

**On winter cough, catarrh, bronchitis, emphysema, asthma : a course of lectures delivered at the Royal Hospital for Diseases of the Chest / by Horace Dobell.**

### **Contributors**

Dobell, Horace, 1828-1917.  
Royal College of Physicians of Edinburgh

### **Publication/Creation**

London : J. Churchill, 1866.

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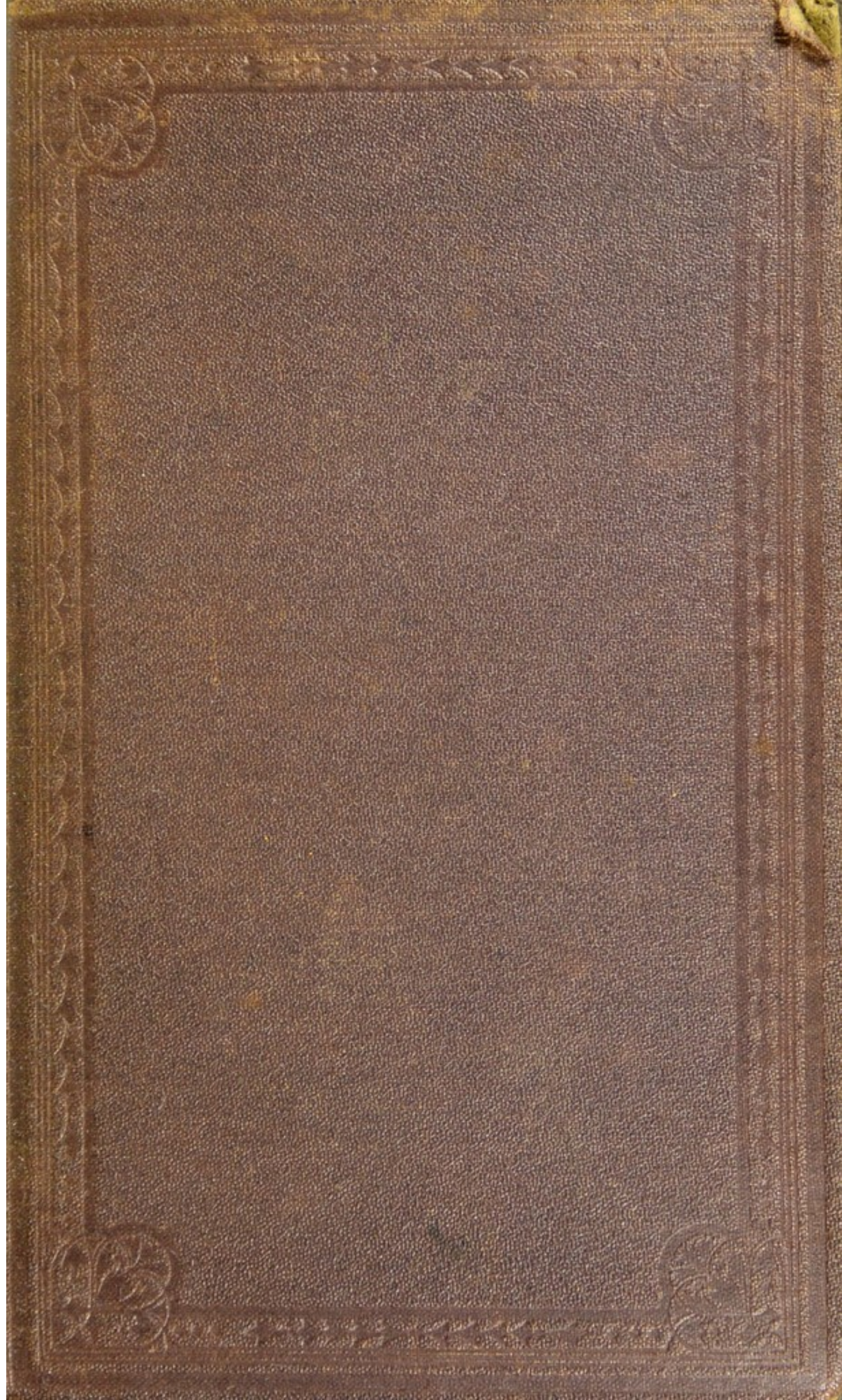
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ON

# WINTER COUGH,

CATARRH, BRONCHITIS, EMPHYSEMA, ASTHMA,

WITH

AN APPENDIX ON SOME PRINCIPLES OF  
DIET IN DISEASE,

A COURSE OF LECTURES DELIVERED AT THE ROYAL INFIRMARY  
FOR DISEASES OF THE CHEST.

BY

HORACE DOBELL, M.D.,

PHYSICIAN TO THE INFIRMARY,  
ETC., ETC.

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## P R E F A C E.

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I THINK it must be admitted that those who have trained their minds to observe correctly and to form conclusions cautiously, cannot pass through a large experience without acquiring wisdom in matters which relate to that experience; and yet, that such persons, if called upon to prove that their wisdom is the result of sound conclusions, based upon a sufficient number of unquestionable facts, may be quite unable to do so to the satisfaction of others. The facts which have been carefully stored up in the mind, like the centring of a builder's arch, until the conclusions which they support have become ripe, are then allowed to slip away one by one from the memory, and others are accumulated as the bases of fresh conclusions, to disappear again in their turn. The wisdom which remains as their fruition is like the seeds gathered together in a granary; it bears no proof of how or whence it came, until some sower has again gone through the process of rearing fresh plants to maturity and winnowing out fresh seed.

This, indeed, is that unwritten wisdom which is the



peculiar property of the sage. That it cannot be transferred or transmitted to others is, perhaps, the chief cause of the slow advance of intellectual progress from age to age. The tendency of the world has been to disregard authority in proportion as the means and facilities of recording facts have multiplied; so that, in the present day, the wisdom of the sage, if unsupported by the record of his experience, is scarcely valued at the price of the hasty conclusions of a novice, who may have picked out and recorded a few facts by which they seem to be supported. The danger of this tendency is, that society may drift into the disbelief of everything; for, if we first disbelieve in every conclusion which is not supported by recorded facts, that is to say, if we disbelieve in the opinions of wise men unless the whole course by which they arrived at those opinions be demonstrated, the next step is to doubt the correctness of all recorded facts, however good the authority of the recorder; and thus, when the demonstration has been given, to doubt its correctness still.

Nevertheless, there is wisdom in this incredulity, if kept within proper bounds. Though it may apparently retard the progress of truth, if the steps that we are permitted to take are made more secure, by being taken slowly, our advancement will be quicker in the end. It is, then,



our unquestionable duty neither to disregard the opinions of wise men, nor to neglect, so far as we are able, to record our own observations as we go along; so that our own conclusions and our future wisdom may have less danger of meeting with disbelief, and of thus proving useless to the world. The great difficulty in this busy life is for men, like ourselves, who have to earn their living by their professional labours, to find the time to do that which they well know to be best, and which they most desire; for, in proportion as our opportunities of observation are great, our opportunities of recording them with care diminish; and so, I fear, it must always happen that much of the wisdom of our greatest physicians will die when they die.

I do not for a moment presume to possess the wisdom of which I have spoken; but the foregoing remarks are applicable to these lectures to this extent—that the number of cases which I have here recorded, and which, therefore, appear as the bases of my conclusions, is really absurdly small as compared with the number of similar cases which, during many years' experience at the Royal Infirmary, and in private practice, have been quite as carefully observed, and have only failed to be recorded because all the available time was spent in examining them with care.



I must, however, point out one important fact in relation to the cases here cited, viz., that they were taken with the utmost fairness as to the conclusions they might justify, no one point having been set before the mind at starting on which to acquire proof or disproof. A simple and open enquiry was pushed into the facts of the cases, to yield what results it might. My conclusions have been forced upon me as the unavoidable results of this enquiry ; but, as all probable evidence is subject to the contingency of having a variable weight in different minds, what has seemed most clearly proved to me may to others appear in quite a different light.

41, HARLEY STREET, W.,

*January, 1866.*

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All Cases not included in the Tables are marked with an asterisk.

The COLOURS are only used as distinguishing marks of the several groups, to avoid the needless repetition of definitions in the Tables.

As all the salient points of the Cases are contained in the Tables and abstracts given in the Lectures, the further details have been omitted; because they would have added twenty-five pages of tabular matter to the Work without equivalent advantage.

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GENERAL DESCRIPTION OF THE FIVE CLINICAL GROUPS REFERRED TO PAGE 4, WITH THE NUMBERS OF THE CASES INCLUDED IN EACH GROUP.

---

GROUP I.—YELLOW.—*Physical signs of Emphysema and not of Bronchitis :  
no history of previous Bronchitis.*

1 Case—No. 20.

GROUP II.—BLUE.—*Physical signs of Emphysema and not of Bronchitis :  
history of previous Bronchitis.*

6 Cases—Nos. 1, 7, 19, 48, 53, 57. (At p. 27, for Case 77, read 57).

GROUP III.—RED.—*Physical signs of Bronchitis, and not of Emphysema.*

18 Cases—Nos. 3, 10, 12, 13, 16, 18, 21, 25, 29, 32, 36, 37, 39, 43, 45,  
49, 54, 60.

GROUP IV.—WHITE.—*Physical signs of both Bronchitis and Emphysema.*

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GROUP V.—(Not included in the Tables).—*Exceptional Cases.*—No physical signs either of *Bronchitis* or of *Emphysema*.

3 Cases—Nos. 80,\* 81,\* 82.\*

ISOLATED CASES *not included in the above groups.*

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# ON WINTER COUGH.

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## LECTURE I.

INTRODUCTION — DIVISION OF CASES INTO FIVE CLINICAL GROUPS —  
RELATIONS BETWEEN EMPHYSEMA, BRONCHITIS, AND WINTER  
COUGH — HOW IS EMPHYSEMA PRODUCED ?

I THINK you will agree with me, that when once a medical man has come before the public to *practise* his profession, the first duty incumbent upon him is to treat disease as successfully as possible.

However much his tastes may tempt him to devote his time and energies to the purely scientific departments of his profession, he must never lose sight of the fact that he is neglecting his duty, in proportion as he allows himself to be led away from such studies as have a practical bearing upon the prevention, relief, and cure of disease. There ought to be no alternative in his mind, between giving up practice altogether, and devoting all his best energies to making his practice beneficial to his patients in the highest possible degree. I make these remarks in explanation of my having, in these lectures, passed by many points exceedingly interesting as subjects of scientific speculation



and enquiry, in order that I might devote all the time at our disposal to those which come more strictly within the limits of practical medicine.

Again, I can but think that, as practitioners of medicine, we ought to look with far more interest upon anything that conduces to the cure or relief of the diseases which affect the largest number of persons, than any rare and solitary cases, however curious they may be. I have not hesitated, therefore, to devote a large amount of time and study to the very common and well-known class of cases which form the subject of these lectures.

I have included them all under the crude name of "*Winter Cough*," because it expresses the one conspicuous symptom, common to them all, which especially brings such cases under the eye of the physician. All the patients had a cough, which was either limited to the winter season, or was much aggravated during that part of the year.

However tedious and wanting in the excitement of novelty a common case of winter cough may be to the medical practitioner, there are few complaints which so painfully absorb the interest and attention of the patient; and as such cases are extraordinarily numerous in all ranks of society in this climate, they represent an enormous amount of human suffering, and from this fact alone demand our most earnest consideration.

I need hardly tell you that at this hospital such cases abound in every form and variety, and afford the widest possible field for enquiry, whether it be into their symptoms and physical signs, their course and treatment, their consequences and terminations, or their causes and natural history.



As two or more winters usually pass before the tendency of the complaint to recur or to become habitual is established in a patient's mind, these cases of winter cough have always a history more or less long, and of which it is often not very easy to get a correct account. But in this history lie just those points of the case which are essential to a proper understanding of the causation of the disease, and of the prospects of cure or relief. It is into this history, therefore, that we should always push our enquiries with great perseverance, taking the utmost care not to be misled by the erroneous and conflicting statements which patients are sure to make, unless we give them time to think over the past, before committing themselves to an account of it.

In order to guard as much as possible against this source of fallacy, and at the same time to secure uniformity in a large number of reports, so as to admit of their chief points being tabulated, I have been accustomed to give to patients a printed list of questions to think over at their leisure, before committing themselves to the answers.

These questions are forty-one in number, and refer to the short breath, the cough, the taking of colds, the past illnesses, the occupation, dwelling, habits, and the family history of the patient.\*

In collating the notes of a large number of cases, the histories of which are taken in this manner, and to which the physical signs and symptoms are attached, I have found that they may be very simply arranged in five clinical groups.

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\* For form of report see Appendix.



1. Cases in which there are physical signs of Emphysema, and not of Bronchitis, and in which there is no history of previous Bronchitis.
2. Cases in which there are physical signs of Emphysema, and not of Bronchitis, but in which there is a history of previous Bronchitis.
3. Cases in which there are physical signs of Bronchitis, and not of Emphysema
4. Cases in which there are physical signs both of Emphysema and of Bronchitis.
5. Exceptional cases, in which there are not physical signs, either of Bronchitis or of Emphysema.

You will at once observe, that these groups give us Emphysema and Bronchitis as the two conditions of disease which, with the exception of a few cases, are ever present, either singly or combined, when there is Winter Cough.

It is evidently, therefore, the leading point of interest, in a practical sense, to know what is the relation of these states—Emphysema and Bronchitis—to each other, and to the complaint in question, viz., Winter Cough.

Is the cough produced by Emphysema, by Bronchitis, or by both, or is it dependent upon some other condition which accompanies both the Bronchitis and the Emphysema?

Is the Bronchitis produced by Emphysema, or Emphysema by Bronchitis?

Is the Bronchitis or the Emphysema produced by the Cough?

In what way, if any, do Bronchitis and Emphysema influence each other?



These are questions which lie at the bottom of our treatment of the disease, whether it be preventive, curative, or alleviative, and I am sorry to say that they cannot all be answered so simply as might at first sight appear, for some of them have already engaged the best attention of excellent pathologists and practical physicians, and the conclusions they have respectively come to have been very different.

As my own views on the subject are in opposition to those of some of the physicians for whose opinions both the profession generally and myself entertain the greatest respect, I cannot put them forward without, to some extent, attempting to support them by arguments; and yet I consider that a right understanding of this part of the subject is so essential to the whole question of the treatment of Winter Cough, that I cannot pass it by. I will, however, attempt to put these views before you as briefly as a due respect for the opinions of others will permit.

The great question of dispute relates to the mode of production of Emphysema.

How is Emphysema produced?

1. Is it due to the forcible expansion of the air vesicles of the lungs during the inspiratory act?
2. Is it due to a compensatory dilatation of the air vesicles rendered necessary by the collapse of neighbouring portions of lung?
3. Is it due to a degeneration of the tissue of the air vesicles, which renders them incompetent to withstand the normal dilating influences during normal respiration?
4. Is it due to the forcible expansion of the air vesicles of the lung during the expiratory act?



You are aware that there are powerful and accomplished advocates for each of these four propositions, and when I assert, as my strong conviction, that the fourth alone can be maintained—that Emphysema can be more satisfactorily explained by the expansion of the air vesicles during expiration, than by any other cause, and that this belief is essential to the proper treatment of Winter Cough—I feel that I am bound in some measure to support the opinion I hold, by arguments which may commend it to your respect, although I cannot venture to occupy your time with such a detailed and abstruse discussion as would be necessary to answer all objections urged, on the various sides of the question.

The first thing to do is to show that the phenomena of Emphysema can be satisfactorily explained by the expiratory theory of its production.

The second is to show that the conditions required by this theory, for the production of these phenomena, are supplied by disease.

And the third is to show that evidences of the existence of these conditions are to be found in the clinical histories and features of cases of Emphysema.

Having done these things, if time allows, I will briefly point out some of the vital objections to the other theories to which I have referred.

Emphysema of the lung, as you well know, may be limited to a few air cells, or may affect one or more lobules, a whole lobe, and even the whole of one or both lungs. It essentially consists in a dilatation of the air cells, which may be slight or extreme, and in attenuation and rupture of the dilated walls of the cells, so that contiguous cells are made to communicate with each other,



and in the further progress of the disease the partitions between the cells almost entirely disappear, and large irregular air sacs are formed by the coalescence of neighbouring cells. In this way the bulk of the affected lung is increased, and its normal elasticity is lost.

You are probably aware that a state resembling Emphysema may be produced in a healthy lung after death by forcibly blowing into the air tubes, and thus over-distending the vesicular structure. Dr. John Hutchinson found that in pumping air after death into the human chest, a pressure of nearly 12 ozs. avoirdupois upon every square inch of surface was sufficient to rupture the pulmonary substance. There can be no doubt that during life the air cells will admit of much greater distension before rupture of their walls occurs than after death, especially if the distending force is applied very gradually. But in essential characters the distension and rupture which occur when the lungs are forcibly distended after death, are analogous to that which occurs during life in the production of Emphysema.

I am aware that this experiment has been supposed to favour the inspiratory theory of the production of Emphysema, but a moment's thought will teach us that it is clearly no illustration of an inspiratory operation. Inspiration is distinctly a suction force exerted from without, in which the air simply follows the expansion of the chest walls, and of the lung, and is accompanied by no *vis a tergo*; whereas, in the experiment quoted, the lung is distended entirely by means of a force exerted from behind.

We see, then, from this experiment, that all that is necessary to the production of Emphysema is an undue



pressure of air upon the internal surface of the air cells.

You are no doubt familiar with the fact, that what is called "Interlobular Emphysema" may be produced by forcible expiratory acts. That during such acts one or more air cells may become so over-distended, that rupture takes place, and the air from the lung is poured into the cellular tissue between the lobules, whence it may find its way into the cellular tissue of the mediastinum, and thence into that of the chest and neck. I well remember a case of this kind which was brought to my notice by Dr. Niell, of Aldersgate-street. He was attending a lady in a difficult confinement, and suddenly, during a violent expulsive effort, the patient's neck and shoulders swelled up to an enormous size, and the cellular tissue was found to be full of air. Many such cases have been witnessed, always occurring during some violent expiratory effort, as during parturition, defæcation, lifting heavy weights, or coughing. A number of cases, in which this accident occurred during fits of coughing, are recorded by M. Guillot (*Archives generales de Médecine*, vol. 2, 1853), and many others, may be found scattered through other works and periodicals.

From these two facts, then,—(1) that distension and rupture of the air cells, similar to Emphysema, have been produced experimentally by undue pressure of air applied to the inner surface of the cells, and (2) that similar distension and rupture of the air cells have been produced accidentally by forcible acts of expiration, it may be concluded that vesicular Emphysema may be produced by pressure applied to the inner surface of the air cells during expiration.



Now, although by those who only approve of complicated modes of proof in scientific matters, this may appear a somewhat short and off-hand way of deciding a difficult question, I think you will find it unanswerable, and that is the important point to look to. If we have once seen unmistakably that a thing can be done, and has been done, we need not trouble ourselves much with arguments which attempt to prove theoretically that it cannot be done. And here we have two very simple illustrations, the one showing that it can be done, and the other that it has been done, and therefore, to my mind, the question is so far settled.

The next portions of the proof required of us, viz., to show that the conditions necessary to the expiratory theory are supplied by disease, and that evidences of the existence of these conditions are to be discovered in the clinical histories and features of cases of Emphysema, cannot be disposed of in so short and off-hand a way. It is beset with all those difficulties which must ever surround questions which deal with complicated vital acts conducted within the body; and in this case an additional difficulty, and a fertile source of error, is introduced by the circumstance that considerable periods of time are in most cases occupied in the development of the effects we have to deal with, and by the circumstance that the cause may have ceased to exist at the time the effects are brought under our observation. We have, therefore, to search in one individual for the cause of effects which we witness in another; a mode of inquiry than which nothing can be more puzzling and open to fallacy. And when the effect is discovered, and the cause found wanting, it is at once competent, for those who believe that the



assigned cause is not the true one, to bring these cases forward as proofs of the correctness of their opinions. Our great endeavour, therefore, should be to find a mode of observation which shall, as far as possible, remove this source of fallacy.

It has appeared to me that this source of fallacy must remain so long as the post-mortem examination of the patient is depended upon for the explanation of the phenomena of his disease. I am speaking especially of the disease now under our consideration, although the remark may apply to others. A simple illustration of what I mean is this:—The examination of the lungs of a man who has long suffered from attacks of Spasmodic Asthma may show well-developed Emphysema. But not a trace may remain of the bronchial spasm which produced the Emphysema; whereas, on the other hand, there may be found many other changes in his bronchial tubes, or in the parenchyma of the lung, due to causes totally unconnected with either the Asthmatic Spasm, or the Emphysematous air cells, while the history of the case may be totally incompetent to determine whether or not any relation existed during life between the pathological changes discovered after death. On the other hand, there were, in all probability, periods in the course of the case when symptoms and physical signs, properly interpreted, might have told what morbid causes were in action, and what organic changes were being produced by them, the course of both being watched and traced.

In making these remarks, I wish particularly not to be misunderstood. I do not the least undervalue the great importance of morbid anatomy, when the conclusions drawn from it are kept within legitimate limits.



