of the movements of the heart being observed. Those who examined M. Groux will recollect that, during a violent expiratory act, the lung of one side came forward in the upper part of the fissure, and formed a distinct elongated resonant tumour; no such result taking place during inspiration. Whatever influence this fact may have on our minds with reference to the expiratory theory of emphysema, it tends, at any rate, to show that wherever there is a weak part, or an absence of compressing power, in the thoracic walls, the lungs will there undergo distension during forced expiration.

The facts and arguments I have adduced seem to me to prove that the objection to the expiratory theory, on the ground that the expiratory act is mechanically incapable of producing distension of any part of the lung, is of an untenable character; and, without expressing my own attachment to any

exclusive theory of the production of emphysema, I may remark that my observations of a considerable number of cases have led me to conclude, that by far the most frequent cause of the partial form of the disease, is to be found in the cough attendant on bronchitis, or some other affection of the bronchial tubes, such as pertussis. The fits of coughing, caused by the repeated attacks of bronchial inflammation, must, it appears to me, so react on the pulmonary vesicles which are most liable to distension, as to produce, after a time, their dilatation and rupture; and I the more incline to this view of the production of the disease, from knowing how difficult it is to produce artificial emphysema by inflation of the lungs, even after death. It may be objected to this view, that emphysema should be more frequently found as a sequence of pleurisy or pneumonia. We know that in some cases of inflammation of the lungs partial emphysema *is* produced. I have seen this when the emphysema was apparently quite recent, and had, probably, been produced during the progress of the pneumonia. Such a result, however, only rarely follows, and I think we have an explanation of the fact in the character and short duration of the cough attendant on this disease, as well as on pleurisy.

Again, it appears to me that the frequency with which we meet with emphysema in tubercular lungs, favours the view I am taking. The deposition of tubercles in the pulmonary tissue filling up the air-sacs in the same way as a pneumonic exudation, with the subsequent gradual contraction of the walls

of the chest, presents no condition favourable to the formation of emphysema, except the cough, which is usually so important a feature of the disease.

M. Guillot has collected a series of cases, to which I have already referred, illustrating the effects of long-continued spasmodic cough in producing emphysema. These cases, recorded with some excellent observations, are fifteen in number; the subjects of them were infants, most of whom suffered from pertussis, but in all, long-continued spasmodic cough was a very prominent symptom. Death took place in all, and the *post mortem* examination revealed the existence, to a very considerable extent in some cases, of sub-pleural emphysema, with, in some instances, extravasation of air into the areolar tissue of the mediastinum, and even of the neck. A careful examination of the sub-pleural vesicles showed that the air had extravasated into them in consequence of rupture of the pulmonary tissue. No mention is made of pulmonary collapse.

The cases recorded by M. Guillot appear to me of great importance, as tending to establish the connection between the cough from which the patients suffered, and the pathological results above referred to; and to prove, beyond the possibility of doubt, that violent expiratory efforts and emphysema may stand in relation to each other as cause and effect.

But although I am disposed to consider that, in the majority of cases of emphysema, such as I have described as *partial lobular* and *lobular*, the most frequent cause is the cough, which may have existed

for a longer or shorter time; yet this view of the determining cause is by no means sufficient to satisfy my mind with reference to that more important and extensive form of the disease denominated *lobar*. Cases of this kind are not unfrequently met with, as I have previously stated, where the disease has gradually crept on without the existence of any severe or long-continued cough, and where there has been little or no bronchitis. It cannot be said that in such cases there has been any mechanical force, except that of ordinary respiration, acting on the pulmonary tissue so as to produce a distension and rupture of the air-sacs. It will scarcely be admitted that the pressure produced by inspiration is capable of bringing about such results, provided the lung tissue be healthy. We know the lungs may be distended far beyond the point to which they ordinarily reach, without any of their fibres giving way; and the point, to which they reach under the condition of extreme expansion of the thorax, falls far short of that to which they may be distended, without injury, when removed from the body after death. In fact, there can be no doubt that very considerable force is required to rupture the healthy lung.

Again, cases of lobar emphysema are not unfrequently seen, where, after death, the whole of both lungs is found involved in the affection, but where there is no appearance of collapse of the pulmonary tissue, or, if any, only to a very partial extent. The existence of such cases seems to me to oppose a

strong argument against any exclusive view of emphysema being produced as a result of pulmonary collapse. It may possibly be said that, in the cases referred to, the collapse is of the diffused kind, and is followed by perfect recovery. Such an explanation would be, I think, a very doubtful one; and it might be fairly asked, since every part of the lungs is more or less affected with emphysema, how the disease has been produced in the collapsed portions.

To what mechanical cause, then, are we to attribute the distension of the lungs under the circumstances just mentioned? From the non-existence of cough and bronchitis, and the prevalence of the disease throughout the entire lung, we cannot attribute any effect to expiratory efforts, and I am disposed to think that the distension is brought about by inspiration; that the lung tissue, being in an unhealthy condition, and abnormally weak, gives way before the pressure which it would, in a state of health, resist; that, having once yielded, it is unable to recover itself, from having lost its elasticity. Consequently, it undergoes further distension, as increased efforts are made to dilate the chest in order to meet the requirements of respiration; until, at length, the thorax having reached its extreme point of dilatation, no further enlargement of the lungs can ensue.

It will be seen that, in advocating this view, I am all the while supposing that the primary step in the disease is a degeneration of the lung-tissue, and that the mechanical distension is a secondary consequenec.

If the views I have enunciated be correct, it follows that not only must we place different kinds of emphysema under different heads as to their pathology, but also as to their determining cause.

CHAPTER IV.

THE SYMPTOMS AND PHYSICAL SIGNS OF EMPHYSEMA.

Dyspnœa and Cough. Hæmoptysis. The Occurrence of Asthmatic Symptoms. Aspect of Patients. General Configuration of the Chest. Movements of the Chest in Respiration; their Variations in different Cases; Causes of these Variations. Character of Inspiration and Expiration, and of the Act of Coughing. Percussion and Auscultation. Character of the Respiratory Sounds. The Physical Signs and Symptoms referable to the Structural Alteration in the Lung-Tissue. Diminution of the Respiratory Function, and of the Quantity of Blood circulating in the Lungs. Diminished Temperature of the Body.

Amongst the most important and frequent of the symptoms of emphysema is a constant, and generally increasing, shortness of breath. It will usually be found that the patient has been for some time subject to this symptom, which is always increased on exertion — more especially such as that of walking up-hill or up-stairs — whenever the stomach is much distended, or on the occurrence of a catarrhal attack. The dyspnœa is mitigated during the summer months, but returns again, with increased severity, with each succeeding winter. Cough is generally more or less present, and is accompanied by expectoration in proportion to the bronchitic symptoms. The expectoration varies in its character, and may be occasionally streaked with a small quantity of blood;

hœmoptysis, however, never exists to any great degree: its occurrence is undoubtedly due to the rupture of the capillary blood-vessels, which takes place as the lung-tissue is distended. Such rupture must frequently occur, but in the majority of cases is unattended with hœmorrhage. The patient usually complains of no pain, but of a general feeling of oppression, or "smothering in the chest," as he often terms it. It frequently happens, in the most severe cases of lobar emphysema, that this last symptom, and the increasing dyspnœa, at times becoming very urgent, are the only circumstances which have attracted the attention of the sufferer to his disease. In other cases, however, and especially where the emphysema is only partial, a close examination will elicit the fact that bronchitic attacks have preceded the affection, and have been associated with it in its progress.

Few, if any, cases of emphysema, especially where the disease is extensive, exist for any lengthened period without the occurrence of asthmatic symptoms. The patients complain of being attacked with difficulty of breathing, and tightness across the chest in the night. The attack usually comes on some time between twelve p. m. and four a. m., and, after lasting a variable time, subsides in a fit of coughing, attended with expectoration. As I shall have hereafter to speak of the close relation existing between emphysema and asthma, I shall not dwell on this part of the subject now.

In advanced cases of the disease, the aspect is

peculiar, and very characteristic; the countenance is dusky, and sometimes has a puffy appearance, the result of venous congestion, and indicative of the imperfect manner in which the aeration of the blood is performed. The nostrils are dilated, and are seen to expand widely at each inspiration; at the same time the angles of the mouth are drawn downwards. The voice is feeble, and there is an inability to sustain a prolonged, uninterrupted expression. The whole body has more or less of a cachectic appearance, and is sometimes much wasted. To these symptoms must be added those of general dropsy, which not unfrequently follows as a consequence of the effects produced by the disease on the right cavities of the heart.