In the next place, I have to show what are the physical conditions required in the chest to produce Emphysema by expiratory acts, and what clinical means we possess of ascertaining that such conditions exist in any given case.

In the operation which we commonly call "straining," we first take a deep inspiration, by which the thorax is distended, and the lungs filled with air, then close the glottis, so as to keep the air locked into the chest, and with the thorax thus tightly distended, we put the abdominal and other expiratory muscles into forcible contraction.

If you were to inflate a small bladder, and then grasp it forcibly in the hand, you would not be much surprised if the bladder were to burst at any point where its walls happened to be weakest and least supported by the hand. We cannot, then, be surprised to find, that, when the distended lungs and chest-walls are forcibly pressed on all sides by powerful muscles, while the escape of the air by the glottis is prevented, they occasionally give way at some of their weakest points. This happens occasionally to the chest-walls themselves, so that portions of lung are actually protruded through them, constituting thoracic hernia. But it more commonly happens that the delicate air vesicles burst at some part of the lung, where the external pressure happens for the moment to be the least, and give rise to inter-lobular Emphysema (see p. 8).

It is, then, very clear, that during these efforts of straining, when, after inspiration, an obstacle is put in the way of expiration, and at the same time muscular pressure is brought to bear upon the outside of the chest, air is driven with force against the inner walls of the air cells;



that, in fact, so far as the air cells are concerned, the operation is similar to the experiment of blowing into the lungs down the bronchial tubes. And as we might reasonably expect the effects of two similar causes are alike, viz., the distension and rupture of the air cells.

These, then, are the physical conditions required in the chest, to produce a strain upon the inner surface of the air cells during expiration. But it remains to show that these conditions are supplied by the circumstances which precede and accompany the occurrence of Emphysema, and also that the external pressure upon the lungs, exerted during these periods of straining, is subject to be weaker in some places than in others.

It is singular that it should not have been recognised, simple and self-evident as it appears, that what applies to the interference with the calibre of the small air tubes applies equally to the large ones. If the calibre of a tube, be it large or small, is justly proportioned to the transmission of a certain volume of air and no more, in a given time, any decrease in the calibre must necessitate either a longer period of time for the passage of the same volume of air, or an increase in the rate at which the air passes. That is to say, a larger slower tide in the first case, and a smaller faster tide in the second case, must pass in the same period of time. But it is clear that a greater pressure from behind is required to urge the smaller-faster tide than that required for the larger-slower tide, if equal volumes are to pass in the same time, as stated, and in proportion to this increase of pressure there will be increase of friction upon the walls of the tubes.

It is evident, therefore, that, as the large bronchi, the trachea, larynx, and nasal passages, are all nicely ad-



justed to carry a given volume of air in a given time, any thickening of their linings, contraction of their walls, or any other cause, which diminishes their calibre, must interfere with this adjustment, and necessitate either a slower tide through them, and thus an increase in the time occupied by the passage of a certain volume of air, or an increased *vis a tergo*, and a quicker tide through them, maintaining the just period at which the given volume passes, but doing so at the cost of increased pressure and increased friction.

In either of these cases the normal respiratory act is seriously disturbed.

It is true that in the case of the nasal passages, so subject to temporary obstructions, special provision has been made, in the power of breathing through the mouth, to avoid, to some extent, the interference with respiration which might otherwise so frequently occur. But below the fauces there is no such safety valve, and the changes in calibre below this point must of necessity disturb the respiratory adjustment.

For these reasons I consider that any decrease of calibre in the nasal passages, larynx, trachea, or large bronchi, must be considered as obstructions to the expiratory tide, and that they are, in fact, much more important obstructions as regards the production of backward pressure than any that can occur in the small air passages, because they interfere with the main thoroughfares of the lungs, whereas the others merely stop the smaller ramifications through which there is not only much less traffic, but the influence of its obstruction is limited to a small number of cells.

It follows, then, that I include, among causes of *sudden*



strain on the air cells, circumstances which have been heretofore overlooked, viz., the acts of sneezing, and of blowing the nose, when they are convulsively or violently performed, and when an abnormal obstruction is placed in the way of the outward tide, as in nasal polypus and tumefaction of the naso-pulmonary mucous membrane.

And I include among causes of obstructed outward tide during *ordinary* respiration, circumstances which have heretofore been overlooked, viz., catarrhal thickening of the mucous lining of the nose, larynx, trachea, and large bronchi.

During all acts of coughing, a certain backward shock of air occurs before the glottis is opened, and before the body to be expelled can be ejected. But this becomes a cause of Emphysema when the cough is unusually convulsive and severe, and when some obstruction is placed in the way of the outward tide, which is unusually difficult to remove.

It has not, so far as I can learn, been recognised, that when the bronchial tubes are diminished in calibre, an obstruction is placed in the way of the outward tide, which does not affect the inward tide to the same extent. Yet such is undoubtedly the fact. It occurs in this way:—The act of inspiration consists in enlarging the capacity of the chest by muscular force, and thus removing from the whole lung the circumferential pressure of its own elastic force; the consequence is, that the expanding lung expands the enclosed bronchial tubes to the fullest extent, and thus favours the influx of air. The normal expiratory act consists in a simple recoil of the elastic lung, followed by that of the elastic chest walls; a pressure is thus exerted on the circumference of the bronchial tubes,



which tends to diminish their calibre, and favours the exit of air; but this expansion during inspiration, and compression during expiration, is all calculated for in the adjustment of normal respiration, and no backward pressure is produced; but so soon as a sufficient diminution has occurred in the calibre of the air passages to require a greater expiratory force than is supplied by the elastic recoil of the lung, an entirely different relation is set up between inspiration and expiration.

So soon as the additional force of muscular expiratory efforts is called for, the circumferential pressure upon the bronchi acts unfavourably to the outward tide. The air cells which lie around the bronchi are pressed upon their walls, and act as causes of increased obstruction to the already diminished passages. In this way diminished calibre of the air passages tells more unfavourably upon expiration than upon inspiration, and becomes a cause of backward pressure of air upon the cells of the lungs during the expiratory act, even in ordinary breathing.

The mode of operation may be watched in the nose, only that there it is exactly reversed. The tendency of the walls of the nose, not the *alæ nasi*, is to collapse during inspiration, and to expand by the pressure of air during expiration. If we experiment upon ourselves, while suffering from the tumid stage of nasal catarrh, we shall find that it is often impossible to inspire through the nose—the suction force exerted by the chest producing collapse of the nasal walls, and complete obstruction—when it is comparatively easy to expire through the nose, in consequence of the passages being opened by the outward tide of air. On the other hand, if we try a similar experiment with the chest, during the tumid stage of



bronchial catarrh, we shall find that it is easy to inspire, when expiration is attended with labour, in consequence of the outward pressure upon the obstructed tubes still further diminishing their calibre.

Supposing it to be granted that, under the circumstances which we have considered, a backward pressure does occur upon the inner surface of the air cells, it remains to be shown that the external pressure upon the lungs during these periods of strain is subject to inequalities.

The principal objection raised to the expiratory theory of the production of Emphysema is an assertion that such inequalities do not exist, an objection which is very well set forth by Dr. Gairdner in the following words:—"Even when the air vesicles are maintained at their maximum or normal state of fulness, by a closed glottis, any further distention of them is as much out of the question as would be the further distention of a bladder, blown up and tied at the neck, by hydrostatic or equalised pressure, applied to its entire external surface." (Monthly Journal of Med. Science, vol. xiii.) This is, in fact, the standing point in the argument, and whatever force it might have as a proof that over-distention of the air cells cannot be produced by expiration, is turned in the opposite direction, if it can be shown that it breaks down at its most vital point, when submitted to the test of experiment; if, in fact, Dr. Gairdner's "bladder blown up and tied at the neck," instead of having "equalised pressure applied to its entire external surface," has the pressure applied unequally, as if grasped by the hand. And whereas there is no evidence to prove the equalised character of the pressure, there is plenty in favour of the unequal pressure.



It seems to be assumed by those who use this argument, that in ordinary normal respiration the air is forced from the lungs by the elastic pressure of the thoracic walls. If this were the case it would certainly be necessary that the weakest parts of those walls should at least be strong enough to prevent any eccentric yielding of the parts to be compressed, viz., the lungs. But I believe this assumption to be entirely false. In a state of health the elastic contractile power of the lung itself is so much in excess of the power of the chest wall to act upon it, that when the thorax has contracted as far as it is possible for it to go, the lung is ready to contract still further, and is actually held back from so doing by its connections with the chest wall. This has been shown by Dr. Hyde Salter, who found in some very carefully performed experiments, that the lung of the dog when released from the thoracic parietes, undergoes a reduction of one-fifth of its volume, and that in the human subject the "elastic contractility of the lung is always drawing on the inner surface of the chest," so that when the ribs have subsided to the exact point at which of themselves they would be disposed to stop they are carried a little further, and only stop when the lungs have drawn them so far beyond their proper point of rest, that the force of recoil thereby generated is exactly equal to the contractility of the lungs." (Lancet, July 29th, August 5th, 1865.) The consequence is, that the chest wall, in ordinary expiration, has only to follow the contracting lung, so that it shall be in a position for expansion when the time arrives for the next inspiration.

There was no need, therefore, that the singular equality of pressure assumed by the argument referred to, should



be provided; and accordingly, in the wise economy of nature, it has not been provided. But when the outward tide is interrupted by some abnormal obstruction, and a call made upon the elastic recoil of the lung greater than that for which it is prepared, the lung instead of taking the lead, and being only followed by the chest walls, falls back upon them for assistance, and they, in their turn, being incompetent by their elastic force to overcome the difficulty, fall back upon the expiratory muscles to assist in the act of compression. In this operation all the inequalities of pressure which may exist become opportunities for the over-distension of the air cells, as they are urged, in this direction and in that, to overcome the obstruction to the outward tide of air.

A striking illustration of the way in which the lungs may be distended during forced expiration, in any direction in which the outward pressure is deficient, was shown in the case of M. Groux, when exhibiting in this country at the different Medical Schools. During a violent expiratory act, the lung of one side came forward in the upper part of the fissure which existed in his chest walls, and formed there a distinct elongated resonant tumour, but no such result took place during inspiration. That in the normal chest inequalities of circumferential pressure exist during expiration, has been clearly shown by Dr. Jenner and others. Thus Dr. Jenner says:—"We have only to watch a person whose chest is exposed during a fit of coughing, to see what a consideration of the anatomical constitution of the thoracic parietes would *à priori* lead one to assert. . . . Before coughing, a person makes a deep inspiration, *i.e.*, he distends as far as possible the air cells, he then closes the glottis, and compresses



forcibly the lungs by the thoracic and abdominal parietes; the moment the compression of the lungs has attained a certain point, he opens more or less the glottis, and the air is driven forward by the muscular effort and the elasticity and contraction of the lungs and of the thoracic walls, with a force proportionate to the compression to which it was subjected before the opening of the glottis.

"Now it is manifest that if there are parts of the thoracic walls which are more yielding, or which, during powerful expiratory efforts with a closed glottis, contract less than others, the air immediately before the opening of the glottis will be driven from the compressed portions of the lung into the air vesicles of the lung situated under such parts of the walls, with a force proportionate to the general muscular and other powers in play, to the local want of compression, and to the degree of yielding of the walls at those particular spots." "That there are such parts," he continues, "and that they are exactly those which are most frequently the seat of vesicular Emphysema, and the sole seats of extreme dilatation of the air cells, is demonstrable. . . .

"That during violent expiration, with a more or less closed glottis, the air is actually driven into the lung vesicles of the apex with power enough to distend them to the utmost, is demonstrated by the supra-clavicular bulging which may be seen during a fit of coughing, and the hand has only to be placed upon the same part to prove that the lung tissue subjacent is, during strong expiration, distended by a considerable force. Percussion proves that the bulging to which I refer is pulmonary.

. . . "The cells of the anterior margin of the lung are, like those of the apex, forcibly distended when



violent expiratory efforts are made with the glottis closed, or imperfectly opened. . . .

“Again, the margin of the base of the lung, the part of the lung in the vicinity of the root of the organ below the entrance of the bronchus, and the little ridge of the lung which lies behind the trachea on the right side forming the posterior margin of what may be termed the tracheal groove on the lung, are, in a like manner, imperfectly supported, and comparatively uncompressed during violent expiration. The base, too, of the left lung, generally, is less firmly supported than is the corresponding part of the right lung—the liver being a more unyielding organ than the stomach, and compressing the base of the right lung more uniformly than the spleen and stomach do that of the left lung. The consequence of this want of support and of compression of the parts of the lung last enumerated is, that they, like the apex and the anterior margin, are the chosen seats of *Emphysema*.” (Med. Chir. Trans., Vol. XL.)

Many other sources of inequality of pressure upon the lungs might be enumerated, amongst which I would suggest the mobility of the heart, which permits it to be pushed downwards, as proved by the change in its position which actually takes place when the upper parts of the lungs become seriously *Emphysematous*.

Again, the possibility of muscular contractions being more powerful and complete in some sets of muscles, and in some sets of fibres, than in others at the same time, is familiar to us all in other parts of the body, and it is only reasonable to suppose that similar irregularities occur when, from any cause, the muscles of expiration are called upon to perform more persistent duties than those for which they are normally intended.



A remarkable coincidence of backward pressure upon the air cells, and absence of uniform external compression must occur when convulsive acts of coughing or sneezing are rendered abortive of their effects by the obstructing body, which those efforts intended to remove, obstinately resisting their force; for the whole muscular force discharged upon the obstructing body must be thrown back upon the volume of air behind it at the very moment when the expiratory muscles have fallen into relaxation and disorder after their convulsive effort.

But even if we could not thus identify the modes and occasions of irregular and unequal pressure upon the superficies of the lungs, there are facts to show that such inequalities must exist.

The fact, for example, that in lobar Emphysema the surface of the lung is often marked with the impressions of the ribs, shows that the pressure from the chest walls is not so exactly uniform, but that the ribs press more and the intercostal spaces less. Again, in lobular Emphysema "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." (Dr. Waters.)

If the equality of external pressure can so completely fail in one spot as to allow such projections to occur and to become permanent, it is clear that it may occur in other parts, and probably in different parts of the chest at different times.

But, in truth, we need hardly go further than the



simple fact with which I started—that distension and rupture of air vesicles producing interlobular Emphysema have again and again occurred during violent expiratory acts, is an unanswerable proof that inequalities of external pressure must occur during expiration sufficient to account for all the effects which we require.

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## LECTURE II.

### CONCLUSIONS—CAUSES OF EMPHYSEMA—ILLUSTRATIVE CASES.

It has now been shown that distension and rupture of the air cells may be produced experimentally by blowing forcibly into the lungs from the trachea, and that distension and rupture of the air cells, causing interlobular Emphysema, have been produced by forcible expiratory acts ; and presuming it to be granted from the arguments which have been adduced—

1. That obstruction to the outward tide of air may be produced by disease.
2. That such obstruction will cause pressure on the inner surface of the air cells during expiration.
3. That pressure so caused may dilate the air cells (as in Emphysema) if the pressure on the superficies of the lungs is subject to be unequal in force at different parts during one act of forced expiration.
4. That such inequalities of pressure do occur.

Then it follows that we have shown as proposed—

1. That the phenomena of Emphysema can be satisfactorily explained by the expiratory theory.
2. That the conditions required by this theory for the production of these phenomena may be supplied by disease.



It now remains to show that—

“Evidences of the existence of the required conditions may be found in the clinical features and histories of cases of Emphysema.” (See p. 6).

Let us, then, enumerate the principal conditions for evidence of the present or past existence of which we have to search in cases of Emphysema.

The required conditions consist of any circumstances which may press the air in the lungs back upon the inner surface of the air cells with greater force than their elastic properties are competent to resist, and thus deprive them of the power of again resisting a distending force which previously they were equal to withstand. They may be divided into two classes—

CLASS A.—Circumstances which may *at once* overstretch the air cells.

CLASS B.—Circumstances which may gradually overstretch the air cells.

These two classes of circumstances often succeed each other, and thus, what one begins, is carried forward by the other.

The conditions included under Class A, or such as may *at once* overstretch the air cells may be conveniently considered under two heads—

1. Violent expiratory acts, as in defæcation, parturition, lifting heavy weights, and the like, performed with a closed glottis.
2. Convulsive expiratory acts, as in whooping cough, croup, laryngitis, fits of sneezing, nose blowing, and the like, when of undue force and opposed by undue resistance.

The second class of circumstances, or such as may



*gradually* overstretch the air cells, may be conveniently considered under four heads—

1. Ordinary acts of coughing, when the free expulsion of the air in the chest is prevented by some unexpected obstruction at the moment when the glottis is opened and the expiratory paroxysm has culminated.
2. Ordinary acts of sneezing and nose-blowing, opposed by considerable obstruction of the nasal passages, and frequently repeated.
3. Ordinary respiration, when the outward tide is sufficiently obstructed by narrowed naso-pulmonary air passages to require muscular expiration.
4. Ordinary acts of coughing, sneezing, or nose-blowing, when some portions of the air cells are deprived of their normal circumferential supports.

*Class A, Order 1.*

Excessive strain from defæcation, parturition, lifting heavy weights, and the like. It is familiar to every medical man that in cases of hernia due to these causes, it is so difficult for patients to recall the exact circumstances, that, even in so serious an injury, the history of when and how it happened is frequently most obscure. The injury is, in a large number of cases, only detected by some accident long after it happened; and in endeavouring to account for it the patient will often refer to several occurrences, either of which might have been a sufficient cause, and will as often be quite unable to remember any occurrences of the kind.



In the case of such an injury as the over-straining of the air-cells, which may not have been attended with any pain or serious inconvenience at the time, and may have long continued to give no serious inconvenience, it is not surprising that patients are unable to give a satisfactory history of its occurrence in a large number of instances, and that, in others, they are able to remember several occurrences, either of which might have been the cause of the damage; so that either way the exact history is left in obscurity.

*Class A, Order 2.*

Fits of convulsive cough, sneezing, excessive nose-blowing, and the like. What has been said of the obscure history of circumstances under the last heading applies equally to this. And in addition it must be remembered, that the damage from these causes will often have occurred at such an early age, that it is impossible for the patients to remember anything about it, and the history, when obtainable at all, is only to be got from parents, guardians, or nurses. For example, in the case of whooping-cough. Nearly every one has had the complaint when a child, and who is to tell which, among all the horrible paroxysms, may have been so severe, or may have happened under such a combination of circumstances, as formidably to overstretch the elastic walls of the air-cells. That such a damage did occur may only come out when, under the influence of time and circumstances, it has become sufficiently aggravated to give rise to some marked symptoms.

It follows, then, that the absence of a clear history of such causes as are included under Class A cannot be taken



as satisfactory proof that they did not exist. On the other hand, we often meet with cases in which the history is so clear and connected that it is difficult to make a mistake.

The following cases may illustrate this statement :—

*Case LXXVII. — Emphysema produced by Laryngitis, aggravated by Bronchial Obstruction latterly. Bronchial Obstruction in partial abeyance at time of Examination from absence of Catarrh.*

Male, aged 44. The breath became short before the occurrence of a cough. It followed an attack of "croup." The croup occurred twenty-five years ago. No regular cough till three years ago. Chest super-resonant. Heart's space slightly encroached upon. Expiration slightly prolonged. No bronchial sounds. Heart's sounds feeble and low down. Pulse feeble. Shoulders high. Neck muscles tense on inspiration.

The breathing became short directly after the "croup," at the age of nineteen, and never was other than short afterwards, but had become shorter and shorter and much worse during the last six years, that is three years before he became subject to cough. The attack called croup occurring at nineteen may have been Laryngitis or severe Bronchitis, with convulsive cough. Having been called "croup," it was evidently attended with great diminution in the calibre of the respiratory passages, or by spasmodic closure of the glottis, and was, therefore, a sufficient cause for strain upon the air-cells and Emphysema. The after history is simply that of gradual increase of the distension by time, and wear and tear. No cough appears to have been set up till three years ago, when he took cold, and since then it had returned with each winter, and with each fresh cold, the mucous membrane having become so irritable that colds set in by *affecting the chest* before



any other part. There was no hereditary taint, and the health was good up to the time of "croup." It should be noted that he was engaged indoors until six years ago, when he took to driving a car, and that this six years corresponds to the period mentioned by him for the increase of his short breath, soon to be followed by cough. The conclusion is, that the bronchial membrane became subject to catarrhal thickening through increased exposure to cold, and hence added to the impediment to breathing, and the Emphysema as a consequence. The absence of signs of Bronchitis at the time of examination was evidently accidental, he being free at that time from cold.

*Case LXX.\*—Emphysema after Croup.*

C. W., aged 26.—Exaggerated thoracic breathing; depressed epigastrium; chest generally super-resonant, most marked in the middle of sternum, encroaching upon the heart's space above, and in the supra-scapular and axillary spaces; least marked between the scapulæ. Inspiration of fair length, expiration of about the same length, both of nearly equal pitch, except when altered for a time by accumulation of mucus, this being removable by expectoration. Rhonchus and crepitation with expiration and inspiration.

When he was seven years old, on returning from a walk with his parents, he was seized with chills and violent cough quite suddenly. A doctor, seen directly afterwards, said it was croup. He was so ill that seven doctors saw him, and he remained ill thirteen weeks. The illness left short breath and some cough, both of which have continued ever since. Before this attack, at seven years old, he was quite healthy. He has never been free from cough since, and breath has never been otherwise than short since; going up stairs or hills, and walking fast, especially aggravate it. A fresh cold is attended with immediate aggravation of his cough, with headache and sore throat.



No other causes than cold aggravate the cough. The causes of fresh cold are, especially, getting warm and going out into cold air, and getting wet. He says, and his mother says, that they are sure the croup was the cause of his troubles, for he never had any other illness. Breath and cough worse the last year. Occupation—a schoolmaster up to 21, then civil engineer three years, and the last two a walking-stick maker. Parents living, healthy.

In this case the Emphysema evidently originated in the convulsive cough of croup, and it had gone on for nineteen years with gradual but very slight augmentation. Some amount of bronchial obstruction had occurred at the time of examination, probably from attacks of catarrh; and the increase of short breath and cough last year were perhaps due to this, which may have been aggravated by his new dusty business of a walking-stick maker.

*Case XX.—Emphysema from Convulsive Cough.* See Index to Cases.

*Case LXXI.\*—Emphysema from Strain in Lifting.*

J. C., aged 56, male, fish porter. Ill five years. Ever since he was a child has had cough in winter, but lost it in summer. The breath was not short till four years ago, when, after lifting a load of about a hundred-weight on to his head, and carrying it, he coughed and spat about half a pint of blood. Since that time his breath has been short, and has got shorter and shorter. This summer, for the first time, his cough has not left him. No asthma in the family. Neck muscles very tight on inspiration, but they have very little effect on the chest. Apices of both lungs very prominent above the clavicles, and very super-resonant. Upper two-thirds of front of



chest very prominent and super-resonant, also the lower parts of the back of the thorax. Heart's space encroached upon from above by super-resonance. During ordinary respiration very little respiratory sound heard, and that very soft, without rhonchus or sibilus. On forced expiration, a very soft, *low-pitched* sound is heard. After forced inspiration, what little movement of recoil occurs is sudden, and neither expiration nor inspiration is prolonged.

NOTE.—There are here no signs of obstructed outward tide, therefore, although the expiratory tide has little force, it is soon over, and the sound produced is low in pitch and soft. The history shows that no short breath, therefore no narrowed calibre of air-passages or Emphysema, preceded the accident. The sudden alteration of the breathing and its persistence, coupled with the single attack of spitting of blood immediately following an expiratory strain, point unmistakeably to the sudden production of Emphysema from overstrained and ruptured air-cells, due to lifting too heavy a weight.

*Case LXXXIII.\*—Emphysema from Strain.*

J. E., aged 64, smith. Accustomed to work in wells, and to be much exposed to cold and damp all his life, but noticed nothing wrong with his chest till ten years ago, when he fell from a height, and saved himself by catching at a rope, on which he hung by one arm for some time. He felt that he had "strained his wind," and also found that he had ruptured himself in the groin. From this time he has been short-winded, and subject to cough with every little cold. The short breath and cough have become worse every year, especially the last four. They are partially relieved in warm weather. Neck muscles very tense and prominent during inspiration; very little respiratory movement. Chest very super-resonant all over. Inspiration very feeble; expiration long and soft generally, but in some places high pitched on forced expiration.



*Case II.—Emphysema from Whooping Cough, the Air-tubes previously narrowed. See Index to Cases.*

*Class B, Order 2.*

Ordinary acts of coughing, when the free expulsion of the air in the chest is prevented by some unexpected obstruction at the moment when the glottis is opened and the expiratory paroxysm has culminated. I have already referred to this combination of circumstances as a remarkable example of the coincidence of strain upon the air-cells, and absence of uniform external compression (p. 21), that is, of all the conditions necessary for the production of Emphysema. Practically, this is an operation frequently occurring in cases of Bronchitis, Tracheitis, Croup, and the like. Yet it is almost impossible to identify it in the history, for it does not happen with every cough, and the very circumstance which gives it point—the pellet momentarily stopping the way—is a moveable one, of which no trace may be found by any post-factum examination. These, however, are just the occasions on which collapse of portions of lung is apt to occur; for the forcible inspiration which follows the expiratory paroxysm sucks back the foreign body into the smaller air tubes, whence it may fail to be again ejected. When this happens, the physical signs during life may indicate the accident, but more frequently it is unnoticed at the time, and when the collapsed lung-tissue, and the Emphysematous cells, are found after death, they are supposed, by the morbid anatomist, to be related as cause and effect, instead of as common effects of one cause.

There is, however, a feature which may lead to the



suspicion of this cause of Emphysema during life, independent of the question of collapse.

The patients complain that they "cough and cough again and again," each time expecting, but failing, to eject the phlegm which they feel to be in their air-tubes, but that, a few minutes after the more severe and fruitless coughing has subsided, a trifling cough will bring up easily that which resisted the severe effort. In these cases I believe we may fairly suspect the existence of such a cause of obstruction as I have described, and must regard it as peculiarly liable to an over-stretching of the elastic walls of the air-cells.

A similar coincidence of over-strain upon the air-cells, at the moment when extreme compression is relaxing, occurs in Spasmodic Asthma. But in this case, instead of a pellet of mucus, too large or too tenacious to pass the glottis, acting as the obstructing cause, the blocking up of the air-passages is due to a sudden accession of spasmodic contraction of their walls in front of the outward tide; in this contraction pellets of mucus may be grasped, and thus assist in plugging up the tube which they would not otherwise have been large enough to stop; but without their presence the spasm itself may be sufficient for the purpose. A patient, struggling for breath under an attack of Spasmodic Asthma, is suddenly permitted to get a freer inspiration through a momentary relaxation of the spasm. The presence of fresh air in the chest excites a cough, but, just as the act culminates, a sudden accession of spasm stops the way, and throws back the air upon the cells. This occurs again and again in every attack of Spasmodic Asthma, especially towards the close of the attack, when the spasm is beginning to



give way, and mucus is accumulating in the air-passages. It is not to be wondered at, therefore, that Emphysema—and that of a general character—is so common a vestige of Spasmodic Asthma. There is another mode in which this complaint leads to Emphysema, but it will be referred to under a future heading.

I am aware that a very different explanation of the relation between Spasmodic Asthma and Emphysema is given by Dr. Salter; but although it possesses all the care and complication of detail which distinguish the explanations of this most diligent observer, I must confess that to my mind it entirely fails in explaining the phenomena.

Dr. Salter says, p. 145 of his work on Asthma:—

“ Adopting that view of Emphysema so ably advocated by Dr. Gairdner—that it is essentially a *compensatory dilatation*, and implicates the neighbourhood of non-expandible lung—I believe the mechanism of the production of Emphysema by Asthma to be as follows:—The bronchial spasm shuts-off the air; the shutting-off the air produces capillary stasis—partial asphyxia; the congested vessels relieve themselves by the characteristic mucous exudation; the continued occlusion of the bronchial tubes, if the spasm does not yield, shuts up this mucus, and prevents its escape, and at the same time, by barring the access of air, prevents efficient cough; so long as spasm lasts, therefore, its escape is doubly prevented by the direct obstruction, and by the want of the natural machinery for expulsion. The tubes affected by the asthmatic contraction thus become obstructed in a twofold way; at first narrowed by spasm, and then completely occluded by mucous infraction. As long as the spasm lasts the escape of mucus is impossible. In the meantime, what-



ever may have been the length of the attack (and we know that it often lasts for days), the inspiratory muscles are making the most violent efforts to fill the chest, and are, in fact, keeping it in a state of extreme distention. The length of time required for the removal of air from a lobule, from which communication with the external atmosphere is completely shut-off by occlusion of its bronchial tube, I do not know ; so I do not know if, *in a single attack of Asthma*, any actual lobular collapse could take place, although in a prolonged attack of some days, I feel no doubt that it would. At any rate, the lobules whose bronchi are occluded cannot yield to the distending force of the inspiratory muscles ; the whole distension of inspiration is, therefore, spent on those portions of the lungs whose communication with the external air is free. The open lobules have to expand for themselves and their occluded neighbours, and undergo an excessive inflation, in proportion to the amount of lung that is non-expansile ; in other words, *become Emphysematous*. If we consider how complete the occlusion must be by this double process of spasm and infarction, how protracted Asthma often is, and how violent are the inspiratory efforts that characterise it, I do not think we shall wonder at any amount of Emphysema that is thereby produced, nor at its being one of the commonest organic changes to which Asthma gives rise."

You will observe that this explanation or theory of Dr. Salter's necessitates the belief, first, that asthmatic spasm is a localised affection, limited in its greatest intensity to a certain number of tubes, and leaving other parts of the lung comparatively unaffected throughout the whole of an paroxysm ; of which there is not only no proof, but on



the contrary every reason for disbelief, from the nature and causes of the complaint.

Secondly, this theory necessitates that lobular collapse should be a constant accompaniment of Emphysema and also of Asthma, and that the Emphysema of asthmatics should be of the lobular kind; neither of which conditions is to any large extent consistent with experience.

Thirdly, if such complete and permanent infarction of bronchi as is required by this theory takes place during an asthmatic paroxysm, it is strangely inconsistent with the rapidity and completeness with which a paroxysm of asthmatic dyspnoea clears off directly the spasm yields.

*Class B, Order 2.*

“Ordinary acts of sneezing and of nose-blowing, opposed by considerable obstruction of the nasal passages, and frequently repeated.”

I have already given the reasons why these circumstances must be included among causes of Emphysema (see Sec. 1), and that they are competent to produce strain upon the air-cells, can hardly be doubted by anyone who will carefully watch his own sensations when suffering from severe nasal catarrh in its tumid and obstructive stages. The backward pressure of the air can be plainly felt at all parts of the chest during sneezing and nose-blowing, and the harder the blow, or the more violent the sneeze, the more forcible and general is the sense of distension.

If these are to be included as causes of over-distension of the air-cells, it follows that we must consider a history of severe and repeated nasal catarrhs as an important



feature in a case of Emphysema. It appears to me to be one of the conditions especially calculated to produce *general lobar Emphysema*, the class of cases which have been regarded as so difficult to account for, that Dr. Waters,—after conducting a very excellent argument to prove that expiratory efforts are the effectual means of producing Emphysema, that inspiration is incompetent to produce it, and that the existence of degeneration of the air-cells is not supported by facts,—actually deserts his vantage ground, and, resorting to an imaginary state of degeneration of the air-cells, assumes the possibility of their distension by *inspiration*, which he then asserts to be the cause of lobar Emphysema, so puzzled is he to account, in any other way, for general distension of the air-cells (Waters on Emphysema, &c., p. 52).

The possibility of nasal catarrh becoming a cause of a severe disease in the air-cells does not appear to have occurred to anyone. I suppose it has been thought that so common and undignified a complaint as “a snivelling cold” was not worthy to be the cause of such an important disease as Lobar Emphysema, forgetful of the trite but true adage, “Great events from little causes spring.”

*Case LXXII.\*—Emphysema from Nasal Catarrh; Loss of Expiratory Power.*

T. W., aged 68, bricklayer, November, 1864.—(See Index to Cases). Very weak; chest thin, large-boned, not distended abnormally; raised in mass with very little expansion during inspiration; resonance generally increased and high-toned; heart's space encroached on from above; inspiration high-pitched, feeble; occasional rhonchal expiration, soft, long, feeble, with occasional high-pitched sibilus; heart's sounds sharp and thin-



valled; epigastric impulse; very little expectoration. Has never had much cough, but the breath has been getting short for five years. He has been long subject to frequent, almost constant nasal catarrh, with much sneezing, and to these he attributes the short breath. Never had any other illness. Every fresh cold in the head makes the breath shorter while it lasts, and when it goes, although the breath is better again on the whole, it gets worse and worse with each successive attack. Not suffering from cold at present time.

In this case careful inquiry could discover no other rational cause for Emphysema than the catarrh, and that had been a very marked feature, giving the patient the impression that it was the cause of all his troubles.

It is probable that, except during an attack of fresh cold, the obstruction to the outward tide had not been very great, but it was getting greater and greater.

(See also Appendix on Post-nasal Catarrh).

### *Class B, Order 3.*

“Ordinary respiration when the outward tide is sufficiently obstructed by narrowed naso-pulmonary air passages to require muscular expiration.”

This is “the head and front of the offending,” for all forms of catarrh, and some other analogous causes, are competent to narrow the air passages at some part of the mucous tract, beginning at the nose, and going down to the remotest bronchi.

No doubt catarrh may occur without producing such effect; that in proportion as it is readily resolved, the effect is avoided.

We are able easily to watch the progress and effects of



catarrhal inflammation when it occurs in the conjunctiva. This mucous membrane may be actively inflamed, and yet be restored to its pristine clearness and tenuity; and such attacks of inflammation may occur again and again, and still leave no thickening of the membrane.

But such is not the usual course of events. It more frequently happens that if the attacks of catarrhal conjunctivitis are often repeated, they become more and more chronic in their character, each time being less readily resolved and more easily re-excited, indicating a less complete cure, until the tenuity and clearness of the membrane are interfered with. What happens here is precisely what happens to all the mucous membranes of the body, if subjected to irritation, whether from cold or from other causes. Indeed, the eye, irritated by inverted lashes or by a granular lid, presents us with an example of gradual thickening of mucous membrane from other causes than cold, just as the naso-pulmonary mucous membranes of the snuff-taker, the stonemason, and the brush-maker present examples of thickening from the irritation of snuff or of dust.

*Case XXIV.—Irritation of Dust, producing Bronchitis and Emphysema.*

Aged 40, male. The breath had been short four years, and became so one year before the cough began. There was no illness at the time; the previous health was good; but the breath had not ceased to be short since it began to be so. The cough came on with copious expectoration twelve months after the breath began to be short, and had continued more or less ever since except once, when it left for a short time about two years after its commencement. Fresh colds always affected the chest first, and were



attended with copious expectoration, and were especially apt to be produced by wet feet. The patient was naturally weak, but had no hereditary taint, except rheumatism on the father's side. He had been a brushmaker twenty-one years, which is a dusty trade.

This history leads to the opinion that the pulmonary mucous membrane had become irritated and thickened by the dust of his trade, and this was followed by bronchitis and further thickening of membrane, as evidenced by prolonged wheezing expiration, and that the Emphysema was a secondary affection.

*Case LXXIII.\*—Thickened Naso-pulmonary Mucous Membrane in a Stonemason.*

T. C., aged 33, male, stonemason, Nov. 8th, 1865. Irritable cough, especially tickling in the throat; expectoration of whipped-up froth; breath short. Had a cold followed by cough last winter, and the breath has been somewhat short ever since, not short before. Cough was better or well during summer, but three months ago came back with a cold, and has been worse this year than last, and breathing is shorter. He gets fat, is temperate. No consumption or asthma in the family. He looks fat and rather bloated. Says his breath and cough are better lying down than up. Heart sounds normal; chest sounds normal, except some transmitted harshness in large bronchi; apices very resonant, as compared with lower lobes posteriorly, which are rather deficient in resonance. Larynx and tracheal sounds very harsh, and expiratory sounds high pitched on forced expiration. Mucous membrane of pharynx somewhat tumid, and the follicles prominent. Says he feels all his trouble in the windpipe.



In this case the upper part of the naso-pulmonary mucous membrane had been long irritated by the stone-dust, which the man inhaled at his trade, and it was therefore easily and severely affected by catarrh when it occurred, so that the membrane remained thickened after the cold left, and was readily susceptible to a renewal of catarrh, which was attended with increased thickening of the membrane. His short breath was evidently due to the narrowed tubes. These were beginning to act as causes of distension of the air-cells at the upper parts of the chest.

Fortunately, mucous membranes, like the skin, have been endowed with a property somewhat analogous to that possessed by erectile tissues. They can become highly turgid with blood, and rapidly return to their less turgid condition without leaving any traces of their former injection. In the skin we call this "blushing," and the mucous membrane, which resembles the skin in so many other points, resembles it also in this, that it can blush.

This is a very important point to bear in mind, as it explains many phenomena connected with affections of the air-passages, which otherwise might very much puzzle us, and which have undoubtedly been stumbling-blocks to many.

The analogy, too, between the mucous membranes and the skin, in this matter of blushing, is kept up in another important respect. If the turgidity of the skin is but transient, it may be accompanied by no perceptible augmentation of secretion; but if it continues, it leads to increased secretion, and with this the turgidity subsides. The same thing happens with the mucous membranes;



they may blush or flush, and return to their normal condition without leaving any effect; but if the flush or vascularity continues beyond a certain time increased secretion is the result, and with this comes relief to the turgid membrane. It is more than probable that no thickening or other damage occurs in the membrane, however often it may flush, unless the turgidity persists long enough, or is sufficiently pronounced to require increased secretion for its relief. Clinical observation leads me to this opinion.

But then, practically, if a mucous membrane is called upon to flush, or become turgid with undue frequency, it loses its capability of quick restoration to its normal state, and gradually the flush becomes more permanent and more pronounced, and increased secretion becomes necessary for its relief. The relief thus afforded becomes less and less complete, and the excitability of the membrane to fresh flushing is augmented until it becomes more and more hypertrophied.

One of the functions of the secretion from mucous membranes is to assist in removing foreign matters from their surfaces. These membranes, therefore, have been endowed with an especial susceptibility to irritations applied to their surfaces. Hence it happens that, although they may suffer flushing from causes proceeding from other sources, as, for example, qualities of blood circulating in their vessels, and sympathetically with other organs through the nervous system, these causes do not so readily excite increased secretion as irritations applied to their free surfaces. Now, it is exactly upon the free surfaces of these membranes that causes of catarrh are most readily applied. Hence catarrh of mucous membranes is especially attended with morbidly increased secretion, and its



too frequent repetition is productive of thickening of the membranes affected.

Of all the causes, therefore, of thickened naso-pulmonary mucous membrane, and consequent narrowing of the respiratory tract of tubes, *catarrh* is that which acts most frequently and prejudicially.

In the same rank with catarrh produced by the contact of an atmosphere meteorologically irritating to the air-tubes, must be placed the effects of those mechanical irritants which in various trades are inhaled with the air, as in the cases already referred to, and also in many others.

Then there is the catarrh produced by sympathy with the cutaneous surface. Then the effects of rheumatic, syphilitic, typhoid, gouty and other poisons in the blood circulating in the lungs, and acting as irritants from within; and in the same rank come the effects of ill-digested, ill-assimilated foods poured into the blood from the intestines, and of poison absorbed from the stomach. I have seen nettlerash of the bronchial and faucial mucous membrane, threatening suffocation, proceeding from the same causes as nettlerash on the skin, and in the same person, affecting at one time the cutaneous, at another the mucous surfaces.

In my opinion, the whole of this series of phenomena in the mucous membranes of the air-passages, the transient flush, the more permanent turgidity resulting in secretion, the increased susceptibility to the repetition of these states, and the final hypertrophy of the membrane, are illustrated by spasmodic asthma. From one or more of the list of causes competent to produce flushing of the bronchial mucous membrane being too often repeated, an undue



sensitiveness to the influence of such cause is induced. At first the flushing is but transient, and produces, through the temporary tumidity of the membrane, a slight dyspnœa, quickly passing off as the flush subsides. By-and-bye, the intensity of the flush becomes greater; and either from this cause alone, or from the superaddition of some cause of nervous depression or excitement, spasmodic contraction of the bronchial muscular fibres accompanies the flush. This, too, may be so transient as to pass away without increased secretion taking place from the membrane, and thus a paroxysm of asthma comes and goes, like the blush on the countenance, as it were "by magic." But more intense and abiding turgidity brings things to a pass beyond the power of the vascular structure to recover itself by resolution, and an attack of asthma is produced, of which the spasmodic contraction and dyspnœa are obstinately persistent, until, at length, the membrane relieves itself by increased secretion; the irritability of the muscular fibres subsides, and the paroxysm passes off with expectoration.

*Case LXXIV.\*—Case of Flushing of Naso-pulmonary Mucous Membrane, sometimes in one part, sometimes in another; sometimes ending in sneezing and secretion from the nose and frontal sinuses; sometimes in dry spasmodic asthma passing off by resolution; sometimes resulting in humid spasmodic asthma passing off with expectoration.*

I have long had the opportunity of watching all these phenomena in a very interesting case, which I have attended for the last ten years.

This gentleman enjoys good general health, is between thirty and forty years of age, of active and temperate habits, but rheumatic constitution. He is, and has been



since his youth, subject to slight attacks of spasmodic asthma, and to severe running and sneezing colds in the head. These are produced by a number of different causes, of which the following are the principal and most undoubted:—Damp feet; draughts of cold air on the face; hot rooms, if the blood gets into the face and head; mental occupation, if continued after the head and face get hot and the feet cold; an extra glass of wine, if it flushes the face; effervescing drinks, if taken when the gas is escaping briskly; bright sunshine; east winds; getting over-heated by exercise, so that the face gets very hot; any food that disagrees with the stomach; sleep; an excess of rheumatic acid in the system.