Amongst the most important of the physical signs of emphysema may be enumerated the following:— The upper part of the chest and the clavicles are very prominent; the neck appears shortened; in fact, the whole of the thorax is drawn upwards, for the ribs have in great measure lost their obliquity, and their anterior extremities are at a higher level than in a state of health. The fossæ above the clavicles are deepened,* and the tense cords of the

* M. Louis speaks of a prominence behind the clavicles in some cases of emphysema, by which an appearance of plumpness was given to the supra-clavicular regions. I cannot say that I have observed such a condition in well-marked cases that have come under my notice; and I am confirmed in the remarks I have made in the text, as to the existence of a depression behind the clavicles, by the testimony of Hasse, who says he has never observed anything like a prominence. The depression alluded to is produced, not by the falling away of the lung, but by the elevation of the clavicles.

hypertrophied sterno-cleido and scaleni muscles stand out in relief. There is increased curvature of the dorsal spine, and the sternum becomes arched instead of straight. The gait is stooping, and the head and shoulders are thrown forwards. When in bed, the patients will often be observed to sit up, with the body bent forwards, and the arms folded and resting on something, in order that the accessory muscles of inspiration may be the more readily brought into play. The ribs are prominent, and the intercostal spaces, generally, appear depressed. In well-marked and advanced cases, there is an increase in the size of the chest, and a rounded or convex condition, especially of its upper part. The existence of an enlarged thorax in emphysema has been maintained by almost all those who have written on the disease; but the fact has been disputed by Dr. W. T. Gairdner, who asserts that there is no enlargement of the chest as a whole, and that the increased capacity of the upper, is counteracted by a diminution of the lower, part. I am disposed to think that Dr. Gairdner is in error in regard to this point, and that he has been misled by the fact that, in emphysema, there is always more movement of the upper costal portion of the chest than of the lower. Indeed the lower ribs scarcely move at all; and in bad cases, what movement of them does take place is just the opposite of that which occurs in health, for, together with the lower end of the sternum, they fall in during inspiration. My belief with regard to this question, of increased capacity of the chest, is, that there exists, for the

most part, a considerable enlargement of the upper part, and no diminution of the lower. There are some cases, however, in which the floor of the chest yields greatly to the expanding lungs, and where, consequently, from the diaphragm being pushed down, the thorax acquires increased depth; hence the dilatation of its upper part is less marked.

The conditions I have described are those which are found when the disease is extensive. When more partial, confined, for instance, to one lung, or to parts of one lung, the prominence of the chest may exist on one side only, and the other symptoms enumerated will be less marked.

The movements of the chest in respiration are peculiar. The breathing is for the most part superior thoracic; there is, however, not much dilatation of the chest, even at its upper part; for the distended lungs having already inordinately expanded the cavity, there is but little room for further enlargement. The accessory muscles of inspiration in the neck, the sterno-cleido mastoid and the scaleni, are seen in constant, and often powerful, action. The chest is raised, and slightly pushed forwards above, during inspiration, whilst below, it sinks in. This recession of the lower part of the sternum, and of the lower ribs, is very obvious in well-marked cases of the disease.

The respiratory movements vary in different cases, depending on causes which I shall hereafter explain. In some patients we find that there is little or no action of the diaphragm in inspiration; in fact, during

this process the abdomen remains flattened, or even concave. Here the upper part of the chest has yielded to the expanding lungs, and is very prominent. In a second class of cases the respiration is strikingly different. At every inspiration the thorax is raised, almost straight upwards, with even less expansion than is observed in the first set of cases, whilst at the same time the abdomen is protruded somewhat forcibly and rapidly forwards. The abdominal protrusion differs from that which takes place in a forced inspiration in health, in that the part protruded is lower; for, whilst the lower abdominal regions are pushed forwards, the upper ones, together with the lower ribs, are drawn inwards.

The different character of the breathing, in these two classes of cases, was accurately pointed out by Dr. Stokes; and I can fully confirm the observations he has made as to the facts I have referred to.

Upon what causes do these differences in the respiration depend? It is obvious, I think, that they must be explained on the ground that, in the one set of cases, the diaphragm has been pushed downwards by the expanding lung, whilst, in the other, it has maintained its normal position, and the principal yielding has been in the upper part of the thorax. From my own observations, I am disposed to believe that the cases, which are not characterised by displacement of the diaphragm, are those where the disease is chiefly located in, or has originally attacked, the upper part of the lungs. Here the bony walls of the chest have yielded before the expanding organs.

Where, however, there is diaphragmatic displacement, I believe it will be usually found that the disease is of a general character, that the whole of the lungs is affected, and that the malady has commenced and progressed in their lower, at the same time as in their upper, parts. I am supported in this view by the fact that these cases are the most urgent in their symptoms, and are characterised by the greatest amount of dyspnœa. I have had opportunities of examining such cases after death, and have found the lungs universally emphysematous, their bases flattened, and the diaphragm, with the abdominal viscera, displaced downwards.

The respiration presents another feature, which, in well-marked cases, is alone sufficient to indicate the nature of the disease. The inspiration is short, and quick, and is followed by a prolonged, and often wheezing, expiration. The former phenomenon is due to the small expansion which the chest undergoes, and the quick action of the inspiratory muscles; the latter, to the slowness with which collapse of the lungs takes place, in consequence of their loss of elasticity.

The act of coughing, in emphysema, is always feebly performed, and expectoration is attended with difficulty. This is of the more serious importance, in consequence of the great liability of emphysematous patients to bronchitis, and of the profuse secretion which occurs in such attacks, if at all acute. The presence of mucus in the bronchial tubes causes irritation, which is only increased if the attempts to

raise the secretion are ineffectual; and thus spasmodic fits of coughing are often produced, and even suffocation is occasionally threatened.

Percussion and auscultation elicit important diagnostic marks in this disease. Where the affection is general, there is increased, and, in some instances, almost tympanitic, resonance over the whole of the chest, most marked towards the apices of the lungs, and along their anterior borders, and, in partial cases, almost confined to these spots, or to one side, if only one lung be affected. Unlike what occurs in healthy lungs, viz., that the clearness of the percussion note is augmented after inspiration, this latter act produces but little change in the resonance of the emphysematous organ. In advanced cases, and especially where both lungs are affected, the præcordial region is resonant, from the heart having been pushed backwards, downwards, and towards the median line, by the expanding and overlapping left lung. In some partial cases the cardiac region retains almost its normal amount of dulness; and again, in other cases, from pleural adhesions having prevented the lung from overlapping the heart, the latter organ may be in its usual position as regards the walls of the chest. Again, the enlargement of the right side of the heart, which so frequently follows in this disease, may sometimes, but I think very rarely, give rise to dulness on the right side of the lower part of the sternum.

An examination of the back of the chest will show that the line of resonance has a lower level than in

health. In the normal state, the lungs do not reach to the bottom of the pleuræ; but, when enlarged by emphysema, their lower margins are pushed down to the extreme base of those cavities. The cardiac impulse can be neither seen, nor felt, in the normal position; in advanced cases, however, from the expanding lung having dislodged, and encroached on the space of, the heart, the latter is observed beating, just below the ensiform cartilage, in the epigastric region. The impulse here is strong, and offers a marked contrast to the pulse, which is generally small and feeble. These phenomena are due to certain changes which take place in the heart, and which will form the subject of future consideration.

I have spoken of the displacement which the heart undergoes in emphysema. There is another circumstance to which I must refer, in connection with its sounds. Generally speaking, when listened for at the usual position of the apex, the sounds are more feeble and obscure than natural; but very frequently, if the examination be made at a lower level, and nearer the median line, they will be distinctly heard. In fact, in consequence of the displacement of the heart, the site at which the sounds are best heard is altered. This remark especially applies to those cases where the heart can be felt beating in the epigastrium, and where, consequently, the displacement is considerable; in other cases, where the heart is merely pushed back and overlapped by the lung, the sounds are everywhere feeble and obscure.

The quantity of air entering and leaving the lungs in emphysema is small; hence the respiratory murmur is feeble, and characterised by peculiarities which a knowledge of the anatomical condition of the lung-tissue, and of the walls of the chest, at once enables us to explain. The inspiratory sound is short, sometimes very faint, and is followed by a prolonged expiratory murmur. This latter is unlike the sound heard in any other affection, and is, in fact, pathognomonic of emphysema.

In the early stages of the malady, the respiratory sounds are merely feeble, and no marked prolongation of the expiratory murmur is heard. It is important to be aware of this fact, and to diagnose the affection at an early period of its course, when there is the greater probability of the use of remedial agents being attended with success. Auscultation may here give more valuable aid than percussion, for some dilatation of the air-sacs may exist, without causing that amount of resonance which would attract attention; in such cases, however, the feeble character of the breathing will clear up any doubt we may entertain.

In some advanced cases of the disease, the respiratory sounds are scarcely audible, if the bronchial tubes are free from inflammatory action, and no spasm exists.

Laennec has described a râle which he considered peculiar to, and pathognomonic of, pulmonary emphysema. He calls it, *râle crépitant sec à grosses bulles.* Most authors who have written on emphysema since

Laennec, have expressed doubts as to the occurrence of any râle characteristic of the disease. I have, on several occasions, heard a râle in emphysema, which, I believe, was similar to that described by Laennec, and I have never had the slightest doubt as to the nature of the cases in which it occurred. The diagnosis, however, was not formed simply from the existence of the râle, but from the presence of other more prominent symptoms. Laennec described the râle as *dry*, and supposed that it was produced by the distension of the emphysematous lung-tissue. The râle with which I am familiar, appears to me of a moist, rather than a dry, character, and to be closely allied to the sub-crepitant râle of bronchitis. In many cases of extensive emphysema it is not heard, and the instances in which I have found it have been those where there has been previous inflammation of the bronchial tubes. My belief is, that the râle is produced, not in the way that Laennec supposed, but in consequence of the presence of a certain amount of fluid in the finer air-passages. As a sign of emphysema, the râle is important, and may assist in confirming a diagnosis; but in consequence of its frequent absence, and of the difficulty which must exist in distinguishing it from an ordinary sub-crepitant râle, it loses a good deal of the value which would otherwise attach to it.

A knowledge of the morbid changes produced by emphysema, affords a ready explanation of the peculiar character of the respiratory movements and sounds, as well as of the other physical signs and

symptoms of the disease. It also enables us to explain the occurrence of the secondary consequences which ensue in the progress of the affection; a subject to be dwelt on hereafter.