The usual effect of any one of these causes is to produce congestion and tumidity of the nasal mucous membrane, which goes on increasing till violent sneezing is produced, and the relief comes in a profuse, very watery discharge from the nose, eyes, and posterior nares. This lasts for an hour or two, and the whole attack subsides and gradually passes away. But if, when the attack is in its first or flushing stage, the forehead and nose are bathed with cold water, or freely exposed to cold air, the sneezing and coryza are postponed, and may be altogether kept off; but then an attack of spasmodic dyspnœa is produced, for which there is no relief, except either a free expectoration or a re-excitement of the former symptoms of sneezing and running at the nose, which will then remove the chest symptoms. When the patient is unusually rheumatic, and the secretions very acid, the tendency to recurrence of coryza or asthma is very much increased, but is certain to be relieved by alkalies. The attacks of coryza can be kept off almost indefinitely by small doses of morphia; but this suppression is always followed by an increase in the tendency to spasmodic asthma, the attacks getting more frequent and severe till the morphia is withdrawn, and the coryza allowed to have its way.

But the coryza and the asthmatic dyspnœa, terminating in expectoration, are only the fully developed forms of numerous slighter attacks of flushing of the mucous mem-



brane, which give rise at one time to temporary stuffiness of the nasal passages, and at another to temporary stuffiness of the bronchial passages with dyspnœa, and their passing off, as if by magic, without any increased secretion.

We see, therefore, in this one case an illustration of both the humid and the dry asthma, so called; that both can be produced by different stages or degrees of the same pathological states, and by the same causes; that these causes may affect at one time one part of the naso-pulmonary mucous tract, and at another time a different part, and that one part is capable of acting for the relief of another.

I have never seen another case in which all these phenomena were so palpable and so conveniently combined, but I have seen many analogous cases, in which they might be witnessed to a greater or less extent. In fact, you will find that all cases of spasmodic asthma bear certain analogies to this case. The repetition of these phenomena, like the repeated catarrhs and other sources of mucous irritation which I have mentioned, leads, step by step, to hypertrophy of the lining membrane of the air-tubes and narrowing of their calibre, and hence, at last, to Emphysema.

We must then consider, among important features in the clinical history of a case of Emphysema, the occurrence of any of these causes of too great and too frequent turgidity of the naso-pulmonary mucous membrane, because they are causes of narrowing of the air passages.

The following extracts from cases in which there were physical signs, both of Emphysema and Bronchitis (see Index to Cases, WHITE GROUP), will show the extent to which these causes of Emphysema were discoverable in the clinical histories.



In nineteen of the cases, the short breathing and the cough came on simultaneously, and the nature of the first attack was thus described :—

*Case V.*—A bad attack of Bronchitis, which left the breath short. No other illness. Previous health good. Never free from cough since.

*Case IX.*—An attack of inflammation of lungs fourteen years ago, after which breath was found to be short. No other illness. Never free from cough since.

*Case XIV.*—Nine years ago a bad cold and cough, which left breath short. No other illness. Cough returns every winter. Better in warm weather.

*Case XV.*—Twenty years ago a bad tickling cough which left breath short. Never free from cough since.

*Case XXVII.*—Ten years ago severe influenza cold and cough, which left short breath. No other illness. Never free from cough since.

*Case XXX.*—Two years ago, while suffering from cough, with tickling in throat (Bronchitis) walked very fast and brought on short breath. Has been free from cough once since, under treatment. Inclination to spasmodic dyspnoea at night.

*Case XL.*—Twelve years ago hacking cough, and much phlegm, which left short breath. Both cough and breath have been better and worse, according to weather, ever since.

*Case XLII.*—Twenty-eight years ago cold in the chest, with tightness and cough. Never free from cough since.

*Case XLIV.*—Nine years ago influenza cold, debility, and cough, with short breath. Never free from cough and short breath since.

*Case XLVII.*—Nine years ago cough and tightness at chest, from exposure to cold. Never free from cough since. Breath better in warm weather.



*Case L.*—Cold and cough twenty years ago, leaving short breath, but breath was better during first five years, worse since. Never free from cough.

*Case LI.*—Influenza and cough ten years ago. Bad every winter since, but better in summer.

*Case LII.*—Cold and cough nine years ago, aggravated since by every cold, which is very often. Better in summer.

*Case LV.*—Four years ago bad cough and cold, from wet feet; left short breath. Never free from cough since.

*Case LVI.*—Violent cough, with pains in chest and sides; left short breath. Cough returns every winter since. Better in summer.

*Case LVIII.*—Sixteen years ago bad cold and cough. Left short breath. Breath has continued short. Cough better in summer, worse every winter.

*Case LIX.*—Twelve years ago severe influenza and cough. Cough and breath aggravated with every cold since, but both better in summer.

*Case LXI.*—Five years ago cold and cough from getting wet. Better and worse since.

*Case LXII.*—Age 18. Had whooping-cough, measles, and scarlet fever, before three years of age, and breath has been short ever since. Cough with every fresh cold. Never free.

In three cases the breathing became short within one year after the cough began. The first attack of cough, and the setting-in of short breath, are thus described:—

*Case XXXIV.*—Twelve years ago cough came on, attended with nervous debility, and soon after the breath began to be short in winter. Never free from cough summer or winter for twelve years; but breath has not



been short in summer till last four years. Cough has gradually got worse and worse, aggravated by change of weather.

*Case XXXVIII.*—Five years ago cough began, with inflammation of chest from cold. Previous health good. Breath first became observably short twelve months after. Cough never absent, but worse in winter. Breath not short in summer.

*Case XLI.*—Three years ago cough began, with tightness of chest and hard breathing. It has been better or absent in summer, but brought back with slightest cold. Breath not short till twelve months after cough, and has ceased to be short in the middle of summer.

In five of the cases, the breathing did not begin to be short till more than one year after the cough came on, and the nature of the first attack is thus described:—

*Case XXII.*—Twenty-three years ago cough began in autumn, more in the day than night. Nothing else the matter. Cough often absent in summer, but returning every autumn. Breath did not get short till thirteen years after cough began. No other illness at time, except inflamed veins in legs. Has been short ever since, getting worse and worse.

*Case XXVI.*—A little over four years ago a very bad cough set in, with cold and violent shivering, and had been renewed with every fresh cold, though absent in summer. The breath began to be short from one to two years after first attack of cough, and had been better and worse, according to weather, ever since.

*Case XXXIII.*—Liable to coughs and colds for years, but a bad attack of cold, violent cough, and swollen glands two years ago. Cough ever since. The breath did not get short till six months ago, and is not observably short now. Never free from cold.



*Case XXXV.*—Twelve years ago cough began, and got gradually worse, though sometimes absent in summer. Breath became short six years ago, after confinement with twins. Short ever since, probably from the strain of parturition.

*Case XLVI.*—Cough began in early life, gradually getting worse each winter; better in summer. Breath became short about twelve years ago (many years after cough) during a cold, breath not short when free from cold.

*Class B, Order 4.*

“ Ordinary acts of coughing, sneezing, or nose-blowing, when some portions of the air-cells are deprived of their normal circumferential supports.”

If collapse of the lung ever acts as a cause of Emphysema, it must, I think, be included under this heading. As it was seen in the case of M. Groux, to which I have already referred, that during forced expiration, the portion of lung, deprived of external support by the deficiency of the chest wall, puffed up and bulged into the fissure, it is possible that the same thing might happen to a portion of lung deprived of its circumferential support, by the collapse of its neighbouring lobule. But I shall have occasion to refer to this point by-and-bye.

I have now enumerated some of the principal conditions, for evidence of the past or present existence of which, we have to search in cases of Emphysema. But I must not leave this part of the subject without pointing out the importance of *caution* in making this search. It generally happens that the only symptom we have to guide us in our enquiries, as to when Emphysema first existed, is the shortness of breath. We endeavour to ascertain at what period in the history the breath was first perceptibly short; and we shall be led to very mistaken conclusions,



unless we take great pains to ascertain whether other circumstances existed at the time the breath became short, which were more likely to have been its cause than Emphysema. The neglect of this precaution has, to my knowledge, often led to wrong theories as to the causation of Emphysema in particular cases, and might easily have done so in some of those which I have recorded.

The following table shows the relation, in point of time, between the beginning of short breath and the beginning of cough in fifty-eight cases:—

TABLE I. (See Index to Cases)	C O U G H B E G A N						
	Before Short Breath in 36 per cent. of 58 cases.			Same time as Short Breath in 47 per cent. of 58 cases.	After Short Breath in 17 per cent. of 58 cases.		
	More than 5 years before Short Breath.	From 1 to 5 years before Short Breath.	Less than 1 year before Short Breath.	Same time as Short Breath.	Less than 1 year after Short Breath.	From 1 to 5 years after Short Breath.	More than 5 years after Short Breath.
White 33 Cases	5*	1	3	19	3	1	1
Per Cent. ...	15	3	9	58	9	3	3
Red 18 Cases ...	7*	0	0	8	1	0	2
Per Cent. ..	38			45	6		11
Yellow 1 Case	0	0	0	0	1	0	0
Per Cent. ...							
Blue 6 Cases ...	3	2	0	0	0	0	1
Per Cent. ...	50	33					17
Total 58 Cases	15	3	3	27	5	1	4
Per Cent. ....	26	5	5	46	9	2	7

\* In one of each of these cases the Breath was reported not short.

NOTE.—In this and all the Tables, the figures are as nearly correct as was possible, without introducing decimals.



This Table shows that in the cases where Emphysema existed—viz., the Yellow, Blue and White Groups—the cough began *before* the short breath in 35 per cent., and came on simultaneously with it in 48 per cent., making 83 per cent. in which the cough either preceded or accompanied the beginning of short breathing.

In the cases of Bronchitis without Emphysema—(see Index to Cases, Red Group)—in which, therefore, the short breath could not have been due to Emphysema—it came on simultaneously with the cough in 45 per cent., so nearly the same proportion as in the cases of Emphysema as to make it probable that the short breath in those cases, at least in its commencement, was more dependent on the Bronchitis than upon the Emphysema.

But the greatest interest with regard to the origin of Emphysema attaches to seven cases in which the cough did not begin till after the short breath. These were Cases II., XVII., XXIII., XXIV., XXVIII. in the White Group; Case XX. in the Yellow Group; and Case LVII. in the Blue Group. It is in such cases as these that the Emphysema is apt to be set down as an idiopathic affection, for want of more careful scrutiny into their history. On making a very close examination, however, we find the following explanations of the relation between the beginning of cough and of short breath. Case LVII. has been already referred to (see Index to Cases), Emphysema was produced by Laryngitis.

In Case II. the short breath which preceded the cough by thirteen years, was attributable to catarrhal thickening of the naso-pulmonary mucous membrane, consequent upon measles; while the cough came on with whooping-cough; and the Emphysema was evidently due to the convulsive cough occurring in a person with narrowed air passages.



In Case XVII. the short breath which was said to have preceded the cough by five years was found to have only been present during attacks of nervous palpitation, which occurred at a time of disturbed health, due to the cessation of menstruation, and had nothing to do with the chest affection. The amount of Emphysema was very slight, and probably dated from an attack of Influenza and Bronchitis, with which the cough commenced.

In Case XXIV. the patient was a brushmaker, and the short breath which was coming on for 12 months before the cough began, was due to thickening of the naso-pulmonary mucous membrane, produced by the inhalation of dust from the bristles used in his trade. The cough came on as the irritation got worse and worse, and the Emphysema had been gradually produced by the narrowed air passages.

In Case XXIII. the cough and short breath had both existed about ten years; but the short breath had preceded the cough by a slight interval. It came on with a bad cold, and was due no doubt to catarrhal tumidity of the mucous membrane, for it ceased to be short when warmer weather came. Attacks of cold and Bronchitis had returned every winter, and gradually produced the Emphysema discovered on examination.

In Case XX. the Emphysema was evidently produced by a very violent attack of convulsive coughing, brought on by an unusually bad London fog, and by subsequent attacks of a similar kind excited by fogs. The short breathing said to have preceded these convulsive coughs was found to have occurred during an attack of piles and palpitation of the heart, accompanied by general illness. The patient was a tailor, and the heart was apparently fatty.

In Case XXVIII. the short breath which preceded the cough was only due to gradual increase of bronchial congestion, which culminated in broncho-pneumonia, when the cough came on. The Emphysema was altogether a subsequent affection.



It appears, then, that in these seven cases in which the short breath preceded the cough, a close enquiry discovers in each case a rational explanation of the phenomenon quite consistent with the expiratory theory of the production of Emphysema.

Table II. shows that in the fifty-eight tabulated cases the average duration of *short breath* at the time of examination was 6·5 years, the maximum duration 28 years, the minimum 1 year; whereas the average duration of the cough was 9·5 years, the maximum duration 50, the minimum 1.

The average age at which the short breath began was thirty-nine years; the average age at which the cough began was 37·5 years.

Hence, in the series of fifty-eight cases the cough preceded the short breathing by three years on an average; and the average age at which the cough began was about 1·5 years less than that at which the short breath began. The details of the several groups are seen in the Table.

TABLE II. (See Index to Cases).	SHORT BREATH.						COUGH.					
	Duration of Short Breath. In years.			Age when Short Breath began. In years.			Duration of Cough. In years.			Age when Cough began. In years.		
	max.	min.	aver- age.	max.	min.	aver- age.	max.	min.	aver- age.	max.	min.	aver- age.
White 33 cases	28	1	10	60	1	31	46	1	11	66	1	29
Red 18 cases .	20	1	7	52	16	32	50	1	17	39	4	25
Yellow 1 case			1			59			$\frac{3}{4}$			59 $\frac{1}{4}$
Blue 6 cases .	25	1	7	47	10	34	20	3	10	40	28	37
Total 58 cases	28	1	6 $\frac{1}{2}$	60	1	39	50	1	9 $\frac{1}{2}$	66	1	37 $\frac{1}{2}$



### LECTURE III.

DIAGNOSIS OF SPASMODIC AND NON-SPASMODIC NARROWING OF THE  
AIR PASSAGES — ILLUSTRATIVE CASES.—OBJECTIONS TO THE  
DEGENERATION, COLLAPSE, AND INSPIRATORY THEORIES. —  
IMPORTANCE OF THE EXPIRATORY THEORY.

We have seen the importance of getting accurate particulars of the early history of these cases, in order to avoid false conclusions as to their nature and origin; and without pretending to have exhausted the subject, I believe I have now laid before you a sufficiently comprehensive list of the circumstances capable of overstretching the air-cells, either *gradually* or *at once*; and for which therefore we have to search in the clinical history of cases of winter cough, accompanied by Emphysema of the lungs.

I shall now pass on from the history of these cases to the clinical features which they present on examination.

I have shown you that narrowing of the naso-pulmonary air-passages holds the most important place among causes of Emphysema; and I now come to consider the means which we have at our disposal, by which to ascertain, during life, whether or not the respiratory tubes are narrowed in any given case. I shall be able to point out some very simple physical signs, the importance of which does not appear to have occurred to other observers than myself, as they are not mentioned in books, but which are, I think, capable of satisfactory demonstration.

I refer especially to certain modifications of *pitch* in the inspiratory and expiratory sounds.



In order to give these their proper meaning and value, it is necessary to bear in mind a few acoustic details. I hoped to have been able to present you with the results of some experiments on this subject with which I have long been engaged; but they have proved more complicated than I anticipated, and I have not been able to get them finished in time for these lectures. I shall not therefore trouble you with more than a very brief recital of the recognised rules with regard to pitch which are necessary to the present question.

Of all the qualities of sound, except loudness, pitch is that most easily and unmistakably distinguished. It refers, as you are aware, to the differences popularly known as "high" and "low"; differences which, if they are at all wide, are caught at once, even by the uneducated ear, but which, to a good musical ear properly educated, are distinguishable when reduced to a mere fraction.

For this reason I attach the greatest practical importance, in physical diagnosis, to all modifications of *pitch*.

*Timbre* is a most valuable quality of sound, and, when thoroughly understood, is competent to give very important indications in disease; but it is far more subtle in its nature than pitch, and therefore more subject to be misunderstood. Its appreciation by the ear is less amenable to education, and its modifications are due to such a variety of subtle causes that it is beset with sources of fallacy, and above all, its characters are exceedingly difficult to describe.

Pitch, on the other hand, is not only easily detected and easily described, but there is one constant condition upon which all its modifications depend, viz., the rate of vibration. All continued sound is but a repetition of im-



pulses, and the pitch depends upon the number of these which occur in a given time. The slower the rate the lower the pitch; the more rapid the rate the higher the pitch. You are aware that this applies equally to all sonorous bodies, and that, although the pitch of a sound elicited from a vibrating string may be raised to the same extent by either halving its length, quartering its weight, or quadrupling its tension, yet that this is only because, by each of these operations, the rate of vibration is affected in an equal degree. And when sounds are produced by the vibrations of air contained in tubes, the same effects are obtained by changes in the length and calibre of the tubes, and by the open or closed condition of their ends, as by alterations in the tension, weight, and length of vibrating strings. All that we have to bear in mind, then, is, that *high pitch and rapid vibration* and *low pitch and slow vibration* are inseparable.

In tranquil normal breathing it is very difficult to detect any expiratory sound when listening to the chest wall; but by giving a very slight voluntary character to the expiratory act, a sound is at once heard. Now this sound will be always found to be of much *lower pitch* than the *inspiratory* sound, if the lungs and air-passages are healthy. And the question arises, Why is this? Do not the inspiratory tide and the expiratory tide pass through the very same tubes, through tubes of the same calibre, and therefore ought not each to produce a sound of the same pitch? That they do not produce sounds of the same pitch is due principally to the difference of *rate* in the two currents; no doubt, something is due to the altered temperature and volume of the expired air. The inspiratory current is much faster than the expiratory; and as



the same volume of air has to be drawn through the tubes in a shorter space of time, the vibrations set up are more rapid, and the pitch is proportionately raised. That such is the case is subject to the simplest proof, for we have only to make a patient snatch a sudden forcible breath, and so increase still further the rate of the inspiratory tide, and the pitch will rise proportionately. On the other hand, if a gentle slow inspiration is taken, the pitch will sink; and then by a sharp, forced expiration, the expiratory sound may be made to *rise* in pitch till it is even higher than the previous *inspiratory* sound; or the same experiment can be tried with a common bellows; the pitch of the sound produced by the rush of air through the nozzle can be raised or lowered by increasing or diminishing its rate.

If, then, in the natural state of things, the inspiratory sound is of higher pitch than the expiratory, it is clear that any alteration in this relationship must proceed from some change in the physical conditions, and ought to excite our attention.

It is evident that if the alteration of pitch in either of the sounds is due simply to an increase of rate in the current of air, other things being normal, the duration of the sound should diminish as the pitch increases; for a shorter time must be required to drive the same volume of air through the same tube at a rapid than at a slow rate. We see this exemplified when a chest, which is not highly elastic, is forcibly expanded, it recoils with suddenness and force; whereas a highly elastic chest expands freely with a less exertion of force, and recoils more slowly and gently. In the first case, the expiratory sound will be short and high pitched; in the second case, longer



and of lower pitch. Exactly these two states and their results are seen in the chests of girls and of boys, especially in the front and upper parts of them. I have often watched this when examining large numbers of boys and girls, one after the other, as I have lately had to do for the Albert Orphan Asylum.

Tell a boy to take a deep breath while you listen to the front of his chest, and you will find the succeeding *expiration* short and high-pitched. Tell a girl to do the same, and the expiration will be longer, softer and lower-pitched; and if, as sometimes happens, you find a boy with a chest like a girl, and a girl with one like a boy, the usual phenomena will be reversed.

I may mention, in passing, that this is, in my opinion, the explanation of the much discussed question of the cause of the perceptible expiratory sound in the earliest stages of tubercular deposits in the lungs. The resiliency of the lung is interfered with, the chest expansion is interfered with, and a shorter and more sudden recoil occurs, driving the air through the tubes with more force and at a greater rate than usual, and consequently raising the pitch of the expiratory sound, so as to make it audible. A certain rate of vibration is necessary to produce sound at all; and it may be that the rate in healthy normal expiration is not sufficient to be sonorous; whereas, the increase of rate to which I have referred, gives vibrations rapid enough to produce sound. I am not here speaking of that still greater change in the expiratory sound due to obstruction to the outward tide, which occurs in more advanced disease, but of a change in character which long precedes this, and is much more important to identify.

Well then, the first and simplest cause of a rise in pitch



in either of the respiratory sounds is an *increase in the rate of current*; and with this, if all else is normal, a decrease in duration must correspond.

But suppose the pitch of one of the sounds is raised, and at the same time the duration is not decreased, then there must be a diminution in the calibre of the tube through which the current passes. For, given, two equal volumes of air driven at the same rate through two orifices, either they will pass in the same period of time, or the sizes of the orifices must be different. Therefore, an expiratory sound which is both long and high pitched, must be due to a narrowing of the orifice through which it has to pass.

But at this point a difficulty occurs which for a long time puzzled me very much to explain.

The difficulty is this:—How can it happen that a change in the calibre of a respiratory tube, through which both the inspiratory and expiratory currents have to pass, should affect one of these currents more than the other? For instance,—if a bronchial tube is narrowed, why does it not raise the pitch of the inspiratory and expiratory sounds in the *same proportion*, and thus maintain the normal relation between their pitch? The explanation is that which I gave when speaking of backward pressure upon the air cells, viz., that the *moving powers* in the two cases do not remain the same. In normal respiration, the expiration is performed by the elastic recoil of the lung tissue, followed up by the elastic recoil of the chest wall; but directly there is an impediment placed in the way of the expiratory current, the expiratory muscular system is called into play, and thus a new element is added to *expiration* which is not added to *inspiration*. An additional



force of a new kind drives on the current in one case and not in the other. But with this new element of force comes that other most important one to which I have also referred, viz., that whereas all *inspiratory* efforts tend to expand the chest, and by taking off the superincumbent pressure, to dilate the air tubes, all *expiratory* efforts tend to compress the lungs, and consequently favour the contraction of the tubes, and this becomes especially the direction in which *muscular expiration* acts. The air cells which lie in the neighbourhood, and often on the walls of the air tubes, are pressed upon them. The operation of forced expiration, therefore, acts in the direction to favour and increase the *narrowing* of the expiratory current, and to increase the pitch of its sound, out of proportion to that of the inspiratory sound.

There is a very interesting exception to this rule, which serves to prove it. In spasmodic asthma, the *inspiratory* sound is raised in pitch more than the expiratory. This is due to the spasmodic contraction of the muscular fibres of the bronchi, excited by the attempts to inspire fresh air. They, in fact, offer a direct opposition to the normal inspiratory act, and almost paralyse it. They narrow the passage of the inspiratory current, and its sound is raised in pitch, in proportion to the rate at which the inspiratory efforts succeed in drawing it through the narrowed passages. But these spasmodic contractions do not, as a rule, offer the same obstruction to the outward tide; for, having been forced to yield to the inspiration, they allow the air to escape with less resistance, renewing the vigour of their contraction with every new attempt to draw fresh air through them.

We have then two valuable diagnostic physical signs plainly demonstrated:—



1. A high pitched long expiratory sound must mean contraction of the respiratory tubes independent of spasm.
2. An abnormally high-pitched *inspiratory* sound, not accompanied by a corresponding change in the expiratory sound, must mean spasmodic contraction of the air passages.

I will mention another clinical fact which, while it presents a third diagnostic physical sign, confirms the correctness of the other two. If a contraction takes place in a respiratory tube, which is *rigid* in its character and so situated that it cannot be favoured by either inspiration or expiration, the pitch of the inspiratory and expiratory sounds is alike affected by it. This may be best observed in affections of the larger air passages, such as rigid contractions of the openings of the larynx, compression of the trachea and bronchi, by tumours and the like.

Everything connected with the signs and symptoms of disease is beset with sources of fallacy, and these physical signs are no exceptions to the rule.

But the principal sources of fallacy in this case are not very difficult to eliminate. They consist in the influence of tongues and plugs of secretion adhering to the walls of the tubes which, by temporarily narrowing the passage at the point where the plug is lodged, and by the vibration of the free ends of the adherent tongues, give rise to deceptive elevations in the pitch of one or both of the respiratory sounds. It is evident that these are moveable causes; and therefore, before deciding upon the meaning of any alteration in the pitch of the respiratory sounds,



the patient should be made to clear the chest by breathing and coughing sharply several times; after which the chest should be examined again. If the alterations of pitch are not essentially changed in position or character by the coughing and sharp breathing, it is pretty certain that they are not due to such moveable causes as plugs and tongues of mucus.

In the case of spasmodic contractions, it occasionally happens that the spasm is so capricious that it suddenly gives way during inspiration, and closes upon the *expiratory* tide; but this reverse of the general rule is but an exceptional case, and is not likely to recur in several consecutive respirations.

Again, we must be on our guard against *transmitted sounds*. A sound or quality of sound generated in one part of the naso-pulmonary tract, may be transmitted through other portions of it. Even changes of sound produced in the posterior nares may be heard down the bronchi; and changes in the quality of sounds generated in the minute air tubes, may be heard in the larger passages. The most common of these occurrences, however, is, that qualities of sound produced by affections of the larynx, are transmitted down the trachea and bronchi, and may be mistaken for changes having their origin there. But really, this source of error may be avoided with tolerable ease. A sound must be most intense at the point of generation unless it is reinforced at some other part; and therefore, when a change is detected in the normal character of a sound, it should be followed with the stethoscope to its points of greatest intensity. It is true that we are again met by a source of fallacy in the different conducting powers of the parts intervening between the source of



sound and the chest surface; but this may be eliminated by resorting to the usual tests of the character of the conducting media. With a little care and a little tact, therefore, we may satisfactorily bring these changes of pitch to which I have referred, into the position of positive diagnostic signs of spasmodic or non-spasmodic narrowing of the respiratory tubes.

These points are illustrated by the following cases:—

*Case XIX.—Emphysema due to narrowed bronchi from repeated catarrh; high-pitched expiratory sound.*

Efforts at inspiration urgent. Elevation of chest marked. Resonance tympanitic. Hardly any inspiration audible. Expiration feeble, *high-toned*, and sibilant. Heart's sounds feeble and distant. Cardiac region much encroached upon by resonance. Fingers not clubbed, but inclined to die. Vertigo after fit of coughing. Loses blood by rectum. Very weak. Lame from childhood.

The cough began eight winters ago with suffocative catarrhal attacks in the morning, and continued to recur throughout each winter, getting better in the summer. The cough set in on the commencement of damp weather in autumn, with cold in the nose and chest, oppression at the upper part of chest and in the nose, preceding shortly the cough. It was not until after five winters that the breath was observed to be short, being first noticed during the occurrence of a bad cold, with pains in the chest. Since that time, viz., three years, the breath had always been short, though much worse in severe weather. His lameness was due to abscess after measles in infancy.

In this case the Emphysema is readily accounted for by bronchial obstruction. The very feeble inspiratory murmur, even on violent efforts at inspiration, showed the extent to which Emphysema had gone, preventing suction-



power; and the high tone and sibilus of the still feeble expiration, showed how small the calibre of the tubes had become. At the time of examination, treatment had been employed by which the cough was relieved; but there is no doubt that the absence of cough and bronchial irritation was only temporary.

*Case LXXV.\*—Spasmodic high-pitched Inspiration; relieved by Stramonium.*

A. B., aged nine years.—Complains of violent cough since getting a severe wetting and cold last summer. Nearly lost cough while in the country at the latter part of the summer, but became again much worse with first cold this winter. The cough comes on in fits.

His mother says he had a "convulsion" at sixteen months old, and has been a delicate boy ever since. She thinks his breath has been shorter than other boys ever since. He has been very subject to sneezing and violent running colds in the head and chest all his life. He did not suffer from cough till last summer, and since then his breath has been very much worse, and his heart palpitates very readily on exertion. Mother asthmatic, and her mother was the same; one sister is inclined to coughs. Chest deformed since infancy; left side flattened, right prominent. Resonance of right fuller than of left, neither super-resonant. Inspiration and expiration of equal length, both rhonchal and of about equal pitch, except that *at the end of a deep inspiration* the pitch rises very considerably. Forced expiration is very rough and rhonchal, but not raised much in pitch. These sounds exist all over chest, except in the lower lobes behind, where the sounds are nearly normal. Heart's sounds normal.

He was blistered, and took Sescarb. and Hydroch. of Ammonia, with Morph. and Ipecac. for a week. The fits of cough were then nearly well, but the breathing oppressed.



On examination the inspiratory sound was found to be very high pitched, the expiratory rough but of lower pitch. Stramonium was then ordered experimentally, to be taken one day and omitted the next, and so on. On his next visit he reported that on the stramonium day his breath was relieved, and got worse again the day he was without it. It was then taken regularly, and on examination no abnormal elevation of pitch in inspiratory sound remained, and he could take deep inspirations without cough. Discharged well in six weeks.

*Case LXXVI.\*—Spasmodic high-pitched Inspiration relieved by Stramonium.*

J. M., aged 44, baker. Ill twelve years. General elevation of chest. Shoulders rather rounded. No super-resonance back or front; in front, rather deficient of the two. Inspiration very high-pitched. Expiration rather long. Heart's sounds feeble. Twelve years ago, first attacked quite suddenly with "tightness" of breath, while in apparent good health. A cough came on at the same time. Similar attacks have recurred about every three months ever since. The attacks, he thinks, are brought on either by "over-heating the blood," or by "inhaling foul smells." They get worse and worse. Habits temperate; has smoked twenty-four years, but when he has an attack he cannot smoke. Has been a baker thirty-two years, but did not suffer from his chest till the sudden attack twelve years ago. No hereditary disease in the family.

Ordered ipecac. and squill pills, and counter-irritation, without relief. Then ordered ext. of stramonium,  $\frac{1}{4}$  gr. three times a day, and he reported that he had been "quite cured ever since he took it." On examination there was no short breath. No spasmodic attacks since taking stramonium. Expiration and inspiration nearly



normal; expiration rather long and harsh, and the inspiration no longer high-pitched.

I have examined the descriptions of the inspiratory and expiratory sounds in a number of cases, taken with no special reference to this question of pitch—taken, in fact, before I had thought much about it, and with other objects in view. In these cases, therefore, no mention of the pitch of the respiratory sounds would have been made unless it was very marked, so that alterations in this respect may have been present many times without being noticed. Nevertheless, I find that in cases of Emphysema and Bronchitis conjoined, the expiratory sound was prolonged in 69 per cent., and that it was, at the same time, described as *raised in pitch* in 74 per cent. of the cases in which it was prolonged.

But since I have paid attention to this subject, I have been able to point out to medical friends and pupils at this hospital, in how very large a proportion of cases of Emphysema the expiratory sound is *raised in pitch*; and also how often it is so changed, in cases of catarrh, which have not yet had time to produce distention of the air-cells.

I have also been able to demonstrate that relief from stramonium, nitre paper, datura tatula, and other means known to relax spasm of the air-tubes, may be safely predicted when the *inspiratory* instead of the expiratory is the sound of which the pitch is principally raised.

Now I must ask, in conclusion, what can explain this raised pitch of the expiratory sound in a case of Emphysema, unless it be narrowing of the calibre of the tubes through which the expiratory tide passes? It is not to



be explained by the susceptibility of Emphysematous persons to spasm of the tubes ; because, as I have already shown, in spasm it is the *inspiratory*, not the *expiratory* sound, which is principally affected.

It is contrary to all acoustic laws to suppose that the pitch would be raised by a current of air being driven through a *tube of normal calibre* by a force *defective* in power. In Emphysema there is this defect in power—there is loss of the power to drive the air out of the chest with rapidity. We find the air passing out slowly, and yet the pitch raised above that which is natural to it, when there is no defect in the driving force, and no increased length of time occupied in the transit. It can but be explained by the simple fact that the passage through which the air is driven is of diminished calibre.

I have now furnished you, as I promised to do, with some means of investigating the causes of Emphysema, which are not open to those objections which I pointed out as applying to post-mortem examinations. Whereas they are competent to forewarn us of an approaching enemy before it is too late to prevent a catastrophe, I wish to impress upon you the fact that, in cases of Emphysema, in which other sufficient causes for its production cannot be found, you will almost always be able to discover, by the means I have now explained, the existence of narrowing of the respiratory passages.

But before turning from this part of the subject, I ought to call your attention to a small but interesting class of cases, which might, if we were not prepared for them, lead us to doubt the correctness of our views as to the causation of Emphysema. I confess that they puzzled me at first.



The sort of case I refer to is this:—You clearly diagnose Emphysema. You search carefully into the history and find no rational cause for the disease, except obstructed tubes. You then examine the chest, confident that you will find signs of such obstruction; but you are disappointed—the expiratory sound is *soft and of low pitch*. It is clear, therefore, acoustically, that one of two things must be the case—either the tubes are of normal size, or, being narrowed, there is a remarkable loss of the driving power by which the current of air is forced through the narrowed tubes. Either of these conditions may, of course, give the same result—viz., a soft, low-pitched expiratory sound; and practically, either of these conditions may exist in a case of Emphysema produced by narrowed air-passages. We may have, in the class of cases to which I am now referring, either of these two complications:—

1. Emphysema produced by narrowed air-tubes—that narrowing still present, and yet the expiratory sound soft and of low pitch.

2. Emphysema produced by narrowed air-tubes—that narrowing no longer present, the expiratory sound soft and of low pitch.

The first of these complications occurs thus:—When thickened and narrowed tubes have produced Emphysema, that Emphysema may be still further and further increased by the obstruction to the outward tide, until the expiratory power is almost lost through excessive dilatation of the air-cells. The chest which, at a former period of the case, when expiratory power remained in sufficient force, would have driven the outward tide of air through the narrowed tubes at a *rate sufficient* to produce



a sound of high pitch, has now so far lost its power, that, except under extraordinary stimulus, it cannot do more than urge on a current so slow and feeble, that even in the narrowed tube it is incompetent to produce a sound of high pitch.

These cases can be cleared up, and their nature made out, by attention to the following points:—1st, the existence of excessive super-resonance; 2ndly, the *great length* of the soft, feeble, low-pitched expiratory sound; 3rdly, and especially, by making the patient give the deepest inspiration he can, and then the sharpest, hardest, sudden expiration he is capable of; and the increased force and rapidity thus given to the expiratory tide discloses the secret of the narrowed tubes, by eliciting their high-pitched sounds.

The second of these complications occurs thus:—When thickened and narrowed air-passages have produced Emphysema, either by the long continuance of the narrowed condition, or by the co-existence of other circumstances during the time of their greatest loss of calibre, the Emphysema may remain as the only vestige of their narrowed state; altered conditions of life—such as change of climate, successful treatment, long freedom from catarrh, and the like—having, in course of time, restored the air-passages to their normal calibre.

This is one of those cases to which I referred when speaking of the unsatisfactory character of post-mortem evidence, if not kept within its proper limits. No post-mortem evidence would here be found to tell that the Emphysema had been produced by narrowed tubes; and as the case would be just one in which the walls of the Emphysematous air-cells would be likely to have become



degenerated, it would be set down as a confirmatory case of the degeneration theory. The degeneration of the cell-walls—which had, in truth, taken place since their diseased distension had occurred—would be the only apparent cause, found after death, of that distension, and would be most probably set down as the cause.

Now the clinical history of such a case, examined into carefully during life, would most likely show that there had been a period at some past date when the patient suffered from those catarrhal affections of the nasopulmonary tract, which were sufficient to produce the thickened tubes, and hence the Emphysema.

The same clinical history would afford evidence of those altered conditions of life, and that altered condition of the patient, which had led to the removal of the nasopulmonary affection. I say, confidently, that the history would afford this evidence in the majority of instances, because I have found, in a somewhat large experience, that such has been the case when examples of this complication have come before me.

The following three cases illustrate the two sets of complications I have described :—

*Case LXXVII\*.—High-pitched expiratory sound. Narrowed primary air-passages. Emphysema beginning; good vis a tergo.*

D. E., aged 64, dealer. Short breath in winter for several winters, but never so short as this year. Cough began before short breath; has returned four or five winters. A loud high-pitched wheeze, with expiration audible across the room, which, on auscultation, is found to originate in the larynx. Chest not barrelled or prominent, or much raised in breathing; abdomen not



retracted. Front and back rather super-resonant in the upper half, and in these parts inspiratory sound is very short, expiratory sound very long, and of higher pitch than inspiratory. In the lower parts the inspiration is longer, and the expiration lower-pitched than inspiration.

*Case LXXVIII\*.—Extensive Emphysema. Narrowed tubes; vis a tergo diminished. Expiratory sound low-pitched till raised by forced sudden expiration.*

E. F., aged 44, coal dealer (formerly schoolmaster). Cough five winters. Suffers from acid dyspepsia. Chest elevation rather exaggerated, walls thin; resonance in excess all over chest, back and front. Inspiratory sound occasionally rhonchal; expiratory sound long, soft, and faint, during normal, quiet, breathing, of about the same pitch as inspiration; but on forced expiration the duration is still very long, but very *much raised in pitch*. These characteristics of inspiration and expiration are especially marked in the front and upper parts; in the back and lower parts expiration is coarse and not so high-pitched. Heart's space almost completely covered by resonant lung; epigastric pulsation.

Breath began to be short five years ago, after a cold and cough; no other illness at the time; previous health good. The attack of cold and cough was long and severe; never quite free from cough since, but every winter it has been much aggravated. The breath has varied, sometimes being free and at others short—always very short when the cough gets worse. East wind, frosts, fogs, are the things most inclined to make breath short. In winter the least exercise makes it short; not so in summer. Colds first fall upon the throat and chest. The things most inclined to give cold are perspiration, exposure to damp, change of clothing. Mother died healthy at eighty; father died at forty-two from Asthma brought on by succession of colds. Conditions of life not unfavourable.



*Case LXXIX\*.—Narrowing of Bronchi, producing great shortness of breath. High pitch of expiration removed by treatment. No Emphysema yet produced.*

C. R., age 55; married. Breath extremely short; cough severe and tearing. Says she had neither cough nor short breath till two years ago, when she was suddenly seized with pains in the chest and short breath, and had a severe attack of Bronchitis, which laid her up many weeks. This was in the winter; as the summer came the cough went; and the breath became quite natural. She is sure the breath was not short while she was free from cough in the summer. In last November (nine months ago) she caught cold, and the cough and short breathing returned, and they have continued getting rather worse than better all through the spring and summer, and now the breath is distressingly short, and cough very troublesome.

No super-resonance or undue prominence of chest; no dulness. Inspiration harsh and rhonchal; expiration higher-pitched than inspiration, and prolonged; heart's sounds feeble. She looks ill-kept.

Ordered three blisters in succession—one on each side of the chest, and one in the front. Ipecac. and comp. squill pill.—She reports at the end of a fortnight that the breath is very much relieved by the blisters, each blister seeming to make it better than the one before.

Conium inhalation; ipecac. squill and quinine. At end of another fortnight, "wonderfully improved." Can now walk as she has not done for months. Harshness of breath sounds diminished, and expiratory sounds much lower in pitch since treatment.

In Case LXXVII\* we find well marked signs of narrowed tubes, conjoined with Emphysema in an early stage; the expiratory force still good.



In Case LXXVIII\* we find the signs of excessive Emphysema, with a long, soft, feeble expiratory sound of *low pitch*; but this pitch *raised*, so as to disclose the existence of narrowed tubes, when the forced sudden expiratory test is applied. We may, in fact, regard this case as the representation of what Case LXXVII\* would become if it went on without amendment.

On the other hand, we find in Case LXXIX\* all the signs of narrowed tubes and impeded respiration, needing only time to develop Emphysema; but under the influence of active treatment the high-pitched expiratory sound characteristic of narrowed tubes disappeared, the normal pitch was restored, and with this a restoration of free respiration. Had this case gone on, therefore, till Emphysema had been produced before the narrowed tubes were cured, as in Case LXXVII\*, we should have had, when the patient was discharged with normal tubes, exactly the complications I have described under the second heading.

According to my promise in a former lecture, I have a few words to say in passing, concerning the vital objections to the degeneration, collapse, and inspiratory theories of the production of Emphysema.

The degeneration theory originated, I believe, in a discovery made by Mr. Rainey. In a lung which he examined after death he found some portions Emphysematous, and although the lung tissue in the immediate neighbourhood of these portions was perfectly healthy, the walls of the Emphysematous air-cells had undergone degeneration. Upon the basis of this, and a few other subsequent observations of a very limited character, it has been assumed that a degeneration taking place in the walls of the air-cells constitutes the primary



cause of Emphysema, the loss of normal resiliency caused by the degeneration leading to the gradual distension of the cells under the pressure of respiration. This theory, as you will see, does not interfere with the question as to whether it is during inspiration or during expiration that the pressure of air on the interior of the cells is exerted; and, supposing such degeneration to take place, Emphysema might easily be produced in the degenerated parts by the backward current of air during expiration. Therefore, there is no inconsistency in holding both the degeneration and the expiration theories.

The rather serious objection, however, to the degeneration theory is this, that there is no evidence at all that the required degeneration takes place, except as quite an occasional and exceptional occurrence. It is perfectly in accordance with the general pathology and clinical history of the degeneration of tissues to suppose that air-cells which have become over-distended, bloodless, and paralysed in function should, in course of time, become degenerated; and it is also very natural, though perfectly gratuitous, that a morbid anatomist, finding this change after death, should attribute the Emphysema to the degeneration instead of the degeneration to the Emphysema; because he has no possible means on the dead subject of determining the true order of succession in the morbid changes which he observes.

Treating of this theory Dr. Waters says: "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 conditions, yet that degeneration *does not* exist is



by no means determined by the failure of our present methods of investigation to demonstrate it" . . . He then goes on to say :—"I shall state briefly the circumstances which induce me to believe that Emphysema is the result of some degenerative process. . . . ."