The lungs being the seat of general expansion, the thorax is kept abnormally distended; hence, between the point to which it is contracted at the end of expiration, and that of its extreme distension, there is much less difference than in a state of health. The lungs undergo but little enlargement at each inspiratory act. There being no impediment to the passage of the air to the extreme parts of the lungs, the air-sacs, the inspiration is rapidly accomplished. Not so, however, the expiratory act. The pulmonary tissue has, in great measure, lost its elasticity, and reacts but slowly after distension. As collapse, or elastic reaction, of the lungs, is one of the most important elements in expiration, any imperfection in it necessarily results in a laboured, slow, and ineffectual expulsion of the air. Further, in advanced states of the disease, when the pulmonary tissue is riddled with perforations, there is an additional cause of impediment to the exit of air from the air-sacs, from the side currents which must necessarily be produced. It is obvious that, under the conditions I have described, the respiration can be, at the best, but imperfectly performed; and that there is no room for increased expansion of the chest walls, nor any possibility of the frequency of the respiratory acts being much increased, to meet the requirements of extraordinary exertion. So long as

muscular action is slight, and the circulation quiet, the lungs may perform their work without producing distress; but anything which tends to accelerate the circulation, immediately upsets the balance, and gives rise to dyspnœa and imperfect aeration of the blood.

M. Louis has endeavoured to account for the dyspnœa in emphysema on the supposition that the walls of the air-vesicles (air-sacs) are thickened, or hypertrophied, in the disease. It is quite obvious, as he remarks, that a structural alteration of this kind would interfere with the reciprocal action between the air and the blood in respiration, and would render that action less perfect than in a state of health. But, setting aside the fact, which Louis himself admits, that no proof has ever been afforded of the existence of hypertrophy, we can have no difficulty whatever in accounting for the dyspnœa, without the recognition of any such theory. The diminished amount of aerating surface, in consequence of the destruction of the pulmonary tissue, and the imperfect manner in which the air is renewed in the lungs, are quite sufficient to explain the difficulty which occurs in the breathing, either when the circulation is accelerated by exertion of any kind, or whenever an attack of bronchitis occurs.

Not only is there, in emphysema, diminished respiration, but also a diminution in the quantity of blood circulating in the lungs; for the anatomical changes which take place in the pulmonary tissue cause an impediment to the flow of blood through it It is an ascertained physiological fact, that, when the lungs are in a state of over-distension, the passage

of the blood through them is less free than when they are more moderately distended; and a similar result is observed with regard to fluids injected into the lungs, when removed from the body. From this we may gather that, in the earliest stages of emphysema, there is an impediment to the pulmonary circulation; and we find, in fact, that, as the disease progresses, the capillary vessels become ruptured and absorbed, and a considerable diminution takes place in the vascularity of the lung-tissue. Hence we have the pale, anæmic, and dry condition, so characteristic of the emphysematous lung, a condition which serves to explain how it happens that the organ is so rarely attacked with pneumonic inflammation, or even extreme congestion.

An important feature observed in this disease is a decrease of the temperature of the body—a condition which results from the diminished quantity of arterial blood circulating in the system, and perhaps, also, from the fact that the blood, which reaches the left side of the heart, has only undergone a partial oxygenation; for both the aerating surface of the lungs, and the power of renewing the air in them, are diminished by the disease. The peculiar aspect of some patients suffering from advanced emphysema—their dull, heavy appearance, the slowness of their movements, and the general torpor of their animal functions—all indicate an imperfect aeration of the blood, and a liability to those secondary diseases which a mal-performance of the respiratory function may engender.

CHAPTER V.

THE SEQUELÆ OF EMPHYSEMA, AND THE DISEASES ASSOCIATED WITH IT.

Bronchitis; its General Characters; Sources of Danger in Severe Cases; Deposit of Fibrinous Clots in the Heart, etc. Asthma; its frequent Occurrence; its Relations to Emphysema. Disease of the Heart; Parts involved; Causes. Dropsy. General Cachexia and Anæmia. Extravasation of Air into the Areolar Tissue of the Trunk. The Relations of Emphysema with Phthisis, etc.

Amongst the most frequent of the affections associated with emphysema is BRONCHITIS. It is rare, indeed, for the former disease to exist for any length of time, without the supervention of the latter. As it is no part of my present purpose to consider the subject of bronchitis generally, I shall merely allude to the peculiarities it presents when attacking emphysematous patients. In its milder, and more frequent form, it offers no features requiring special comment. Of the treatment appropriate to it I shall speak in a subsequent chapter. Pathologically, the disease, whether assuming a mild or severe aspect, appears to me one of congestion, or of inflammation of a low type, rather than of an active character. It is attended, when severe, with very profuse secretion;

a circumstance, which, coupled with the fact that expectoration is less easily accomplished than when the lung-tissue is in a healthy state, seriously complicates the affection, and increases the danger of death from apnœa. The inflammation sometimes runs on very rapidly to suppuration, and copious purulent expectoration occurs. Even when this has been the case, an examination of the bronchial tubes after death may reveal but little vascularity of the mucous membrane, especially in the smaller divisions, which I have found full of pus, but yet presenting a somewhat pale and anæmic appearance. In the larger bronchial tubes more evidence of inflammatory action will be seen, and especially if the attacks of bronchitis have been frequent.

If these severe cases of bronchitis are not at once appropriately treated, the symptoms very rapidly assume a most urgent character, and life is in imminent danger. Such cases as these, where the early symptoms have been neglected, are occasionally met with, and that, more especially, in hospital practice, and in the colder seasons of the year. Several have fallen under my notice; and I have preserved notes of some which have been admitted into hospital, and which I have carefully watched till recovery or death took place. Such cases present, for the most part, the following symptoms, when first seen: — The surface of the body is cold, the lips are blue, the whole integument is more or less livid, and the respiration very laboured; there is little or no general expansion of the chest on inspiration, but a rapid elevation of

its upper part, with slight protrusion forwards, and in some cases a rapid descent of the diaphragm; the pulse is quick, weak, and small; the voice is feeble, and sometimes the patient can only speak in a whisper; the chest is very resonant; coarse, moist râles are heard all over the lungs; and, if the strength is not too far gone, there is copious expectoration. The mental functions are seriously impaired; in fact, from the gradual blocking up of the bronchial tubes, apnœa is coming on. Unless immediate relief be afforded to such cases, they rapidly go on from bad to worse, and death soon closes the scene. A *post mortem* examination reveals, not only the existence of a large quantity of purulent and muco-purulent fluid in the bronchial tubes, but also the presence of fibrinous clots in the cavities of the heart, and in the large vessels connected therewith.

There are two causes which tend to destroy life in these cases, and it is of the utmost importance that we should bear this fact in mind whenever we have to deal practically with the disease. The first, is, the imperfect aeration of the blood, producing the phenomena of slow apnœa, and resulting from the accumulation of fluid in the bronchial tubes; — the second, is, the constantly increasing obstruction to the circulation of the blood, from the gradual encroachment made on the calibre of the blood-vessels about the base of the heart, and even of the cavities of the heart themselves, in consequence of the formation of fibrinous clots.

The formation, during life, of the fibrinous clots I

have just alluded to, is a very interesting circumstance in these cases, and serves to explain the very small and feeble character which the pulse assumes, although, at the same time, the heart may be acting vigorously. In some cases, I have seen, after death, a large portion of the cavities of the heart occupied by these deposits, and the calibre of the pulmonary artery and aorta seriously diminished by them. It is obvious that these deposits are only secondary to, and, in fact, produced by, the first cause of death to which I have alluded—viz., the imperfect aeration of the blood. In proportion as the bronchial tubes become loaded, so does the pulmonary circulation, and subsequently the general circulation, become retarded, and thus a condition is produced favourable to the coagulation of the fibrine in the blood.

We have no means of ascertaining the presence of these clots during life, except from the general symptoms; these latter, however, often afford us sufficient indications, and I have been able to diagnose, during the progress of a case, the existence of deposits, and to verify the opinion I had formed by a *post mortem* examination.

Asthma, occurring with greater or less severity, is a very frequent attendant on emphysema; so much so, that I am not aware that I have ever seen a case where the organic disease has been of long standing, in which asthmatic symptoms have not supervened. The attacks come on, for the most part, during the

night, and, after lasting for varying periods, terminate in the usual manner of such seizures.

An explanation of the occurrence of asthmatic symptoms at night has been sought for, on the supposition that a certain amount of congestion of the lungs takes place during that period, from the position assumed by the body, and the want of activity, during sleep, of the respiratory process. This congestion is supposed to set up an irritation, which gives rise to a reflex act, resulting in a spasmodic contraction of the muscular fibres of the bronchial tubes. It is by no means improbable that this view is generally correct; and, especially with regard to emphysematous patients, the occurrence of congestion of the lungs during sleep need excite no surprise; for when we consider the imperfect manner in which the pulmonary circulation is carried on, under the most favourable circumstances, we have no difficulty in understanding how congestions may take place, when the circumstances of position and inactive respiration tend to retard the circulation.

But whatever may be the exact pathological explanation of these attacks, certain it is that they very frequently occur, and form a most distressing symptom, in cases of emphysema. Their frequent repetition leads to that hypertrophied state of the muscular fibres of the bronchial tubes, which I have described in speaking of the morbid anatomy of the disease.

In all cases of asthma, a careful examination of the lungs should be made, in order to ascertain

whether emphysema exist; and I believe that in the majority of cases it will be found. Without entering into a consideration of the general question of the nature of asthma, or of its existence as a purely spasmodic affection, unconnected with any organic disease of the lungs, I may, however, remark, that my own experience leads me to conclude that by far the most frequent cause of asthma is emphysema. Although I do not deny that asthma, commencing as a purely spasmodic affection, may give rise to partial emphysema, I yet believe that it far more frequently happens that the emphysema is the primary disease. Except the cough, from which asthmatic patients suffer, I cannot see any cause which would be likely to produce emphysema in them. The spasmodic closure of some of the bronchial tubes, and the violent efforts that are made to inspire, do not appear to me probable causes of the dilatation and rupture of the air-sacs; and, for the reasons I have assigned in speaking of the determining causes of emphysema, any theory which regards the disease as a supplementary lesion appears to me deficient.