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

"2. 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.

"3. The hereditary nature of the disease, as shown by the observations I have alluded to (Dr. Jackson's).

"4. The manner in which the disease is influenced by certain remedial measures, which are known to act beneficially on other diseases attended with degeneration (Iron and Strychnia)."

With regard to the first and second of these propositions, it is evident that, if Emphysema may be due to the long list of causes which we have already discussed ; and if the date of its production may in some cases be many years back, so that the history of the occurrences on which it depends may easily escape observation ; while in other cases it may be due to causes acting gradually and quietly, of which Dr. Waters does not take account ; and if affections of the primary air passages are to be included, as I have asserted, among its sufficient causes ; we have no need to resort to an imaginary state of degeneration to account for all the difficulties which Dr. Waters sets forth in the first and second propositions.



In answer to the third proposition (See Tables of Hereditary Transmission). If due to such causes as affections of the naso-pulmonary tract, dependent on diatheses the tendency to which is known to be hereditary, there is no need to resort to any other explanation of the asserted hereditary character of Emphysema. (The tables show that the hereditary tendency is greater in Bronchitis than in Emphysema).

In answer to the fourth.—Seeing that the remedies referred to by Dr. Waters as improving the condition of Emphysematous patients are Strychnia and Iron, which are known to be beneficial in muscular debility and anæmia, all the benefit they have been found by Dr. Waters to confer on the sufferers from Emphysema may be easily attributed to improved blood and muscular tone in the patients, enabling them better to contend with a wearing and weakening disease.

The next theory I propose to mention is that originated by Dr. Gairdner of Edinburgh (See *Edinburgh Monthly Journal*), an able and ingenious observer, whose suggestions are always worthy of careful consideration.

“It has been shown by the researches of Gairdner 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. His opinions may be summed up as follows:—Adopting the view that Emphysema is produced by the force of the *inspired* air acting upon 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." (Dr. Waters).

In this assumed relation of collapse and Emphysema, as cause and effect, there are two obvious sources of fallacy :—

1. The occurrence in the same lung of the two states, when examined after death, is presumptive evidence of their being both dependent upon some common cause, rather than of one being the cause of the other.

2. In order to prove that Emphysema is only an effect of collapse of lung, every case of Emphysema ought to present evidences of collapsed lung. Again, even supposing a collapsed lobule of lung to be competent to lead to Emphysema, as assumed by Dr. Gairdner, it clearly could not be a sufficient cause for more extensive Emphysema than would represent, by increase of bulk, the loss of bulk due to collapse ; and therefore would be an utterly insufficient explanation of a large number of the worst cases of lobar Emphysema.

I have suggested that probably the *inequality* of pressure upon the air-cells, caused by a portion of lung substance becoming suddenly diminished in bulk by collapse, may become a cause of local Emphysema in the adjoining vesicles when the expiratory act exerts its pressure on the lungs ; that, in fact, the air which under ordinary circumstances would be forced onward in the outward tide, finds so little support from the walls of the cells adjoining the collapsed lobule, that it distends them instead of passing outward.

But even supposing this operation to take place, there is no need to resort to the inspiratory theory to explain the Emphysema. It comes naturally under the operation



of expiration. A cell, the walls of which have lost their normal support, will be virtually in the same position, with regard to its liability to distension, as one which has an obstruction placed in the way of the free escape of its contents under pressure, and an undue force thereby exerted upon its inner wall.

But, although I am free to admit the possibility of Emphysema being so produced in the cells lying close to the collapsed part, I confess I am quite at a loss to see how portions of lung can become Emphysematous through the influence of collapse of other portions lying at a distance from them.

It is true that the Emphysematous patches have been seen lying side by side with the collapsed, but these are exceptional cases, and the contrary is the rule. Collapse of the lung is most frequent in its posterior and lower parts; Emphysema is most frequent at the apex, and along the margins of the lungs.

I think, then, as I suggested just now, that instead of taking for granted, that because these two states—collapse and Emphysema—are often found to co-exist, although Emphysema often exists without collapse, therefore they are related as cause and effect; we should rather turn to see if there is not in these cases a third phenomenon, which stands in the relation of *common cause to the other two*. Such a phenomenon does indeed exist in these cases, although we miss it altogether in a post-mortem examination. This phenomenon is a convulsive straining cough. No case of collapse of lung is likely to take place without the occurrence at the time it happens of a fit or fits of straining convulsive cough, and the cases in which collapse is likely to occur are just those in which



straining and convulsive cough is likely to be one of the features of the attack, whether collapse happen to take place or not. It is nothing but what we ought to expect, then, in accordance with what I have endeavoured to point out as the operation of the expiratory theory, that when we find collapse of portions of lung, we should also find other portions Emphysematous.

Of the Inspiratory theory there is no need to say much, seeing that I have shown, as I think satisfactorily, that it is during expiration that the strain is put upon the air-cells. The inspiratory theory has been marshalled under the great name of Laennec as its champion, and I am inclined to think that but for this prestige it would never have had many supporters. But it so happens that the support which Laennec gave to this theory is of such a character that it does in truth support the expiratory theory instead, for if applied to the inspiratory it will not bear a moment's consideration.

Laennec is supposed to have believed that "Emphysema is occasioned by an over-distension of the air-cells, from accumulation of air taking place in them in consequence of the obstructed condition of the bronchial tubes, the air being forced through them by each inspiration and not evacuated by expiration, so that it accumulates."—(Dr. Waters, p. 39.)

You observe here that Laennec provides the essential conditions for dilatation by the expiratory theory, viz., obstructed tubes, and a freer inspiratory tide, and a more obstructed outward tide; but the supposition necessary to make inspiration the dilating cause is palpably absurd, for if, as stated, the air were forced into the cells by inspiration, and not forced out of them by expiration, thus



allowing it to accumulate, it is clear that the cells would be rapidly distended beyond measure, and rupture, with interlobular Emphysema, would occur as a necessity. Even supposing the accumulation to be ever so slight at each inspiration, when we consider the number of inspirations every hour, and the consequent accumulation which would take place, it is clear that no lungs could remain unruptured for twenty-four hours. But the whole thing is brought back within the bounds of common sense when we recollect that, instead of the air being allowed to accumulate, clinical examination of the disease teaches us that the expiration is prolonged in proportion to the obstruction of the tubes, and that all the auxiliary expiratory powers are brought into requisition, because it cannot be permitted by the organism for one hour that the air admitted by inspiration should exceed its normal proportion to that discharged by expiration. And in this clinical fact we get the proof of the fallacy of the idea of accumulation, introduced to make it possible for the inspiratory act to cause Emphysema; and, at the same time, we find introduced the element necessary to produce Emphysema by expiration, viz., a backward pressure upon the interior of the cells.

I have now shown you that the expiratory theory, when understood as I have attempted to explain it, and when it is made to comprise all the causes of Emphysema which I have enumerated, has this among its other advantages, that it is competent to explain all the phenomena which have been brought as proofs of the correctness of other theories.

Now, I fear you may think that I have spent an unnecessary amount of time in discussing the question, whether



Emphysema is produced by the inspiratory or by the expiratory act, and I must hasten to show you why I place so much importance on this question, and have thought it worthy of so much consideration in relation to *Winter Cough*. It is because the views we entertain upon this point lie at the very root of all our practice, when we come to deal with *Winter Cough*. It is therefore of the greatest practical importance.

I have the more satisfaction in holding the view I have been trying to inculcate, because it is the one which gives us the most hope in the *treatment* of *Winter Cough*; which stimulates us to the most careful and far-sighted plans in the management of *Winter Cough*; and enables us to give our patients the most cheerful encouragement under their sufferings. And lastly, but by no means least, it is a view which dictates a *preventive* policy; which leads us to understand when we are dealing with germs which, if *un-nipped*, may develop into disastrous diseases, and leave behind the most serious vestiges, and when we are dealing with these vestiges, which may be increased in extent and severity if we do not remove every trace of the germs from which the original diseases sprang. (See Treatment).



## LECTURE IV.

CATARRH.—BRONCHITIS, ETC.—ILLUSTRATIVE CASES.—EXCEPTIONAL CASES OF WINTER COUGH.—DISEASE OF HEART.

I HAVE now to direct your attention to that large class of cases of Winter Cough in which no Emphysema has yet been produced.

The cases which I have placed in the third or Red Group of our clinical arrangement, in which we find evidences in the history, physical signs, and general symptoms of inflammation, congestion, and irritation of the naso-pulmonary mucous membrane, more or less permanent and chronic, and especially apt to be recurrent and to produce Winter Cough. It is only necessary to examine these cases with care to discover the reasons why Emphysema had not resulted from the catarrhal affections.

### ABSTRACT OF CASES.

*Case III.*—The bronchial affection was only of one year's date. There was evident bronchial obstruction, but no severe stress had been put upon the breathing; and, therefore, longer time was required to develop Emphysema.

*Case X.*—At the age of fifty, no considerable obstruction of the bronchi had become permanent, although the patient had been subject to cough from a boy; so much had he escaped, that the breath had only begun to be



short three years ago, commencing during an attack of pleuritic pain; and at the time of admission, his respiration was "free."

*Case XII.*—Spasmodic contraction of the bronchi occurred directly catarrh reached them; and this was always quickly relieved by free exudation from the mucous membrane, which, at the same time, removed tumidity of the membrane. Thus there was no considerable obstruction to the outward tide.

*Case XIII.*—The short breath was caused, in the first place, nineteen years ago, by dilated heart—the cough not occurring at all till seven years after, when the heart disease had gradually produced pulmonary congestion. The patient had led an inactive life, principally occupied with her needle, prevented by the heart affection from taking exertion, so that no great stress had been put upon the respiratory organs. The cough had not been severe, as a general rule; and on taking fresh cold, she got free exudation, and thus the bronchial membrane was easily relieved. The severe symptoms, indicating serious bronchial obstruction, with which she was admitted to hospital, were quite recent. These circumstances explain the absence of Emphysema.

Supposing the bronchial obstruction from which she was suffering on admission to continue, and Emphysema to result, there can be no doubt that this case seen some years later would be taken in the ordinary course as one of Emphysema with intercurrent Bronchitis and consequent dilatation of the heart. The peculiar family history—which shows that the mother, father, three brothers, and two sisters had suffered from "asthma,"—would be taken as striking evidence of the hereditary character of Emphysema. Had Emphysema existed, a fallacy might easily have arisen from the fact that short breath, dated from a severe confinement, in which a tumour in the



uterus interfered with natural labour. It might plausibly have been argued that Emphysema was produced by forcible expiratory efforts at that time. All of these versions are negatived by a cautious enquiry into the past history of the case; and it well illustrates the importance of taking such care as I have already advised before forming our judgments.

*Case XVI.* is very remarkable—no Emphysema existing after a cough of twenty years' duration. No interference with breathing occurred till seven years ago, and then it was probably due to exhaustion from Dysentery, coupled with a thin-walled heart. The breath had only been short at intervals, since that time, when suffering from colds. We are almost obliged to look at the physical signs in this case to explain the history. We find there that the *expiratory sound was normal*—the principal morbid sounds accompanying the inspiration, which was harsh, and accompanied by ronchus and high-toned sibilus in various parts; and in conjunction with these, it may be noted that the cough had been nearly dry, and almost confined to the paroxysms in the morning. Its character, therefore, was probably due to spasm of the bronchi—the interference being to the inward, not to the outward, tide.

*Case XVIII.*—A Winter Cough of ten years had not produced any serious amount of bronchial obstruction. There had been complete freedom during the summer months; and, probably, this accounted for the absence of permanent thickening of the mucous membrane.

*Case XXI.*—Attacks of simple bronchial catarrh, not very severe in degree, and speedily yielding to treatment (at least, it was so with the attack for which she was admitted), had recurred for eight winters, and quite disappeared in summer. No Emphysema had resulted at the time of admission, but the physical signs showed that the smaller bronchi were beginning to diminish in calibre.



It might be expected, therefore, that if this went on, Emphysema would sooner or later result; but at present there was not sufficient obstruction to the outward tide to produce it.

*Case XXV.*—The cough had existed throughout the greater part of life, but had only been severe during two years; and during that time the attacks had been characterised by bronchial spasm excited by contact of cold air; so that, at the age of fifty-three, no considerable change had taken place in the bronchial membrane, and the outward tide was not seriously obstructed.

*Case XXIX.* was a delicate boy of seventeen, who was becoming subject to attacks of Bronchitis, through exposure to vicissitudes of temperature. They were frequent, but evanescent. No bronchial obstruction had yet been produced, and no Emphysema. He was easily relieved by treatment; and, by changing his occupation, ceased to suffer from his bronchial attacks.

*Case XXXII.*—The cough had existed in winter during seven years, but there had been complete freedom from it during summer. The breath became short very soon after the cough first occurred, and came on during an attack of Dyspepsia. Both short breath and cough were excited by cold, but were of the spasmodic character; and the absence of Emphysema is accounted for by the inward current being more affected than the outward, and by the complete freedom from cough and Dyspnœa during summer.

*Case XXXVI.*—As there were no signs of interference with free respiration anywhere, except in a very circumscribed spot, and no history of any attack of disease of a more severe character than the present one, there was no reason for Emphysema to have been produced, although the cough was of twelve years' duration.

*Case XXXVII.*—The essential character of the res-



piration was, impediment to the *inward* current from bronchial spasm. There was also a slight amount of tumidity of the bronchial membrane, but not sufficient to present any considerable obstruction to expiration.

*Case XXXIX.*—This again was a case of evanescent spasmodic obstruction to inspiration, leading, by repeated attacks, to more and more irritability of the bronchial membrane. Although of fifteen years' standing, no Emphysema had resulted—the obstruction to the outward current being slight.

*Case XLIII.*—No obstruction was presented to the outward current—the inspiratory sound being alone affected. Although the cough had returned for fifteen winters, no Emphysema had resulted. The long and harsh inspiratory sound was due to bronchial spasm, superadded only two years ago.

*Case XLV.*—Although the man was accustomed to lifting weights and other active exertions, and had suffered from cough as long as he, at 54 years of age, could remember, no emphysema had resulted; and the explanation is found in the fact that the expiratory power was free.

*Case XLIX.*—At the time of examination the obstruction to the outward tide was considerable, and such as should, if continued, exert a dilating influence on the cells; but the resonance was normal, and the heart's space not encroached upon. The breath had been short since early childhood during good health, and had remained so ever since, yet there was no Emphysema to account for this. The heart-sounds were feeble, and the pulse the same, though the patient looked hearty and well. The feeble heart seems to be the only explanation for the long-continued short breath before the occurrence of cough. No cough occurred till seven years ago, when she was seventeen years old, and then only came with ordinary symptoms of cold, not apparently in any relation to the



old-standing short breath. It had only existed in winter, till twelve months before admission; during that twelve months it had been continuous. I am led to conclude that the physical signs, present on examination, were of recent origin,—the obstruction to expiration not having existed long enough to produce dilatation of the air-cells, this change probably taking place less readily in consequence of the good health of the patient.

*Case LIV.*—Had all the conditions necessary for the gradual production of Emphysema, but none of them had existed more than four years, the severe symptoms much less than this, therefore, time had not yet served to develop dilation of the cells.

*Case LX.*—Was only seen during the existence of catarrh, the expiration was long and rough at that time, the inspiration dry and harsh. There had been no short breath till six months before admission, and then only during fresh attacks of catarrh. It is fair to assume, therefore, that the bronchial obstruction was not a permanent condition, and hence that, although the patient had been subject to cough for twelve years, the freedom from Emphysema is explained by the want of sufficient and permanent bronchial obstruction to produce it. The cough had always occurred with cold, and disappeared when free from cold, so that the mucous membrane had had time to recover in the interim. Doubtless the repetition of such attacks over a long period of years, if neglected, would in time produce sufficient bronchial obstruction to lead to Emphysema.

From a careful examination of these cases, it is seen that the reasons why Emphysema had not occurred, were either that there was not sufficient obstruction to the outward tide, or that the obstruction which existed had been of only short duration, and time was yet wanting to develop the Emphysematous condition. Many of them



are, in fact, only incipient cases of Emphysema; while others teach us the important practical lesson, that so long as we can maintain a free expiratory power during chronic Bronchitis, or when we can so cut short attacks of bronchial affection as to restore the normal freedom of the outward tide in a very short time, we may avoid and prevent the occurrence of Emphysema. (See Treatment).

#### GROUP V.—EXCEPTIONAL CASES.

Although the great majority of cases of Winter Cough are either cases of Bronchitis or of Bronchitis and Emphysema conjoined, there is a certain number which do not belong to either of these classes, and require to be carefully distinguished in practice. I shall not, however, have time in these lectures to do more than briefly allude to the chief of these, leaving their further consideration to some future occasion.

1. Post-nasal catarrh. (See Appendix).
2. Follicular disease of the pharynx.
3. Superficial inflammation and serration of the edges of the soft palate.
4. Elongated uvula becoming relaxed and œdematous with every fresh attack of cold.
5. Chronic recurrent laryngeal catarrh.

These may each exist separately, but they are often found associated in a single case.

The first three may all be illustrated by Case LXXX.\*

4 and 5 may be illustrated by Cases LXXXI\* and LXXXII\*.



*Case LXXX\*.—Winter Cough from Affection of the  
Naso-pharyngeal Mucous Membrane.*

W. W., aged 17, clerk, August, 1845. At the age of twelve months had whooping-cough badly, lasting from June of one year to June of the next, after which it left. Ever since this with every cold he has had a cough, both summer and winter, of spasmodic character (occurring in fits). Breath never affected except just after a bad fit of coughing. Has been known as the best runner at his school, and runs up and down stairs without any short breath. The cough came on last winter with a cold, and continued into this summer, not leaving till he went to Walton-on-Naze. He lost it there, and it did not return until after he came home and got wet, when it came back, and it is now bad, especially at night, rousing him with a fit of coughing out of his sleep. Expectorates pretty freely every morning yellowish mucus. No signs of either Emphysema or Bronchitis, no cough-sounds in the chest, but hoarse cough-sounds and rough respiration in the larynx, fauces, and posterior nares. No morbid heart-sounds. Soft palate, ragged and red at edges, posterior wall of fauces tumid, mucous follicles red and swollen, nostrils very tumid and discharging watery mucus freely. Cough has a laryngeal twang, chest feels tight after coughing. Hereditary rheumatism in family, urine deposits uric acid and urates. For six years after living in the valley of Kingston-on-Thames, has been subject every autumn to formation of small boils and ulcers in the nostrils, where they continued to break out, and the nostrils remain tumid all winter, interfering considerably with the nasal passage, and making the breath feel very hot. Three years ago had a gathering in the meatus of left ear, since which there has been ringing and partial deafness. Left ear, tympanic extremity of meatus red and irritable, scar of small abscess; right ear very moist, and tympanum looks sodden. Rheumatic pains in right forefinger; inclined to contraction of flexor tendons; rheumatic



pains in shoulders. In this case the condition of the nasal and pharyngeal mucous membrane appears to be the cause of cough. The history, and super-resonant chest, &c., would accord with the existence of slight Emphysema, but there is no short breath and no altered breath-sounds. Ordered alkaline saline of ammonia, soda, potass, and citric acid, a lozenge-pill of morphia and liquorice, and counter-irritation to the neck and behind the ears. September 4.—Cough quite gone; ordered to continue alkalies, till rheumatic symptoms are gone, and paint throat with tinct. galls; syringe nose with weak infusion of galls. September 26.—No cough, throat looks nearly normal, nostrils still tumid. November.—Continues well, and the nostrils are now so much better that he considers them well, though I can see that the mucous membrane is still thickened.

*Case LXXXI.\* — Winter Cough from Pharyngeal and Tracheal Congestion.*

F. W., 56, winter of 1864, a stout, light haired man, formerly a sea-captain, conjunctivæ pale. Complains that for several winters he has had a bad cough, last year especially severe. It has now set in as usual with all the old characters. The cough is convulsive in character, with severe straining on the diaphragmatic attachments, often produces retching before it ceases, and leaves the chest, back and front, very sore. Not much expectoration. He is not otherwise ill. It always dates from a cold. Careful examination of the chest discovers all the sounds of percussion and auscultation to be normal. The cough-sounds do not reach the chest. The only abnormal sounds are high-pitched, dry, inspiratory, and expiratory, and, especially, cough-sounds in the larynx and top of the trachea. The uvula is long and tumid, whole pharyngeal mucous membrane tumid, and this condition extends into the larynx. Treatment directed to this state of mucous membrane, removed the cough.



*Case LXXXII.\*—Winter Cough from Chronic Thickening of the Laryngeal Mucous Membrane and Recurrent Laryngeal Congestion.*

H. W., aged 10, schoolboy, had "croup" two or three times when a baby, and has had cough off and on in winter ever since, generally with a croupy sound. This summer the cough did not leave as usual, but has continued all through; as soon as it begins to get a little better he seems to get fresh cold and it gets worse again. He has always been very ready to take cold, but the tendency increases. His mother says his breath is not short, but he says it has been shorter than other boys for some time; he cannot run upstairs so well as others, and gets "winded" sooner in running. He says he gets hoarse very easily, especially if the weather is at all damp. Damp aggravates his cough. No hereditary tendency to disease. Chest raised much in inspiration. Resonance normal. No abnormal sounds anywhere except in the neighbourhood of the larynx—traced by greatest intensity to larynx—in which both inspiration and expiration are harsh; expiration considerably higher pitched than inspiration, and expiration becomes stridulous in larynx during cough.