It is well known that, in cases of emphysema, secondary affections of the HEART constantly supervene. It has been the opinion of many pathologists that the right cavities alone become affected; but the researches of Gairdner, Lebert, and others have satisfactorily proved that, in the majority of cases, when emphysema is extensive, and of long standing, the cardiac disease is not confined to one side. My

own observations tend to confirm this opinion, and to show that the form of heart disease, most frequently associated with emphysema, is a general hypertrophy and dilatation of the ventricles; for I have never seen a *post mortem* examination of a case of extensive and long-standing lobar emphysema, in which the left cavity, as well as the right, was not affected. Lebert, in his *Pathological Anatomy*, speaks of twenty-five autopsies of cases in which there was extensive emphysema; and of these, in sixteen there was a marked affection of the heart. Of the sixteen, there were nine in which the hypertrophy was general, instead of being confined to the right side; in six there were changes peculiar to general hypertrophy, with mitral disease in addition; and in one there was insufficiency of the aortic valves.

Louis mentions that he met with diseased heart in ten, out of nineteen, cases, in which emphysema was more or less present; and Hasse states that, in extensive emphysema, he has only found heart disease absent once.

The frequent occurrence of cardiac disease, in connection with emphysema, is a fact fully established. That the disease is produced as a consequence of emphysema, we can have no difficulty in believing, when we consider the nature of the lung affection, and especially that the palpitation, and other cardiac symptoms, are always preceded by distress in the breathing, and other signs, indicative of the pulmonary malady.

Hypertrophy of the ventricles is not the only

change which takes place in the heart, for valvular disease is frequently found. The deposits and thickening, which occur about the valves, are no doubt secondary to the changes which take place in the muscular walls, and must be attributed to the general mal-nutrition produced by the pulmonary disease.

When we consider the anatomical arrangements of the pulmonary tissue, and especially of the pulmonary blood-vessels, in emphysema, we can at once give a rational and satisfactory explanation of the causes which lead to hypertrophy and dilatation of the right cavities of the heart. The impediment which exists to the circulation through the lungs, in consequence of the physical condition of the lung-tissue, and the imperfect aeration of the blood, together with the diminution in the number of the pulmonary blood-vessels, must necessarily give rise to an overloaded state of the right cardiac cavities, and to increased action on their part.

No such explanation as that just given will, however, apply to the hypertrophy, so commonly found, of the left ventricle; for there is a diminution in the quantity of blood which finds its way into that cavity, and consequently, on this account, rather less call for muscular action than in a state of health. We might therefore infer that we have in these circumstances an element of atrophy, rather than of hypertrophy. But morbid anatomy teaches us that the latter usually exists. Whence, then, does it arise?

It appears to me that we must look, for an explanation of the fact, to the effects produced on the heart

by the displacement it undergoes in the disease. This displacement is always the greatest where the emphysema is most extensive, and it is in such cases that the left ventricle becomes most hypertrophied. As the lungs expand, the heart is pushed away from its normal position; and consequently, the direction of the axis of its cavities is altered with reference to that of the vessels connected therewith. The ventricles of the heart are so placed, in a state of health, with regard to the arteries which issue from them, that no impediment exists to the onward passage of the blood, and the circulation is effected with the smallest possible expenditure of muscular force; but all displacement of the heart necessarily alters the mutual relations between these several parts, and produces an embarrassment of its action—an embarrassment that can only be overcome by more powerful contraction. We consequently find that a result follows, similar to that which takes place in aortic regurgitation, viz., hypertrophy.

I can see no other valid reason that can be assigned for the occurrence of this hypertrophy of the left ventricle, than the one I have mentioned. The same cause must also have an influence in producing the changes which take place in the right ventricle; but here another cause is also at work.

In speaking of the symptoms of emphysema, I have alluded to the powerful impulse of the heart, often felt in the epigastric region, as being in marked contrast to the smallness and feeble character of the pulse; as felt, for instance, at the wrist. A knowledge

of the condition of the heart, and of the state of the pulmonary circulation, serve to explain these phenomena. The powerful impulse of the heart is the result of its hypertrophy, and embarrassment from position; whilst the small pulse is due to the small quantity of blood the left ventricle expels at each beat, and its feebleness to the fact that the force of the left ventricle is expended, in part, in overcoming the resistance which exists to the passage of the blood from the ventricle, in consequence of the altered position of the latter—and in part merely, in distending the arterial tubes. The diminished circulation through the pulmonary tissue, and the accumulation of the blood in the venous portion of the circulating system, sufficiently account for the small quantity of that fluid which the left ventricle has to react on at each beat.

As a consequence of the disease of the right cavities of the heart, and the impediment which exists to the flow of blood through the lungs, in emphysema, congestion of the entire venous system results, and is often followed by DROPSY. Cases of emphysema may, indeed, go on for a long time, without the occurrence of any dropsical symptoms whatever; and in other cases nothing more is produced than a slight œdema of the legs and feet. Where, however, the right cavities of the heart have become much dilated, tricuspid regurgitation ensues, and the dropsy often assumes a severe character.

Emphysema, when it exists in a general form, is

attended in its progress with more or less of CACHEXIA and ANÆMIA. In some cases there is much wasting of the muscular system, even before any symptoms of dropsy occur. I have seen instances of this kind, more especially in young people, where the disease had existed for several years, and where there was a good deal of emaciation and muscular atrophy. This wasting of the muscles and fat sometimes causes a marked contrast between the appearance of the trunk and limbs, and that of the face. The latter is often somewhat full and puffy — a circumstance which results, in part, from causes I have already alluded to, but probably also, in part, from an hypertrophied condition of the respiratory muscles of the face, which are constantly brought into more than ordinary action.

Further, as a consequence of emphysema, we frequently find the complexion assume a sallow and anæmic appearance, not unlike that met with in disease of the kidneys, and other serious organic affections — an important symptom, indicative of an impoverished state of the blood, and general mal-nutrition.

The occurrence of the secondary consequences to which I have alluded renders emphysema a disease of serious import. I think it has been the practice to look upon the affection in rather too favourable a light, as regards the consequences which may ensue from it; and to take rather too unfavourable a view, as I shall endeavour to point out hereafter, of its remediability. There can be no doubt in the minds of those who have carefully studied the

disease, that, although in a great many cases it is very chronic in its character, yet, in some, it undergoes a more rapid development, and, when extensive, seriously compromises the safety of those who suffer from it, and tends to shorten the duration of life. As, however, with organic affections of the heart, patients may, with care, live on for a considerable period, so with emphysema; by avoiding those circumstances which may induce acute bronchitic attacks, or which tend to favour the development of the secondary consequences I have spoken of, danger may be averted, and life prolonged.

There is another circumstance which I must refer to here, viz., that emphysema is a more serious affection when it occurs in the adult, than in the aged. In the former, the requirements of respiration are much greater than in the latter; the circulation is more active; the quantity of blood in the system is larger, and nutrition is more vigorously carried on. Any interference with so important a function as that of the lungs will necessarily be followed with consequences of a more severe character, in the early or middle periods of life, than in old age. It is especially also in adults, as far as my observations go, that severe cases of bronchitis, such as I have described in a previous part of this chapter, occur, and which so frequently lead to a fatal result.

I have already spoken of the extravasation of air into the areolar tissue of the neck and trunk, in pulmonary emphysema. I have met with only one case

in which this accident occurred. The patient who was the subject of it was a young man, whom I saw with Mr. Swift, of this town, and who had suffered for some time from symptoms of phthisis. A day or two before my visit was made, a puffy swelling appeared about the neck, which soon extended. When I saw the patient, the emphysema had spread upwards, to the face, and downwards, over the chest and abdomen, as far as Poupart's ligament, on either side. Death ensued in a few days; and on making a *post mortem* examination, tubercular deposit was found in the lungs, with only a slight amount of emphysema. The areolar tissue about the root of one of the lungs, and in the mediastinum, was emphysematous, and served to show the source from which the air infiltrated beneath the skin had proceeded.

The relations of PHTHISIS to emphysema is the next subject to which I wish to refer. I shall not enter into any lengthened consideration of the question; but confine myself, for the most part, to a brief statement of the results of my own observations on the cases that have come before me. An impression has prevailed that emphysema and consumption are incompatible diseases; but I think the pathologists of the present day have abandoned this view, as contrary to the teachings of experience.

That emphysema and tubercular deposit are frequently found in the same lung, has been shown by the observations of numerous writers; and I may adduce the results of my own researches, as tending

to confirm the fact. In a large proportion of the *post mortem* examinations of phthisical cases that I have seen, since my attention has been directed to this subject, I have found emphysema present to a greater or less degree. M. Gallard, who has written an able paper on the question,* states that he also found a similar condition of the lungs in all the cases of phthisis which he examined during the time of his enquiry. The emphysema, he says, does not always exist on the surface of the lung, but is often found in the fissures between the lobes, and even in the interior of the organ. My investigations, with reference to this point, have not been carried out with the same degree of care as those of M. Gallard appear to have been; but I have found patches of emphysema along the borders of the lungs, and on their surface, in the cases to which I have alluded above.

I have no doubt that these patches of emphysema are produced in the manner I have described, in speaking of the determining causes of the disease, viz., by the effects of the cough, which is usually so prominent a feature in consumption; and that, in the vast majority of cases, the emphysema is a secondary consequence of the tubercular deposit. But an important question arises here, viz., whether tubercle is ever deposited in emphysematous lung-tissue. The fact that the two affections exist in the same lung, by no means answers the question in the affirmative.