I of course exclude all cases of tubercle in the lungs, although, in their early stages, they are often associated with a Winter Cough which may be scarcely noticeable through the summer; thus it is but a passing and very transient phase in their history. It is, however, very important to bear in mind that a slight cough coming on with the winter for a second or third time may be a sign of the presence of tubercles in the lungs.

It is to be remembered that, as in some of these cases, the expiratory tide may become obstructed, and as all



are accompanied by cough, which may be more or less *convulsive*, they may any of them become complicated with Emphysema under certain circumstances.

#### DISEASES OF THE HEART.

It will be observed that I have said scarcely anything about Disease of the Heart, either as a cause or effect of Winter Cough. The reason is simply this: that as a cause it chiefly acts as a predisposer to Bronchitis; and that as an effect, although a very important one, it has no special relation to the points upon which I have principally dwelt; and our time does not permit me to enter upon a new department of the subject which could only be fairly treated at considerable length. Under the head of treatment, however, I may be able to find room for a few practical remarks with reference to cardiac complications.

#### COUGHS AND COLDS.

Having now cleared the way by the discussion of some of the most important difficulties connected with the relations between Bronchitis and Emphysema, let us return to the consideration of the lessons to be learnt from the facts which I have tabulated.

The first tables give the answers to the following questions relating to coughs and colds (See form in Appendix):—

1. What was the nature of the first attack of cough?
2. When free from cough since the first attack?
3. Is the cough aggravated by any other causes than fresh colds, and if so, what are they?
4. On catching cold, does it affect first the nose, throat, or chest?



*Nature of First Attack of Cough.*

TABLE III. cases). (See Index to	Cold with Bron- chitis.	Inflam- mation of Lungs.	Con- vulsive attack.	Spas- modic attack.	Whoop- ing Cough.	Came on gradu- ally.	First attack slight; 2nd se- vere ne- glected.	Severe Dry Cough.	Not noted, or not remem- bered.
White 33 Cases	27	1	0	1	1	2	0	0	1
Per Cent. ...	82	3		3	3	6			3
Red 18 Cases...	7	1	0	0	0	0	1	4	5
Per Cent. ...	39	6					6	22	27
Yellow 1 Case .	0	0	1	0	0	0	0	0	0
Per Cent. ...			100						
Blue 6 Cases ...	4	0	0	0	0	1	0	0	1
Per Cent. ...	67					16.5			16.5
Total 58 Cases	38	2	1	1	1	3	1	4	7
Approx.perCent.	66	3	2	2	2	5	2	7	12

Table III. shows the character of the first attack of cough in the different groups. In 66 per cent. of the fifty-eight cases it was described as an attack of cold with Bronchitis; and this was the case in no less than 82 per cent. of the white group. In 7 per cent. of the fifty-eight cases it was described as a severe "dry cough," which was most probably acute Bronchitis, especially as it occurred only in the cases showing signs of Bronchitis. In the blue group — in which there were signs of Emphysema, but not of Bronchitis—the first attack of cough was described as cold and Bronchitis in 67 per cent.



*When free from Cough since First Attack.*

TABLE IV. (See Index to Cases).	In Summer Weather.	When free from Cold.	Occasionally with no assignable cause.	Never.
White 33 Cases ...	15	3	0	16
Per Cent.....	45	9		48
Red 18 Cases .....	7	0	2	8
Per Cent....	39		11	44
Yellow 1 Case .....	0	0	0	1
Per Cent.....				100
Blue 6 Cases.....	4	0	0	2
Per Cent.....	67			33
Total 58 Cases .....	26	3	2	27
Approx. Per Cent.	45	5	3	47

Table IV. shows that of 40 cases of Emphysema 19 were free from Cough in summer weather, and 3 when not suffering under the effects of a cold. So that in 22 out of 40 cases, or about 55 per cent., Emphysema could exist unaccompanied by cough; for we know that Emphysema is not a complaint which could have appeared and disappeared, again and again, as the cough did.



*Is Cough aggravated by any other causes than Colds, and if so, what are they?*

TABLE V. (See Index to cases.)	No.	Change of weather.	Sudden change of temperature.	Moist Air.	Fog.	De-ranged Stomach.	Excitement of exertion.	Inappreciable causes.
White 33 Cases .	23	4	1	0	0	1	2	2
Per Cent.....	70	12	3			3	6	6
Red 18 Cases ...	14	0	0	0	0	2	0	2
Per Cent.....	78					11		11
Yellow 1 Case ...	0	0	0	0	1	0	0	0
Per Cent.....					100			
Blue 6 Cases.....	5	0	0	1	0	0	0	0
Per Cent.....	83			17				
Total 58 Cases ...	42	4	1	1	1	3	2	4
Approx. Per Cent.	72	7	2	2	2	5	3	7

Table V. shows that the cough was not aggravated by any other causes than fresh colds, in 70 per cent. of the White Group, in 78 per cent. of the Red Group, and in 83 per cent. of the Blue Group. It appears from this table, therefore, that the cough in Emphysema becomes a prominent feature in proportion as the patient is subjected to attacks of catarrh. This is especially seen in the blue cases, in which the cough was aggravated only on the occurrence of fresh cold, in the largest proportion of any of the groups, and it will be further illustrated as we go on.



*Whether on catching Cold it affects first Nose, Throat,  
or Chest.*

TABLE VI. (See Index to cases.)	Nose.	Throat.	Chest.	Nose and throat simultaneously.	Nose and chest simultaneously.	Throat and chest simultaneously.
White 33 Cases	13	0	16	0	0	4
Per Cent.....	39		49			12
Red 18 Cases...	6	3	4	2	0	3
Per Cent.....	33	17	22	11		17
Yellow 1 Case	0	0	1	0	0	0
Per Cent.....			100			
Blue 6 Cases ..	1	0	4	0	1	0
Per Cent.....	17		67		17	
Total 58 Cases	20	3	25	2	1	7
Approx. per ct.	34	5	43	3	2	12

The influence of catarrh is further shown by Table VI., which describes the mode of attack assumed by fresh colds in the different groups. In thirty-four per cent. of the whole series of fifty-eight cases, the colds began in the nose, and in three per cent. in the nose and throat simultaneously, so that in about thirty-eight per cent. the colds spoken of in the other tables, as accounting so largely for the aggravation of both the short breathing and the cough, began as attacks of ordinary nasal catarrh.



The proportions in which the colds commenced at different parts of the mucous tract in the several groups are worthy of remark. Thus, in the cases of Bronchitis without Emphysema, the nose and throat take precedence in about seventy-eight per cent.; the chest in only twenty-two per cent. In the cases of Bronchitis and Emphysema conjoined, the nose and throat take the lead in about fifty-one per cent.; the chest in forty per cent. In the cases of Emphysema in which Bronchitis was only an occasional affection, the nose takes the lead in thirty-three per cent.; the throat not at all; but the chest takes precedence in sixty-seven per cent.; and in the one case unassociated with Bronchitis, neither nose nor throat is mentioned; on the occurrence of cold it affected the chest at once, but this patient was not subject to colds, they only occurred on getting wet.

In forty-five per cent. of the fifty-eight tabulated cases, the cough "*left in summer weather.*" This was the case in about forty-nine per cent. of the cases of Bronchitis and Emphysema (blue and white), and in about thirty-nine per cent. of the cases of Bronchitis without Emphysema.

But the "*cough never left*" in forty-seven per cent. of the fifty-eight cases, and this happened in about forty-six per cent. of the cases of Bronchitis and Emphysema (blue and white), and in about forty-four per cent. of the cases of Bronchitis with no Emphysema (red).

Of the forty-nine per cent. of the cases of Emphysema and Bronchitis in which the cough "*left in summer weather,*" the largest proportion belonged to the *blue group*, in which Bronchitis was not a constant attendant, being absent at the time of examination.

From these figures it would seem that it is the Bron-



chitis and not the Emphysema which is the essential cause of the cough, and that the presence or absence of cough under different meteorological conditions is dependent upon changes in the state of the Bronchial affection, not of the Emphysema.

We are led by these considerations to enquire whether there is anything in the circumstances connected with the two sets of cases—the forty-five per cent. in which the cough *left in summer weather*, and the forty-seven per cent. in which it *never left*—to account for this important difference. I have therefore placed in *separate* tables (VII. and VIII.) the principal facts relating to these points in the two sets of cases. Examining into the twenty-six cases in which cough left in summer, it is found that the colds to which the returns of cough were attributed *fell first upon the chest* in about sixty-three per cent. of the cases of Emphysema and Bronchitis (blue and white), and in only about fourteen per cent. of the cases of Bronchitis with no Emphysema (red); whereas the colds *first affected the nose or throat* in eighty-five per cent. of the cases of Bronchitis and *no* Emphysema, and in only thirty-seven per cent. of the cases of Bronchitis *with* Emphysema.

In the twenty-six cases in which *the cough never left*, the colds *fell first upon the chest* in about sixty-one per cent. of the cases of Bronchitis and Emphysema (blue and white), and in about sixty-two per cent. of the cases of Bronchitis and *no* Emphysema (red); whereas colds *fell first upon the nose or throat* in about thirty-seven per cent. of the red cases, and in about thirty-nine per cent. of the blue and white cases.

On comparing these two sets of facts, we see a gradual



assimilation between the cases of Bronchitis without Emphysema and those with Emphysema in respect to the way in which they are affected by colds, and that this takes place in proportion as the Bronchitis becomes a deeply-seated and abiding affection.

We find that when the cough left in summer weather, that is, when the Bronchitis was cleared up by this meteorological change, it was in the Emphysematous cases that the colds fell first upon the chest in the largest proportion. In those subject to Bronchitis alone the colds fell first upon the nose and throat, and only affected the chest by creeping down the mucous tract; so that a person whose bronchial affection is complicated with Emphysema is in a much worse position, with regard to the effects of future exposure to colds, than one in whom it has not become so complicated. He takes cold in the chest at once, and there is no opportunity, therefore, for saving his chest by arresting the cold before it has reached it, as may be done in the case of colds beginning in the head and travelling down towards the bronchi.

But when the cough never left, that is, when the Bronchitis was not cleared up by a change to warm weather, a different average prevailed; the colds which aggravated the cough fell first upon the chest in nearly the same proportion in the cases of Bronchitis without Emphysema as in those with Emphysema, viz., red, sixty-two per cent., blue and white, sixty-one per cent.

The necessary condition, then, to determine the colds to fall at once upon the chest, is the existence in the Bronchi of a semi-inflamed condition—a condition of imperfectly cured Bronchitis—which keeps up a constant irritability and susceptibility to a renewal of the half-



cured disease immediately an exciting cause is presented. The only connection we are able to trace between the presence of Emphysema and the tendency of colds to fall first upon the chest, lies in the fact that, in such cases, the Bronchitis has extended deeply into the bronchial ramifications, and has become so permanently lodged in the smaller bronchi that it remains but partially cured there, even when the balmy air of summer soothes the larger bronchi and upper parts of the naso-pulmonary tract with which it comes into direct contact, and thus allays irritation and relieves the cough.

With respect to the tendency of fresh colds to fall first upon the chest, the cases of Emphysema and Bronchitis in which the cough left in summer weather, are like the cases of Bronchitis *without* Emphysema, in which the cough never left, and like the cases of Emphysema and Bronchitis in which also the cough never left. In all three cases the colds fell first upon the chest in nearly the same proportions, viz., sixty-three per cent., sixty-two per cent., sixty-one per cent.

It is, then—and this is the important practical deduction—to the gradual creeping down of Catarrh from the nose to the throat, from the throat to the bronchi, and from the large bronchi to the smaller ramifications, and to the greater and greater difficulty of eradicating the disease as it gets deeper and deeper, that our special attention must be directed. Thus we see the importance of leaving no means untried which can give a chance of radically and permanently removing every lurking trace of Bronchitis, before we let a patient consider himself safely cured, in every attack of this disease that comes before us for treatment. (See Treatment.)



We must not be satisfied because the cough is better, or even if it has left; for we have seen that this may be the case, under favourable atmospheric influences, and yet enough disease lurk behind to bring it back with the first return of an irritating cause. We must only be satisfied when, by all our methods of examining and testing the respiratory powers, we cannot detect a lingering trace of disease in the bronchial mucous membrane, and even then we have to consider by what means we can maintain this condition till it has become habitual. (See Treatment.)

I must remind you here of all that I have already attempted to prove respecting the causation of Emphysema, and of the important position which neglected Bronchitis and obstructed bronchial tubes hold in the category of causes.

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TABLE VII. — *Free from Cough in Summer*

No. & Colour of Case.	Whether Breath has been other than short since it began to be short.	State of Breath between attacks of Cough.	Whether Cough is aggravated by any other causes than colds.
2 White.	It was always short.	Short.	Going from hot to cold, or cold to hot.
14 White.	Yes; in Summer.	Very short.	No.
16 Red.	Yes; only short at long intervals.	No answer.	When stomach is deranged.
18 Red.	Worse in foggy weather.	Easy.	Yes; frequently cough without fresh cold.
19 Blue.	Much worse in bad weather.	No answer.	No.
21 Red.	Yes; only short with cough or excitement.	Good, unless excited.	No.
22 White.	No; always short in hot rooms, during exercise, or stooping.	Generally good.	No.
23 White.	Yes; in summer.	Short in winter.	No.
26 White.	Yes.	Middling.	No.
28 White.	Yes; scarcely short in summer.	Very short at times.	No, except over-exertion.
29 Red.	Yes; only short with cough.	Good.	Yes; heavy suppers and indigestible food.
32 Red.	Yes; only short in winter.	Rather short in winter.	No.
35 White.	No; worse with colds.	Short.	No.
39 Red.	Yes; in summer.	No answer.	No.
40 White.	Yes, at times, in clear fine weather.	Rather short; varies with weather.	No.
41 White.	Yes; in mid-day in summer.	Short.	No.
43 Red.	No.	Quite well till 2 years ago.	No.
46 White.	Yes, if free from cold.	Much less short.	No.
48 Blue.	No; better and worse.	Not short till after last attack.	Moist atmosphere.
51 White.	No.	Varies.	Fog.
52 White.	Yes; in summer.	Well.	No.
53 Blue.	Yes; in summer.	Rather short of late.	No.
56 White.	Yes; in summer.	Short.	Excitement.
57 Blue.	No; always bad from dust, or fast walking.	Very short.	No.
58 White.	No; worse with every cold.	Short.	No.
59 White.	Yes; from March to September.	Very short from September to March.	No.

*Cases in which Cough*

Colds felt first on Chest in 63 per cent. Blue and White.

" " " 14 " Red.



*Weather in 26 of 58 Cases=45 per Cent.*

What gives cold most easily, what most often.	Symptoms of attack of Cold which leaves Cough.	Whether Cold affects first nose, throat, chest.
Damp and draughts.	Strangling pains in throat and wind-pipe.	1st throat, 2nd chest.
Wet weather.	Cold and chills.	Chest and throat.
East winds, and draughts while hot.	Cold in nose and eyes going to chest in 24 hours.	1 nose and eyes, 2 throat, 3 chest.
Draughts and night air.	Rigors.	1 throat, 2 nose and head, 3 chest.
Damp weather.	Oppression at front of chest, soon followed by running at nose and cough.	Nose and chest simultaneously.
Change from heat to cold.	Tightness at chest and throat, with no cold in head.	Chest.
Draughts.	Running at nose, and sense of cold in nostrils on inhaling.	1 nose and head, 2 chest.
Sudden change of temperature; getting wet.	Hoarseness.	1 chest.
Wet feet.	Rigors.	1 chest.
Cold winds.	Soreness at chest.	Chest; head seldom affected.
Damp feet and night air.	Dry huskiness in throat.	Nose, head, and throat at once.
Night air.	Irritation in throat.	1 throat, 2 chest, seldom any cold in head or nose.
Sudden change of temperature; draughts.	Cold and running in eyes and head.	Sometimes running cold in head first, sometimes chest.
Draughts and damp.	Running cold.	1 nose, 2 throat, 3 chest.
Damp and fog.	Rigors.	1 nose and eyes, 2 chest.
Wet and draughts.	Pain at chest and difficult breathing.	Chest.
Sharp winds; wet feet.	Cold in head.	1 nose and eyes, 2 chest.
Damp and fog; change of weather.	Tightness at chest, short breath, much phlegm.	Throat and chest.
Very slight changes of temperature.	Running cold, with fever and soreness in chest and throat.	1 nose and head, 2 throat and chest.
Cold, foggy, or night air.	Soreness in chest.	Chest.
Change of weather.	Cold in head.	Nose and head.
Wet feet or draughts.	Languid feeling.	Chest.
Wet.	Rigors.	Chest.
Damp and draughts.	Cold in nose and chest.	1 nose, 2 chest.
Fog.	Stuffing of nose and chest, short breath, no running cold.	1 nose, 2 chest.
Currents of cold air, and many other causes.	Cold in nose and head, weakness, pains in limbs, low spirits.	Nose and head.

*left in Summer Weather.*

Colds felt first on Nose or Throat in 37 per cent. Blue and White.

" " " " 85 " Red.



TABLE VIII. — *Never free from Cough*

No. and Colour of Case.	Whether Breath has been other than short since it began to be short.	Things most inclined to make Breath short.	Date and nature of first attack of Cough; how long before or after short Breath.
1 Blue.	Yes, in summer.	Accumulation of phlegm.	7 yrs. ago, 3 yrs. before short breath, came by degrees, worse year by year.
5 White.	No.	Getting cold.	Bronchitis, bad attack, left short breath.
7 Blue.	Yes, in summer.	Fog and fast walking.	14 yrs. ago, 7 yrs. before short breath, pleurisy and rheumatic fever 7 yrs. ago, 2nd attack.
9 White.	No, short with every cough.	Cough.	14 yrs. ago, attack of inflammation of lungs, cough and short breath since.
10 Red.	No.	Damp weather.	Tickling cough since a boy.
12 Red.	No.	No answer.	7 yrs. ago, 1 week before short breath, severe cold.
13 Red.	Yes, for a month or two in summer.	Atmospheric changes.	12 yrs. ago severe congestion of lungs. Breath short 7 yrs. before, after confinement.
15 White.	No.	Colds.	20 yrs. ago, tickling cough in throat, produced short breath.
17 White.	No.	Exertion.	2 months ago, 5 yrs. after, short breath, bad cold and stoppage in nose.
24 White.	No.	Running or fast walking.	3 yrs. ago, 1 year after short breath, spitting large quantities of phlegm.
25 Red.	No, but worse at times.	Chills.	A little cough since childhood, worse last 2 yrs. with paroxysm of dyspnoea.
27 White.	Yes, rarely.	Cold.	10 yrs. ago, same time as short breath, from exposure.
31 White.	Yes, only short with cough.	Cough only.	Cough from infancy, but at 3 bronchitis, worse since; now 19.
33 White.	Yes, between attacks of cold.	Colds.	Always subject to coughs, bad attack with cold 2 yrs. ago, before short breath.
34 White.	No, not for 3 or 4 years.	Slightest exertion.	12 yrs. ago just before short breath, slight at first, worse and worse.
36 Red.	Not short.	No answer.	12 yrs. ago, violent cold.
38 White.	Yes, in summer.	Colds.	5 yrs. ago cold, inflammation of lungs, followed by short breath.
42 White.	No, but better and worse.	Change of weather—dust.	28 yrs. ago, same time as short breath, tightness at chest.
44 White.	No.	Exertion.	9 yrs. ago, same time as short breath, violent strangling cough.
45 Red.	No.	Cold weather.	Severe cough all life.
47 White.	Yes, in summer.	Exertion.	9 yrs. ago, same time as breath.
49 Red.	Yes, in summer.	Fog and Damp.	7 yrs. ago, cold and cough.
50 White.	It was better for 5 yrs. after first got bad 20 yrs. ago.	Exertion.	20 yrs. ago, same time as short breath, cold and cough.
54 Red.	No, but only bad when the cough comes.	Colds & coughs susceptibility increases.	4 yrs. ago, same time as short breath, very severe cold and cough.
55 White.	No.	Colds.	4 yrs. ago, same time as short breath, severe cold.
62 White.	No.	Colds.	Always subject to colds and coughs, worse last 6 months.

*Cases in which*

Colds felt first on Chest is 61 per cent. Blue and White.

" " " 62 " Red.