* Mémoire sur l'Emphysème Pulmonaire, etc. Archives Générales de Médecine. Paris: 1854.

Rokitansky, in his *Pathological Anatomy*, remarks, that the venosity and cyanosis, which ensue in emphysema, from the changes that take place in the blood-vessels, etc., constitute the leading grounds for the immunity of emphysematous patients from tuberculosis.

Before this theory, that the condition of the blood in emphysema produces an immunity from consumption, can be admitted, the existence of such immunity must be proved. Although it may be true that, as a rule, tubercle is not often found in patients suffering from general emphysema, yet, that such patients enjoy no complete immunity from the deposit, is shown by the concurrent testimony of numerous investigators. I have seen several instances confirmatory of this view, where, for instance, both lungs were emphysematous—one entirely free from any symptom indicating morbid deposit, but the physical signs and general symptoms pointing to the existence of tubercular disease in the other. I believe that where general lobar emphysema exists, if there be a tubercular diathesis, the so-called venosity of the blood will not prevent the deposit of tubercle taking place. In a patient who died in the Northern Hospital, in June, 1860, I found one lung full of vomicæ; the opposite lung was large, pale, and had all the external appearances of an emphysematous lung. On examination, it was found studded throughout with semi-transparent tubercular matter. A small piece of the lung was carefully examined under the dissecting microscope; and it

was at once seen that the tubercular matter had been deposited in the air-sacs and ultimate bronchial tubes. By careful manipulation, portions of the tubercular matter could be drawn from the cavities they occupied, and perfect moulds of these cavities were thus obtained.

But although I cannot agree with the view that the venosity of the blood, in this disease, produces an immunity from tuberculosis, there can be no doubt that the condition of the blood-vessels of the lungs renders emphysematous patients, for the most part, exempt from certain other pulmonary affections, such as œdema, severe hæmorrhage, and pneumonia. It is very rare to see œdema of an emphysematous lung; and although a slight amount of blood may occasionally appear in the sputa of patients suffering from emphysema, yet anything like severe hæmorrhage never occurs. Further, I have in no instance seen an emphysematous lung the seat of pneumonic consolidation. In one instance where emphysema existed, and where death took place from pyæmia, I found a small abscess, with slight inflammation surrounding it, in the substance of one lung; but the remaining portion of the organ was free from anything like inflammatory action.

Inflammation of the pleura by no means unfrequently exists in connection with emphysema, as is proved after death by the presence of adhesions, sometimes of a very firm character. I believe the occurrence of pleurisy, in these cases, must be considered as quite an accidental circumstance, and that

it in nowise results from, or is produced by, the organic disease. In the most extensive cases of emphysema, no pleuritic adhesions are usually found. One result of these adhesions, when extensive, is to prevent the production of emphysema in the parts which lie immediately beneath them.

CHAPTER VI.

THE TREATMENT OF EMPHYSEMA, AND OF THE DISEASES CONNECTED WITH IT.

Treatment Considered under Two Heads: 1st. Of the Disease itself; 2nd. Of the Secondary Consequences. General Principles of Treatment. Remedies to be Used. Dietetic and Regiminal Treatment. Treatment of Bronchitis in its Acute Form. Cases. The Use of Turpentine in Severe Cases. Treatment of Chronic Bronchitis; of Asthma; of Cardiac Disease, Dropsy, etc.

The general treatment of emphysema requires to be referred to under two different heads. We have, *first*, to take into consideration the treatment of the disease itself, to examine whether the morbid condition of the lung-tissue admits of being checked in its progress, improved, or cured; and, *secondly*, we have to consider the treatment of the various secondary affections which follow as consequences of, or are associated with, the primary disease. And first, with regard to the treatment of the disease itself.

It cannot be doubted, I think, that this question has not sufficiently occupied the attention of physicians, and has not received the consideration it deserves. As I have previously remarked, numerous investigations have been made with reference to the morbid anatomy of the disease, and speculation has been

extremely rife as to its determining causes. But in regard to the question of treatment, the end and aim of all pathological enquiry, little appears to have been done; and the affection seems to have been looked upon as one for which but slight remedial aid could be given, except in so far as to relieve the secondary symptoms which follow in its train. It is true that Dr. Stokes and some other writers have expressed their opinion of the possibility of restoring the lung-tissue, in some measure, to a healthy state; but no definite line of practice has been laid down, by which this desirable result could be obtained. In fact, the therapeutical directions in our systematic works have been most meagre; and it is by no means improbable that the pathological views of the disease, entertained by most physicians, have tended to produce this result. The following quotation, from the work of Dr. Stokes, giving his opinion on the question, will probably also express the general view that has prevailed:—"To the practical physician, however, the great point of consideration is, that the disease of the lung is the result of bronchitis; and that for its prevention, alleviation, or cure if that were possible, the treatment must be conducted on this principle." It is true that this was written many years ago, and it is possible that the author of it may have modified his opinion in accordance with our more advanced pathological views; for, as I have already shown, there has been an increasing conviction of the existence of some morbid change in the lung-tissue, as a primary element in the worst forms of emphysema.

On the view we take of the pathology and determining causes of the disease, the whole question of its treatment hinges; for, if we look upon it as simply and solely the result of bronchitic affections, our efforts will be solely directed to the prevention of these attacks; and we shall be apt to consider, when our efforts have been directed to this end, that we have done all that is possible towards the alleviation or cure of the primary disease. This, in my opinion, would be to take a very narrow view of the question, and one neither warranted by clinical investigation nor pathological enquiry. If, however, we recognise the disease, in its worst forms, as depending on some degeneration of tissue, analogous in its nature to, although not identical in its character with, that which we know attacks other organs besides the lungs, we may hope, by the recognition of the principle, to be guided to a practice more appropriate and successful than that which a more contracted view of the nature of the disease would lead us to.

Morbid anatomy affords us a clear insight into the physical condition of the lung-tissue in emphysema; and we may fairly ask ourselves whether we are in possession of any means by the use of which we can hope to restore it to a healthy state. That, when the perforations in the walls of the air-sacs have taken place, and the walls themselves have become ruptured and torn, it is possible, by the use of any remedial measures we are now acquainted with, to restore the lung-tissue to a normal condition, we are not, in our present state of knowledge, warranted in

asserting. The perforations, once established, probably always remain; and the lung-tissue, once ruptured and torn, probably rarely undergoes, at the very best, but partial restoration. But whilst these facts are admitted, it is by no means implied that the disease is beyond our control. On the contrary, that condition of the lung which precedes the perforations, viz., the simple distension of the air-sacs, is, I believe, capable of great amelioration; and not only so; but even when the disease has proceeded beyond the stage of distension, although it is less amenable to treatment, yet a material improvement may in most cases be produced.

If the view I have taken of the pathology of emphysema is correct, and that the primary step in the disease is a morbid action, producing, in the first place, a loss of elasticity in the walls of the air-sacs, and, subsequently, their disorganisation; and if this morbid action is the result of disordered or imperfect nutrition, we may reasonably hope that, if we can improve the latter, the progress of the degeneration may be arrested, or checked, and possibly, even, that the elasticity of the lung-tissue may be restored. It is clear that this view involves an important point in therapeutics; and, if correct, will form an equally important basis on which to rest our general principles of treatment.

The main principles, in my opinion, which should regulate our treatment of emphysema—setting aside for the present the management of its secondary affections—are such as guide our practice in all

cases where we have to deal with, what we term, a degeneration of tissue; as, for instance, in Bright's disease of the kidneys, fatty degeneration of the heart, etc. We must consider the disease in the light of a constitutional affection, to be treated by constitutional remedies. All measures which tend to invigorate the system, to give tone to the walls of the heart, and to improve the condition of the blood, should be resorted to. In carrying out these indications, we must bear in mind the peculiarities of the malady we have to deal with, and the impossibility of applying, to patients suffering from it, the same matters of detail, in regard to treatment, as we can safely recommend to those who suffer from disease of the heart, or renal affection.

The indications to which I have referred, must be carried out both by medicinal and regiminal treatment; and first, with regard to the former.

Amongst the remedies for internal administration, which I have found the most useful in the disease, is iron. I was led to employ it from a knowledge of its value in those diseases which are attended with malnutrition and cachexia, and especially the two I have alluded to above, viz., Bright's disease of the kidneys, and fatty degeneration of the heart. I have employed it in a large number of cases, of which I have preserved notes, in dispensary, hospital, and private practice; and I have found its administration followed by so much benefit, that I consider it the most valuable medicinal agent we possess for the treatment of emphysema. It is necessary, as is the case in all

diseases of a degenerative character, that the use of the remedy should be continued for a considerable time — many weeks, or even months — if the full benefit is to be derived from it. It may be necessary to omit it occasionally for a short time, as symptoms may arise indicating its temporary disagreement with the patient; but no special directions are required on this head.

I am not aware that one preparation of iron is to be preferred to another; whatever form of the drug is found to agree best, should be persevered with. The preparations I have used most are — the ethereal tincture of the acetate of iron; the tincture of the sesquichloride; the sulphate; and the compound steel pill. The first of these forms — the ethereal tincture of the acetate — is a very valuable one, especially when bronchitis is present; as, from its stimulating properties, it acts as an expectorant. I have found it disagree with some patients, who were, however, able to take the sulphate, or some other form.

Small doses of quinine, combined with the iron, may be often given with apparently decided benefit. The quinine, no doubt, assists in improving the tone of the digestive organs — a matter of the greatest importance in the management of the disease. Mineral acids may also be given with a similar view. As will be seen further on, I have used iron somewhat largely, in cases of acute bronchitis occurring in emphysematous patients, at that period of the disease when the more urgent symptoms have passed off. Of the mode in which I have administered the

remedy under these circumstances, I shall speak hereafter.

I shall not refer, in detail, to the exhibition of remedies which may be called for to relieve any dyspeptic symptoms that may arise. Mild bitters may be sometimes required, and occasionally small doses of a mercurial, with compound rhubarb pill, or some other mild aperient. Dyspeptic symptoms should, however, rather be met by general management and diet, than by the administration of drugs. Practically, I for the most part confine myself to the medicines I have mentioned above, except in regard to the sparing use of expectorants, and remedies addressed to the bronchitic and asthmatic symptoms, from which the patients generally suffer more or less. The iron must not be given in large doses, nor yet very frequently; but in the same manner, and on the same principles, as we give it, and other tonics, in such diseases as I have expressed my opinion that emphysema is closely allied to. In some cases, where there has been a good deal of wasting, I have found cod-liver oil of service.

Strychnia has been recommended as a remedy for emphysema, with the view of improving the tone of the muscular fibres of the bronchial tubes. Dr. Walshe states that he has tried it in a small number of cases, both endermically and by the mouth, in sufficient doses to produce obvious effects on the voluntary muscles, without in the slightest degree modifying the symptoms of the disease.

The failure of strychnia is not at all to be wondered at; and the remedy must have been given under an entirely erroneous view of the pathology of emphysema. The affection is not one primarily affecting the bronchial tubes, but the lung-tissue itself; and if any secondary consequences ensue, with reference to the bronchial muscles, they are those of spasmodic contraction, rather than of paralysis.

The regulation of the diet, in emphysema, and the general management of the disease, are of as much importance as the medicinal treatment. On these points it will be sufficient to indicate a few principles, without entering into any minute details. The diet should be nourishing, and a moderate amount of stimulants should be allowed, depending more on the habits of the patient than any particular rule. The food should be easy of digestion, and of a dry, solid character—*i. e.*, possessing plenty of nutritious matter, in proportion to its bulk. Overloading of the stomach should be especially avoided, as well as everything which has a tendency to produce flatulence; for these conditions always give rise to dyspnœa, and often to palpitation of the heart. When we consider the effects of an overloaded stomach, or of distended bowels, in impeding the descent of the diaphragm, and thus interfering with the action of the lungs, and in embarrassing the movements of the heart—even when both these organs are in a normal state—we need be in nowise

surprised that more serious symptoms and greater distress should be produced by the same causes, when the respiratory surface of the lungs is diminished, and the heart's action already laboured in consequence of displacement. It is doubly important, in emphysema, that the nature of the food should be carefully looked to ; in order that, not only the primary digestion may be well performed, and no local cause may exist to impair the function of the lungs, but that the secondary process of assimilation may also be as perfect as possible, so as to ensure a supply of healthy blood for the nourishment of the lung-tissue. But further; an additional reason exists for the regulation of the diet, in the fact that emphysematous patients often suffer from dyspepsia.

An important principle to be borne in mind, in the treatment of emphysema, is to endeavour to keep the blood moderate in quantity, but rich in quality. It is obvious that, when the aerating power of the lungs is diminished, the smaller the quantity of blood sent to them, the more efficiently will it undergo its proper chemical changes, and the less will be the dyspnœa produced. It is with this further object in view that I have recommended that the food should not be of a bulky or watery character, but very nutritious, and as digestible as possible. In accordance with the same principle, it is desirable that the quantity of liquids taken should be small, in order that the blood-vessels may not be, even temporarily, overloaded with a watery fluid. As a drink, wine, or wine and water, is to be preferred to malt liquors,

which have a great tendency to produce flatulence, especially when the digestive organs are weak. Another principle of treatment to be looked to, is that of giving the lungs as little work as possible, and letting the patient breathe a pure air; so that the greatest amount of aeration of the blood may be produced, in proportion to the quantity of air inspired. Unfortunately, in the treatment of visceral disease, the physician cannot give the organ he has to deal with absolute rest from the performance of its function. Unlike external parts, which may be placed almost in a state of physiological rest, the internal organs, the lungs, the heart, the liver, etc., and more especially the two first, can know no period, however short, of absolute repose. But although this result cannot be fully accomplished, it must nevertheless be aimed at in many visceral diseases, and more especially in emphysema. All violent exercise, or great physical exertion of any kind, must be strictly prohibited. It is not that all exercise is to be proscribed; on the contrary, a moderate amount is to be encouraged, and this should be increased as we find the condition of the lungs improving. Exercise in the open air, moderate walking, carriage exercise, etc., form important adjuvants in the general treatment of emphysema.

In a very large proportion of the cases of emphysema which come under our notice for the first time, the symptoms of bronchitis, either acute, sub-acute, or chronic, are present. Most frequently the bron-

chial inflammation is of a chronic form; but occasionally it assumes symptoms of a very severe character, such as I have described in a previous chapter.

With reference to the general principles of treatment of all forms of bronchitis occurring in emphysema, I believe the greatest caution should be used in the administration of remedies which have at all a depressing character; and my experience does not lead me to coincide with an opinion expressed in some works on diseases of the chest, as to the great value of local bleeding, blistering, and tartar emetic, in the affections we are now considering; nor do I consider them as essential agents in their removal. I have never found it necessary to abstract blood in any way; and although blistering, or some form of counter-irritation, is often of the greatest service, I believe that few cases require, or are benefited by, the exhibition of tartar emetic. It is very possible that, in the earliest periods of an acute attack, when the bronchial mucous membrane is dry and swollen, a few doses of antimony may be given with advantage; but the period when this remedy is likely to be of service is soon gone by; and when once secretion, always profuse in these cases, has occurred, my belief is that tartar emetic is a remedy which ought not to be administered. Further, with reference to the treatment of the less acute forms of the affection, I have never found it necessary to administer antimony; and my opinion is, that all cases will be most satisfactorily managed without it;

and that, even in the use of ipecacuanha, we should be as cautious and sparing as possible.

I do not wish to enter, here, into a discussion of the general principles on which bronchitis should be treated; but simply to refer to the indications which should guide us in those cases where the inflammatory affection occurs in connection with emphysema; and the observations which I shall make must be considered as entirely referring to such cases. We must never forget that our patient is suffering from a depressing organic disease; and that he labours under a physical difficulty to get rid of the secretion which accumulates in the bronchial tubes. We must bear in mind that we have to guard against, and obviate, the tendency to death, first, from slow apnœa, and, secondly, from the deposit of fibrinous clots in the heart and great blood-vessels.

I have spoken of the dry condition of the bronchial tubes in the early stages of bronchitis, and the possibility that antimony may be usefully administered at that period; but the fact is, that, in consequence of the more or less chronic inflammation, which is almost invariably present in emphysematous patients, the bronchitic malady creeps insidiously on from the chronic, to the sub-acute, or acute, form; and it usually happens that, when we are called to these cases, we find the bronchial tubes more or less loaded with fluid.

What, then, is the general line of practice which ought to be adopted, in these bronchial affections? I believe it should be essentially of a stimulating and

sustaining character, and that all measures which have a tendency to depress the patient, or to diminish his expectorating power, should be avoided; whilst, on the other hand, those should be resorted to which tend to obviate death from asphyxia.

These indications will be most effectually carried out by the use of such medicines as ammonia, ether, squills, and in some instances ipecacuanha, etc.; and by the free, persistent, and regular administration of alcoholic stimulants — wine or brandy. There is no better mode of exhibiting the medicines I have mentioned, especially in the earlier stages of the disease, or when thirst or irritability of the stomach is present, than in combination with an effervescing saline. They may also be given, in a later stage, with decoction of senega, or any other preparation of a similar character.

The alcoholic stimulant must be given at regular intervals — every two, three, or four hours, according to the urgency of the symptoms; and we must not be deterred from administering it, although the skin be hot and the pulse rapid: in fact, as a general rule, the more rapid the pulse, the more freely stimulants should be given. After having used both wine and brandy, in these cases, and carefully watched their results, I have been led to prefer the former; and in fact, in all cases of so-called asthenic inflammation, I believe, as a rule, wine is to be preferred to brandy, or any other form of alcohol. It may, however, at times be necessary, from the urgency of the symptoms threatening asphyxia, to administer

the more powerful stimulant for a short time; and it may happen that the wine is not well borne by the stomach—in which case also brandy must be used. In one of the cases detailed below, this latter circumstance occurred; the patient rejected everything that was swallowed, until a few doses of brandy were given, which checked the vomiting at once, and after being continued for two days, wine and food were again borne.

In these acute cases, as soon as the symptoms have begun to improve, and whilst the secretion of the bronchial tubes is still profuse, I have seen great benefit follow the administration of iron, in frequently repeated doses; as, for instance, ten to fifteen or twenty minims of the tincture of the acetate, every four or six hours. Under this treatment I have seen the quantity of mucus expectorated rapidly diminish, and all the other symptoms improve. In sub-acute and chronic cases, we may begin very early with the use of iron; but, as I shall have hereafter to speak of it, in connection with such cases, I shall defer what I have to say of its action till then.

In addition to the measures which I have recommended, counter-irritation should be resorted to. Blisters are often highly beneficial, but in many cases I think some milder form of application, more extensively used, is to be preferred; and I know of nothing better than croton oil liniment, freely rubbed in all over the front of the chest. Turpentine fomentations also are of great service, especially in the early periods of the attack.

The diet should be carefully attended to; it should be of a digestible and nutritious character, and regularly administered. The bowels should be kept free from all accumulation; for this has a tendency to distress the breathing, and embarrass the action of the heart. A purgative, mercurial or otherwise, may be given in the first instance; but, for the most part, enemata should be used, in preference to aperients given by the mouth.

As illustrating the principles which I have endeavoured to lay down in the preceding remarks, for the treatment of acute bronchitis occurring in emphysematous patients, I shall quote the following cases, which I have selected from my hospital note-book, as being well-marked instances of their kind.

Both the patients referred to, at one time, had symptoms of so severe a character, that death appeared imminent; both were treated with similar remedies, and both made a satisfactory recovery. They were carefully watched by the students, the house surgeons, and myself, and notes of their progress were taken from day to day. Although these notes are somewhat lengthy, I prefer to give them without any alteration, and with all the circumstances of detail which were drawn up at the time.

Mary J., aged 20, single, was admitted into the Liverpool Northern Hospital on April 22nd, 1862. She said she had suffered for some time, but only to a slight extent, from cough and shortness of breath; she had never been laid up till about a week

before admission, when she was obliged to take to her bed, and had been attended by a surgeon from a dispensary.

On admission, the countenance was dusky; the eyes suffused; there was great general distress, with urgent dyspnœa. The pulse was 148, and very feeble; the respiration rapid. There was rather increased resonance all over both lungs, with resonance in the cardiac region. The impulse of the heart could not be felt. There was scarcely any lateral expansion of the chest on inspiration. The thorax was raised by the cervical muscles at each inspiration, and the lower part of the abdomen forcibly protruded. Coarse and subcrepitant râles were heard all over both lungs, with prolongation of the expiratory murmur. The tongue was furred; the skin hot; and there was great thirst. She was ordered an ounce of port wine every four hours, and one ounce of the following mixture every four hours:—Carbonate of ammonia, ʒ ss; spirit of chloric ether, ʒ ij; water, ℥ viij. For diet, strong beef-tea was ordered.

On the 23rd, she had had no sleep. The bowels had acted freely. The pulse was 144, the respirations were 48, per minute. She had expectorated a large quantity of frothy mucus. She was ordered to take an ounce of port wine every three hours, and to continue the mixture.

On the 24th, the pulse was 136; the respirations were 44. She had been purged several times. The chest-symptoms were somewhat relieved. The mixture was stopped on account of the purging, and the

wine increased to an ounce and a half every three hours.

On the 25th, she had slept well. The bowels were still rather relaxed, and the skin somewhat hot. The tongue was cleaner, and she expressed herself as better. The pulse was 148; the respirations were 48. There were still moist râles all over both lungs. She was ordered to continue the wine every three hours, and to take an ounce of the following mixture every four hours:—Solution of acetate of ammonia, ℥ iss; tincture of opium, ʒ ss; water, to ℥ vi. Croton oil liniment was ordered to be rubbed over the chest, and fifteen minims of Battley's solution to be given at night.

On the 26th, the pulse was 120; the respirations had fallen to 32. She had passed a fair night; the bowels had acted once; there was less expectoration. In the evening, she complained of feeling very sick; the administration of some brandy relieved her. She was ordered to continue the wine and mixture, and to have twenty minims of solution of morphia at bedtime.

On the 27th, the pulse was 120; the respirations were 40. She had slept well; the bowels were quiet; and the tongue clean. The sputa were frothy, and less abundant. The mixture was stopped. She was ordered to continue the wine, to have another application of the croton oil liniment, to take twenty minims of laudanum at bedtime, and to have some meat.

On the 28th, the pulse was 120; the chest-

symptoms were much relieved, but the expectoration was still copious. She was ordered to take ten minims of the ethereal tincture of the acetate of iron every four hours, and to continue the wine.

On the 29th, the pulse was 118; the breathing was better; she had slept well, and taken her food.

On May 1st, she was improving; coarse crepitation was still heard at the back of both lungs.

On the 2nd, the pulse was 100; the respirations were 32. The dose of tincture of iron was increased to fifteen minims every four hours, and some laudanum and squills were added to it.

On the 3rd, the pulse was 106, and feeble. There was less sputum, and it was more tenacious. The wine was reduced to eight ounces daily.

On the 6th, she was up. The pulse was 100, sitting. The tongue was clean, and the cough gone. There was very little expectoration. The appetite was good. She was ordered to take the iron mixture three times a day, and the wine was reduced to six ounces daily. From this date she rapidly improved.

On the 10th, the pulse was 68; the breath-sounds were free from râles, but there was a prolongation of the expiratory murmur, especially over the back of the lungs.

On the 15th, she was discharged.

Mary B., aged 19, was admitted into the Liverpool Northern Hospital on April 21st, 1862. She said she had suffered for five or six years from cough and difficulty of breathing, but had never been laid up.

She was as well as usual on April 17th, four days before admission. On the following day, she felt a headache, but did not take to her bed till the 20th. No treatment had been adopted.

On admission, the skin was hot and perspiring; the pulse was 128, and small. There was urgent dyspnœa. The lips were somewhat blue, and the eyes suffused. The breathing was superior thoracic, with but little or no action of the diaphragm.* The upper part of the chest was very prominent; and there was increased resonance over both lungs, with resonance of the cardiac region. Coarse and subcrepitant râles were heard all over the chest, with prolonged expiratory murmur.

She was ordered an ounce of port wine every four hours, and a mixture containing carbonate of ammonia and chloric ether; with strong beef-tea.

On the 22nd, she had passed a restless night, and had vomited both her food and wine. The pulse was 114; the respirations were 48. There was copious muco-purulent expectoration. The general symptoms were about the same as on the previous day. She was ordered to omit the wine and ammonia, and take a teaspoonful of brandy every half hour, till the sickness was stopped.

On the 23rd, she said she had not vomited since she began to take the brandy, of which she took four or five doses. She had rested better. The

* The movements of the chest, in the two cases I have quoted, serve to illustrate the different characters of the respiration referred to in the chapter on Physical Signs and Symptoms.

countenance was improved; the pulse was 118; respirations 48; the expectoration was still copious. The urine was loaded with lithates, and contained a small quantity of albumen. She was ordered half an ounce of brandy every three hours; and four ounces of port wine during the day.

On the 24th, she expressed herself as better, took her food, and had not been sick.

On the 25th, she had passed a good night; the sputum was still abundant, but more frothy; the pulse was 124; the respirations were 48. She was ordered eight ounces of port wine daily, and an effervescing saline with carbonate of ammonia.

On the 26th, the urine was examined, and found free from albumen.

On the 27th, croton oil liniment was ordered for the chest.

On the 28th, she was much improved. The pulse was 100; respirations 40. She had taken her wine and ammonia regularly. There was but little dyspnœa; and the râles over the lungs were coarser. The expectoration was still copious. She was ordered to omit the ammonia, but to continue the wine, and to take ten minims of the ethereal tincture of the acetate of iron every four hours.

On May 1st, the sputum was less copious and more tenacious. The pulse was 100; the respirations were 32; and the appetite was good.

On the 2nd, the dose of tincture of iron was increased to fifteen minims every four hours.

On the 3rd, the pulse was 90; respirations 25.

The tongue was clean, and the expectoration much diminished. She complained of the cough giving her some trouble. She was ordered twenty minims of tincture of iron and ten of laudanum three times a day, and meat diet. The wine was reduced to six ounces daily.

She rapidly recovered from this date, but was kept in the house for some time to be treated for the emphysema. She took a course of iron, with good diet; and under this treatment she gained flesh, and her cough and dyspnœa materially improved.

She left the hospital on June 9th.

The cases I have just detailed serve to show the beneficial effects that may be produced, on certain severe forms of bronchitis, by the free and persistent use of alcoholic and other stimulants, etc. I do not wish, at the present time, to enter into any general observations on the therapeutic value or mode of action of alcohol, and I shall therefore content myself with remarking that, in watching the results of the remedy in cases similar to those I have described, and, in fact, in all so-called asthenic inflammations, there is nothing more striking than the effects it produces on the circulation; for we find the pulse gradually diminishing in frequency and increasing in power—a sure sign that the patient is benefiting from the treatment adopted, and that the disease is taking a favourable course. In the management of all inflammatory diseases, the character of the pulse constitutes one of

the most important guides as to the propriety of administering stimulants or withholding them; and it cannot, I think, be doubted that, if our knowledge of the indications afforded by the pulse were more accurate and defined, our treatment of these acute affections would be characterised by more uniformity and a greater measure of success.

It occasionally happens that these cases of bronchitis, either from neglect or extreme severity, assume a character which I have described in a former chapter, when the surface of the body becomes cold, and the pulse exceedingly small and feeble; and when, from the accumulation of fluid in the bronchial tubes, and the inability of the patient to expectorate, asphyxia is threatened. My experience of the treatment of cases which have reached this degree of severity is, that the ordinary stimulants, alcohol, ether, ammonia, etc., are but of little avail to rally the patients from their semi-asphyxiated condition. I have given brandy and other stimulants, in moderate and in large doses, at frequent intervals, and apparently without the least benefit being produced. On making a *post-mortem* examination of cases of this kind which have terminated fatally, I have found, as I have previously stated, not only the bronchial tubes loaded with fluid, but the cavities of the heart and of the great blood-vessels connected therewith more or less occupied with fibrinous clots. Although, in all probability, when cases assume the severity I have described, a large per-centage of them will prove fatal, and although

we can scarcely expect that medical art can effect much for their relief, yet there is one mode of treatment which I have found succeed, when alcoholic and other stimulants have failed, and which I therefore think it worth while briefly to allude to.

The practice I refer to consists in the administration of moderately large doses of turpentine, on a plan suggested, I believe, by Dr. Corrigan, of Dublin. This medicine has often been recommended in diseases affecting mucous membranes, and in some cases of hæmorrhage its value is unquestionable; but I am not aware that it has been much used by practitioners in such cases as I am referring to.

I will briefly relate a case in which this treatment was adopted, and in which it produced the most satisfactory results.

Peter C———, 22 years of age, a sailor, was admitted into the Liverpool Northern Hospital on the 22nd December, 1860, with acute general bronchitis and emphysema. When first seen by me, about noon of the day of admission, he was suffering from all the symptoms I have elsewhere described, and appeared to be rapidly sinking into a condition of asphyxia. Alcoholic and ethereal stimulants had been already given, and were freely continued for several hours. Towards the evening, about four or five o'clock, I saw him again. The symptoms had not at all improved, but were rather worse than better. He was ordered dram doses of turpentine in aromatic water every two hours. The symptoms

began immediately to improve, the dyspnœa became less, and the skin warmer and of a more healthy hue. The following day, as the turpentine had produced a little strangury, it was omitted. The patient at once began to relapse, and the symptoms soon became as threatening as ever. A half-ounce dose of turpentine was ordered. Marked relief to the lung symptoms followed its administration; but, as soon as its effects had passed off, viz., in about four or five hours, a relapse ensued, and another half-ounce dose of turpentine was given, and followed up by others. In this way five or six half-ounce doses of the medicine were taken. The result of each dose was, that great relief was given for a time; but, as the effects of the turpentine passed off, the bad symptoms returned, to be again relieved by a further dose. After each dose, the patient appeared stronger, and the interval before the subsequent one was given was prolonged. At the end of five or six days from his admission, no further relapse took place, and the turpentine was stopped. During the time he took the turpentine, he was also taking alcoholic stimulants; these, no doubt, acted beneficially, but the oil seemed to be the great remedial agent.

This case was very carefully looked after by the house surgeon, who administered the turpentine, under my directions, according as it was required; and I could not help thinking at the time, and I am still of the same opinion, that the man owed his life to the assiduity with which he was watched, and the repeated exhibition, at the right time, of the remedy

which was found to act so powerfully on him. He suffered rather severely from strangury during the time he was taking the oil; but this symptom subsided as soon as the medicine was discontinued.

The following were the results which followed the administration of the turpentine, and which were apparently produced by it. The expectorating power of the patient was increased, the dyspnœa was relieved, and the effusion into the bronchial tubes was diminished. After the turpentine was left off, and the urgency of the symptoms had subsided, ethereal tincture of the acetate of iron was given, and continued for some weeks. Under this treatment a steady improvement took place, and the patient was discharged six weeks after his admission, his general appearance being then much improved, his dyspnœa lessened, and his power of taking exercise far greater than it had been, he said, for a long time.

It will be observed that, in quoting the above case, I have not spoken positively as to the number of doses of turpentine the patient took, nor as to the number of days he was under this treatment. I have mentioned five or six. The fact is, that the early notes of the case are lost; and although all the statements are in the main correct, yet, in the above matters of detail, I have been unable to speak in that positive manner which is always desirable when cases are quoted. This does not, however, affect the argument as to the value of the remedy made use of.

I cannot strengthen the case by adducing others to support it; I give it, therefore, as an isolated one, for simply what it is worth. The results of the treatment were so satisfactory, that I am of opinion that, in these extreme cases, when other measures fail, we might fairly have recourse to it. Caution is, however, necessary, in the use of the remedy; it must not be given in large doses at first, or it may produce great depression. The best plan is to begin with half dram, or dram, doses, every two or three hours; and then, if necessary, to give the larger dose less frequently.

In a case very similar in symptoms to the one just quoted, a half-ounce dose of the oil was given at first; it produced some sickness, but had no effect in rallying the patient from his semi-asphyxiated condition, and he subsequently sank. After death, both lungs were found highly emphysematous throughout, and firm fibrinous clots existed in the cavities of the heart, as well as in the pulmonary artery and aorta; a condition probably precluding all chances of recovery.

The administration of an ipecacuan emetic in bronchitis, for the purpose of relieving the overloaded bronchial tubes, is sometimes productive of great benefit, and might be of service in those cases of threatened asphyxia which I have alluded to. Not having had any practical experience of it under these circumstances, I cannot speak as to its value.

I will now briefly refer to the treatment of the

chronic form of bronchitis, which is so constantly associated with emphysema; and as we very frequently find asthmatic symptoms co-existent with the bronchial inflammation, and are often called upon to treat the two affections simultaneously, it will be well to consider the measures which are appropriate for their relief at the same time. As, however, I do not wish to enter into the general question of the treatment of asthma, my remarks on this head will be very brief.

With regard to the bronchitic symptoms much good can be effected by counter-irritants and expectorants, such as squills, etc. I believe one of the best external applications we can use is croton oil liniment, which should be rubbed over the chest from time to time, as circumstances may require, and it will be rarely found that, as soon as the eruption has appeared, relief does not immediately follow.

In the treatment of these cases, we must bear in mind that, in almost all emphysematous patients, there is, at all times, more or less congestion or inflammation of the bronchial mucous membrane, and that this is always attended with increased secretion. Our object must be, not simply to adopt such treatment as will tend to relieve a present inflammation, and enable the patient to get rid of the excess of mucus which troubles him; but we must further endeavour to act on the relaxed mucous membrane, and give tone to its capillary vessels, so as to obviate the condition which causes these constantly recurring attacks. Although, primarily,

expectorants are called for, yet it is desirable to disturb the stomach as little as possible with nauseating drugs, the more especially if the digestive organs are weak. The regimen and treatment should be of a decidedly tonic character, and carried out on the principles which I have recommended in speaking of the general treatment of emphysema.

I have already referred to cases of bronchitis in which I have administered iron, when the more acute symptoms had passed off, and I have spoken of its value in emphysema itself. Independently of the influence which the remedy seems to have on the pulmonary tissue, I believe it produces a most beneficial effect on the congested and relaxed bronchial tubes in sub-acute and chronic bronchitis. In the cases above referred to it was given somewhat largely when the more acute symptoms had subsided. In other cases, of a sub-acute or chronic form, I have been in the habit of giving it, in doses of from ten to fifteen minims of the ethereal tincture, or the tincture of the sesquichloride, three times a-day; and, from having very frequently administered it, and seen the rapid manner in which profuse secretion from the bronchial tubes has been checked, and the other symptoms relieved, it has appeared to me to be the best medicine we can give in such cases, with the view of bringing about the secondary result which I have adverted to above. It may be combined with expectorants, or given with wine, or with a bitter tonic, when these are indicated. Besides

its general effect, it appears to act as a local astringent on the bronchial mucous membrane, and to possess, in suitable cases, advantages over all other remedies of a similar character we are acquainted with.

For the relief of asthmatic symptoms occurring in emphysema, I have generally found most benefit from the administration of the ethers, ethereal tincture of lobelia, and especially from the smoking of stramonium and tobacco. I generally recommend patients to smoke a pipe or two of stramonium just before going to bed, and this I have very constantly found to have the effect of giving them a good night, and preventing them being disturbed with an attack of dyspnœa. Tobacco I have found very useful in some cases, and especially with patients who were not previously in the habit of smoking it; but, as far as my experience goes, it loses its influence over the affection sooner than the stramonium does.

If the measures I have referred to should fail to produce relief, which I think will be rarely the case in asthma depending on emphysema, other remedies which have been at various times found useful in the affection may be resorted to.

It forms no part of my present purpose to consider any special treatment which may be required for the cardiac disease, dropsy, etc., which follow as secondary consequences of emphysema. If the general principles I have referred to for the treatment of

the primary disease are carried out, I believe the best means will be adopted to prevent both that state of the heart which is followed by dropsical effusions, and that condition of the system at large which gives rise to anæmia and atrophy.

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From the "Medical Times and Gazette."

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clear marks of the author's own personal investigations and dissections, in this still somewhat obscure field of anatomy. As he truly observes, our language has been hitherto singularly deficient in treatises on the anatomy of the lungs, and no doubt this fact is in chief part to be attributed to the very difficult nature of the investigation of the structure of these organs. In the first part of his work, Dr. Waters gives us a very interesting sketch of the historial part of this subject, bringing us down from the times of Hippocrates to our own; and as a *résumé* of what had been previously done by different observers in advancing our knowledge of the anatomy of the lungs, this chapter is very valuable. The author then proceeds to give a general and particular description of the anatomy of the respiratory organs, and, as we have already said, we meet distinct proofs of his having seen and touched all these things himself, and of his not trusting to the dicta of his predecessors. The most interesting part of the work is that which treats of the ultimate structure of the lungs. What there is of novelty in his researches, however, has already been given to the readers of this journal, in a full summary of a paper which was last year read by him to the Royal Society. It will be found in this journal for July 16, 1859. We will, therefore, only say of this account of the ultimate pulmonary tissue, that it is the most complete of any given in the English language, and has the advantage of being liberally illustrated by those necessary helps to learning — well executed microscopic cuts. These microscopic views of the lung-tissue, it is also fair to observe, have a merit which is somewhat rare at the present moment — they are many of them *original*. Every one who is much accustomed to peruse the anatomical, physiological, and pathological works of the day, must have been often struck by the repetition of the same microscopic views, copied and passed on from book to book, which so continually meet his eye; and in this particular case of the lungs there is no great exception to be found to the fact. These novel representations, copied from nature, are truly refreshing to look at. We often wonder that those who thus pass on microscopic views from hand to hand are not a little doubtful as to the propriety, lest they be, in fact, propagating erroneous views. For these reasons we give especial credit to Dr. Waters for his carefully revised specimens of views of the lung-tissue. Dr. Waters gives, at the end of his work, a summary of his conclusions regarding the structure of the lungs, and we cannot do better, in concluding our notice of this excellent Essay, than give his views thus summed up."

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