Whether Cough is aggravated by other causes than colds.	What gives Cold most easily, what most often?	Symptoms of attack of Cold which leaves cough.	Whether Cold affects first nose, throat, or chest.
No.	Changes in weather, especially foggy and cold weather.	Chilliness.	Chest.
No.	Getting wet.	Pain at chest.	Chest.
No.	Wet feet, draughts.	Rigor.	Chest.
Yes, change of weather.	Damp feet, draughts.	Sneezing and cramp in chest.	Nose and head.
No.	Change of weather.	Weakness & languor.	Chest and throat.
No.	Change of weather.	Tightness at chest.	Head and nose.
No; had severe influenza 29 yrs. ago.	Perspiration after slight exertion.	Pains in chest and back.	Violent running of nose and hoarseness in throat.
No.	Wet and fog.		Chest.
No.	No answer.	No answer.	1 nose, 2 chest, not subject to colds.
No.	Wet feet, cold winds.	Copious expectoration.	Chest
No.	Damp feet, exposure to weather.	The slightest cold leaves a cough.	Chest.
Yes, dense foggy air will bring it on in night if weather changes.	Draughts.	Drowsiness and headache.	Nose and head.
Yes, at times very bad, without cold.	Damp, especially N.E. wind, nothing like damp.	Pains in side and shivering.	1 chest, 2 throat, 3 head.
No.	Change of weather, draughts.	Slightest cold gives running at nose, sneezing, and cough.	Most often nose first, sometimes not.
Yes, at times without apparent cause.	Change of weather.	Difficulty of breathing.	Chest and throat, not nose.
No.	Wet and cold.	Cold in chest.	1 throat, 2 chest, not head.
No.	Heats and colds, change of weather.	Cold in chest.	Chest.
No.	Change of weather.	Shortness of breath, increased by cold.	Chest.
No.	Damp, fog.	Influenza cold, and when running stops cough comes on.	1 nose, 2 head, 3 chest.
No.	Damp weather.	Short breath.	Chest.
Wet and fog—clear most relieves.	Cold wind.	Headache and tightness at chest.	Chest.
Always worse in winter.	Damp and fog.	Pains betw. shoulders	Chest.
No.	Fog and damp, overheating.	Pain in chest and back	Chest.
No.	Change of weather.	Running at nose, sneezing, soreness at throat and chest.	1 nose, but soon goes to chest.
No.	Wet feet, draughts.	Bleeding from nose.	1 nose, 2 chest.
Exertion.	Draughts.	Colds.	1 nose and head, 2 chest.

ugh never left.

Colds felt first on Nose or Throat is 39 per cent. Blue and White.

" " " " 37 " Red.



## LECTURE V.

SHORT BREATHING.—EXTRACTS FROM CASES.—RESUMÉ.—CONDITIONS  
OF LIFE.—GENEALOGY.

PASSING from the questions relating to the coughs and colds we come to those which concern the short breathing—

1. Has the breath been other than short since it began to be short, and if so, when and why?

2. What are the things most inclined to make the breath short?

*Whether breath has been other than short since it began to be short.*

TABLE IX.	Yes, in sum- mer.	Yes, be- tween attacks of cough.	Only short after cough- ing.	Only short occa- sionally	No, but better and worse.	No.	Not short.
White 33 Cases .....	9	3	0	0	0	21	0
Per Cent. ....	27	9				64	
Red 18 Cases .....	4	0	2	3	0	7	1
Per Cent.....	22		11	17		37	6
Yellow 1 Case .....	0	0	0	0	0	1	0
Per Cent.....						100	
Blue 6 Cases.....	4	0	0	0	2	0	0
Per Cent.....	67				33		
Total 58 Cases .....	17	3	2	3	3	29	1
Approx. per Cent. ..	29	5	3	5	5	50	2



*Things most inclined to make Breath short.*

TABLE X.	Exer- tion.	Change of wea- ther.	Cold wea- ther.	Damp or Fog.	Dust or hot rooms.	Cough only.	Getting cold.	Phlegm accumu- lating.	Not short.
White 33 Cases	9	2	0	9	2	2	9	0	0
Per Cent. ..	27	6		27	6	6	27		
Red 18 Cases	4	2	4	3	0	0	4	0	1
Per Cent.....	22	11	22	17			22		6
Yellow 1 Case	1	0	0	0	0	0	0	0	0
Per Cent. ...	100								
Blue 6 Cases...	3	0	0	2	0	0	0	1	0
Per Cent. ...	50			33				17	
Total 58 Cases	17	4	4	14	2	2	13	1	1
Approx. per Ct.	30	7	7	24	3	3	22	2	2

Table IX. shows that out of 40 cases of Emphysema, 13 stated that they had been free from short breath "in summer weather," and 3 "between the attacks of cough." So that in 16 out of 40 cases, or 40 per cent., Emphysema appears to have existed either without necessitating shortness of breath; or, at least, without a sufficient amount of shortness of breathing to be observable by the patient.

On further examining Table IX. we find that, whereas the short breathing was so much relieved by summer weather, as to be said by the patients not then to exist; in 29 per cent. of 58 cases, the short breathing was



not so relieved, but remained permanent summer and winter, in 51 per cent.

The relief by summer weather occurred in the different groups of cases in the following proportions, viz. :—

Blue group	.	.	.	67 per cent.
White „	.	.	.	27 „
Red „	.	.	.	22 „

It appears, then, that the greatest per centage of relief to short breathing by summer weather occurred in the blue group—the cases in which there was history of previous Bronchitis, but none present at the time of examination, in which, therefore, Bronchitis was an occasional but not a constant attendant; and this occurred in the same proportion, viz., 67 per cent., as that in which the same group of cases were relieved of cough by summer weather; and Table X. shows that “fog and damp,” the common exciters of Catarrh, were the things most inclined to make the breath short in 33 per cent. of these same cases. It is probable, therefore, that the short breath and the cough were both due to the same state, viz., Bronchitis, which we have already found from the other cases to have been the essential cause of cough. But we find (Table IX.) that in 7 cases, or 37 per cent., of the Red Group, in which Bronchitis existed with no Emphysema, the breath never ceased to be short; and in 3 of the same group, or 17 per cent., the breath was “only short occasionally.” Let us examine the abstracts of the cases, therefore, to see if there are any circumstances which can explain these three different conditions of breath in the same group of cases, viz. :—

Breath relieved by summer weather	.	.	.	22 per cent.
„ never relieved	.	.	.	37 „
„ only occasionally short	.	.	.	17 „



We can have no difficulty in understanding that the short breath of Bronchitis should be relieved by the soothing influence of warm summer air upon the mucous membrane. It will not be necessary, therefore, to go through these cases; but that it should *not* be so relieved needs some explanation; and on examining the following Extracts from the cases, we find the probable solutions of the difficulty:—

In Case III. the cough had commenced about two and a half years ago, breath had been short at the time, and both had got better afterwards; but since twelve months the Bronchitis and short breath had not been relieved, a severe attack having occurred at that time, which, having been neglected, had crept down into the finer tubes; and, although the breath was never other than short during the last year, both that and the cough were aggravated by all unfavourable changes of weather. Short breath was due, therefore, to the *deep seat of the bronchial affection*, and to the consequent persistence of congestion and tumidity of the mucous membrane.

In Case X. although the cough had existed for about forty winters, the breath had not been short more than three or four years, dating from a more than ordinarily severe cold, with pains in the chest, during which it was evident that the bronchial affection *had crept deeper down*, and that a permanently thickened condition of the mucous membrane had been established.

In Case XII. the permanent short breath was due to the deep-seated character of the Bronchitis. The patient was subject to aggravations of dyspnœa, due to spasmodic closure of the bronchi when irritated by fresh accessions of cold.

In Case XXV. the breath was “always short, but specially so at times.” Although he had had a cough



all his life in winter, the breath had only been getting short five years, and had especially been worse the last two years, during which time the cough had been worse and more abiding, and he had become subject to paroxysms of dyspnœa. There was a question in this case as to the existence of a fatty heart, which might account for the paroxysmal dyspnœa,\*but the permanent short breath was evidently due to slight but abiding *deep-seated Bronchitis*.

In Case XLIII. there had been winter cough for sixteen years, but the breath had only become permanently short for two years, and in this, as in the other cases, its permanence was explained by the Bronchitis having *taken a deeper seat* than heretofore since a more than usually severe cold two years ago.

In Case LIV. the breath had always been rather short, but not *bad* unless the cough was troublesome. The cough had never left since it began in a very severe attack of Bronchitis. Every cold had aggravated it, and these colds became more and more frequent, showing an abiding condition of tumidity and irritability of the mucous membrane extending into the smaller tubes. That the patient had not yet become Emphysematous was, probably, due simply to want of time for such a state to be developed, her habits being quiet and sedentary, and thus not putting much strain upon the breathing.

In Case XLV. the cough had existed many years—"all his life"—in winter, but the short breath dated from Influenza ten years ago, when the Bronchitis got a deeper seat, and left such a susceptibility of the mucous membrane to Catarrh, that, although the breath always got better if the cough did, fresh colds were so easily excited that he never was free, and latterly a tendency to spasmodic contraction of the tubes had been set up as a complication.

In all of these cases, then, we find the explanation of the want of relief to the short breath in the same causes as



in the want of relief to the cough, viz., *the deep seat of the bronchial affection*, and its aggravated character, the mucous membrane having lost its power to recover under the soothing influence of summer weather. Except in those cases in which the tendency to spasmodic action was added, there was no reason to attribute the permanent short breath to any other cause than *permanent diminution in the calibre of the bronchi* by abiding deep-seated Bronchitis.

But now, what shall we say of the cases in which the short breath was only an occasional occurrence? Can we find in them why it was short on these special occasions? They were Cases XVI., XXI., XXXVII.

In Case XVI. the cough had existed twenty years; for some time it was only a winter cough, but latterly it had not altogether left in summer. But the breath was not perceptibly short, except on some special occasions when either unusually cold weather produced temporary tumidity of the naso-pulmonary mucous membrane, or when some cause of unusual physical exhaustion caused a failure of the action of a somewhat thin-walled heart. The worst instance of short breathing had been brought on by a severe attack of dysentery following great mental anxiety.

In Case XXI. the breath was "not short unless the heart was made to palpitate" by severe fits of coughing, by excitement, or hurried exercise. The patient's general health was delicate, and she was anæmic.

In Case XXXVII. the breath was "only short at intervals, but gets more frequently so." The description of the symptoms shows that they were spasmodic attacks excited by fogs and damp weather.

These, then, were exceptional cases, in which the



bronchial affection was not sufficiently severe to produce short breath, but in which some superadded conditions—as anæmic palpitation, tendency to spasmodic asthma, and a thin-walled heart—subjected the patients to attacks of dyspnœa under certain combinations of circumstances.

We come back, then, to the leading point of importance—THAT WHEN THE BREATH IS SHORT IN BRONCHITIS, IT IS FROM DIMINISHED CALIBRE OF THE NASO-PULMONARY AIR PASSAGES. When it is only short in winter, it is because this condition is still susceptible of relief by summer weather. When it is permanently short, it is because the affection of the naso-pulmonary tract has crept down into the deeper recesses of the lungs, and become a more severe and abiding affection. And, putting all these sets of cases together, we see that these same conditions of the naso-pulmonary tract are the principal causes of variations in the degree and in the persistency of the short breathing, whether the Bronchitis is complicated with Emphysema or whether it is not. In a word—that although Emphysema is unquestionably a cause of short breath, and, in the most severe cases of the disease, a very serious cause, yet that bronchial disease—diminished calibre of the naso-pulmonary passages—is the condition which claims our most important consideration. Emphysema is but a vestige of which diminished calibre of the air-cells is the commonest cause. (See Treatment.)

#### CONDITIONS OF LIFE.

The influence of the conditions of life upon Bronchitis and Emphysema is partly illustrated in Tables XI., XII., XIII., XIV., XV. Table XI. shows that 43



per cent. of the 58 patients smoked tobacco, and Table XII. shows that in these twenty-five persons who smoked, it "eased the cough" in 4 per cent., "aggravated it" in 16 per cent., "promoted expectoration" in 24 per cent., and "had no effect either upon the cough or the short breathing" in 56 per cent.

In the majority of cases, then, it had no effect, and in the rest the effects were pretty equally divided between good and bad, with a slight preponderance on the side of good, except in the case uncomplicated with

WHETHER TOBACCO WAS SMOKED.			EFFECTS OF TOBACCO ON THE COUGH AND BREATH IN THE 25 CASES IN WHICH IT WAS SMOKED.				
TABLE XI. (See Index to Cases).	Yes.	No.	TABLE XII.	Eases cough.	Aggra- vates cough.	Promotes expecto- ration.	No effect.
White 33 Cases	16	17	White 16 Cases	1	1	4	10
Per Cent. ...	48	52	Per Cent. ...	6	6	25	63
Red 18 Cases...	5	13	Red 5 Cases ..	0	1	1	3
Per Cent.....	28	72	Per Cent.....		20	20	60
Yellow 1 Case	1	0	Yellow 1 Case	0	1	0	0
Per Cent. ...	100		Per Cent.....		100		
Blue 6 Cases ...	3	3	Blue 3 Cases ...	0	1	1	1
Per Cent. ...	50	50	Per Cent.....		33	33	33
Total 58 Cases	25	33	Total 25 Cases	1	4	6	14
Approx. per Ct.	43	57	Approx. per Ct.	4	16	24	56



Bronchitis, and in that the only effect was to aggravate the cough. So far, then, as these cases are concerned, there is no reason to connect tobacco-smoking with them at all in the relation of a cause of the diseases; its effects were so slightly marked that it would appear to be a thing which may be safely left to the patient's taste whether it be used or not. It may be worth while to observe here, that it makes a great difference in the effect upon patients' coughs and breathing, whether they smoke the tobacco themselves, and thus get the soothing properties with the carbonaceous smoke, or

*The Quantity of Fermented Liquors taken habitually.*

TABLE XIII. (See Index to Cases).	None.	Moderate.	Excessive.
White 33 Cases .....	6	25	2
Per Cent. ....	18	76	6
Red 18 Cases .....	3	13	2
Per Cent. ....	17	72	11
Yellow 1 Case.....	0	1	0
Per Cent. ....		100	
Blue 6 Cases .. ....	3	3	0
Per Cent. ....	50	50	
Total 58 Cases .....	12	42	4
Approx. Per Cent. ...	21	72	7



only remain in the presence of others who are smoking, and thus get little else than the carbonaceous smoke. Many who can smoke with pleasure are much irritated in their coughs and distressed in breathing by the smoke of others.

Table XIII. shows that 72 per cent. of the 58 patients took fermented liquors in moderation, 7 per cent. took them to excess, and 21 per cent. took none at all. From these results it does not appear that there is any special relation between the influence of alcohol and the diseases in question.

From Table XIV. it is seen that 98 per cent. of the 58

*Whether Living is regular and Spirits good or bad.*

TABLE XIV. (See Index to Cases).	Habits of Life.		Spirits.	
	Living regular.	Living irregular.	Spirits good.	Spirits bad.
White 33 Cases...	33	0	19	14
Per Cent. ....	100		58	42
Red 18 Cases ...	17	1	17	1
Per Cent. ....	94	6	94	6
Yellow 1 Case ...	1	0	0	1
Per Cent. ....	100			100
Blue 6 Cases .....	6	0	1	5
Per Cent. ....	100		17	83
Total 58 Cases ...	57	1	37	21
Approx. per Cent.	98	2	64	36



patients lived regular lives. The details of the cases showed that they all lived upon ordinary mixed diet of meat, vegetables, and bread; that their hours for meals and for work presented nothing worthy of special remark; and that the descriptions given of the rooms in which they lived by day or by night did not contain any particulars of importance.

The right-hand half of Table XIV. shows the habitual state of the spirits in the several groups of cases, and it may claim a word in this place before passing to the next table. In the 58 cases the spirits were good in 64 per cent. and bad in 36 per cent. But a very marked difference is seen in the different groups; thus, in the cases of Emphysema, with history of only occasional attacks of Bronchitis, and no present symptoms of it (Blue), the spirits were bad in 83 per cent.; whereas in the cases of Bronchitis conjoined with Emphysema (White) they were bad in only 42 per cent.; and in those of Bronchitis uncomplicated with Emphysema (Red) they were bad in only 6 per cent. In the one case of Emphysema, unassociated with Bronchitis, past or present, they were bad. It would seem, therefore, that in proportion as the Emphysema predominated over the Bronchial affection, the spirits were depressed. This may have been due to the fact that the symptoms of Bronchitis are more apt to get better and worse, and thus to render the patient's sufferings less monotonous than in Emphysema.

Table XV. shows the occupations of the 58 hospital patients whose cases were tabulated, and as a correct statement of facts it may be of use when added to other tables of a similar kind; but as the 58 patients followed 27 different kinds of occupation, and no more than 7 followed



one of them, and that was domestic, the next highest number being 6, which was needlework, I do not think any conclusions as to the effect of occupation upon Winter Cough can be drawn from these numbers alone. They must be considered rather as representing the proportions of the different classes of poor persons who seek relief at public charities. For influence of occupation see "Transactions of National Association for Promoting Social Science," and special treatises on the subject.

TABLE XV. (See Index to Cases).		OCCUPATION.																										
		Domesic.	Shoemaker.	Smith.	Driver.	Hostler.	Shopman or Woman.	Clerk.	Sempstress.	Engineer.	Laundress.	Baker.	Brasscutter.	Plumber.	Goldsmith.	Carpenter.	Sewing Machinist.	Missionary.	Messenger.	Policeman.	Artificial Flo-rist; no arsenic	Bookworker.	Collector.	Cooper.	Distiller.	Boxmaker.	Tailor.	Brushmaker.
White .....	4	3	1	3	1	4	1	5	3	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	2
Red .....	2	0	0	0	0	0	1	1	0	0	0	0	0	0	0	1	2	1	1	1	1	3	1	1	1	1	0	0
Yellow .....	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
Blue .....	1	1	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1	0
Total .....	7	4	1	4	1	4	3	6	3	1	1	1	1	1	2	2	1	1	1	2	3	1	1	1	1	2	2	2

## CLIMATIC INFLUENCES.

We come now to a much more important class of influences than any of the conditions of life at present mentioned, viz., such as may be generally denominated climatic. These we find scattered through most of the tables, and they may be enumerated as follows:—summer weather, change of weather, cold weather, damp and fog or moist air, sudden changes of temperature, draughts of cold air, cold winds, wet feet, getting wet.



To take these seriatim, and collect some accounts of their influences from the tables:—

1. *Summer Weather* produced freedom from cough in 45 per cent. of the 58 cases; relieved the short breath in 29 per cent.
2. *Change of Weather* was the thing most inclined to make the breath short in 7 per cent. of the 58 cases; aggravated the cough in 7 per cent.
3. *Cold Weather* was the thing most inclined to make the breath short in 7 per cent. of the 58 cases.
4. *Damp and Fog or Moist Air\** were the things most inclined to make the breath short in 24 per cent. of the 58 cases; aggravated the cough in 4 per cent.; were the most potent causes of fresh colds in 19 per cent.
5. *Sudden changes of Temperature and Draughts of Cold Air* aggravated the cough in 2 per cent. of the 58 cases; were the most potent causes of fresh colds in about 37 per cent.
6. *Cold Winds* were the most potent causes of fresh colds in 10 per cent. of the 58 cases.
7. *Wet Feet and Getting Wet* were the most potent causes of fresh colds in 31 per cent. of the 58 cases.

We have already seen from Tables V. and X. that fresh colds were the only causes which aggravated the cough in 72 per cent. of the 58 cases, and that they were the especial causes of short breath in 22 per cent.; and Table XVI. shows what are the most frequent

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\* It is worthy of note that fog, damp, and moist air are almost invariably found together, producing similar effects, as though the moisture of the foggy air were the injurious element.



and potent causes of these fresh colds, to which so much mischief is attributed. They are wet feet in 17 per cent. of the fifty-eight cases, getting wet in 14 per cent., cold winds in 10 per cent., draughts of cold air in 16 per cent., fog and damp in 19 per cent., sudden changes of temperature in 21 per cent.

*What gives Cold most easily.*

TABLE XVI. (See Index to Cases).	Wet Feet.	Getting Wet.	Cold Winds.	Draughts of Cold Air.	Fog & Damp.	Night Air and Draughts of Cold Air.	Sudden Changes of Temperature.
White 33 Cases.....	5	5	3	6	7	0	7
Per cent.* .....	15	15	9	19	21		21
Red 18 Cases.....	3	2	3	0	3	2	5
Per cent. ....	17	11	17		17	11	28
Yellow 1 Case† .....		1					
Per cent. ....		100					
Blue 6 Cases .....	2	0	0	3	1	0	0
Per cent. ....	33			50	17		
Total 58 Cases .....	10	8	6	9	11	2	12
Approximate per cent.	17	14	10	16	19	3	21

\* In Five Cases "Draughts of Cold Air" was joined with Wet or Wet Feet.

† Not subject to Colds.

A conclusion of a practical kind is very forcibly brought out by these results, viz., That persons suffering from Winter Cough might have hoped for immense relief from their sufferings through such protections from these very simple climatic influences as were to have been provided by the proposed "Crystal Sanatorium." (See Treatment.)



## GENEALOGY.

Our next subject is the tendency of Emphysema and Bronchitis to become hereditary, and to affect collaterals. With some difficulty, I have succeeded in constructing tables which I believe present the facts in their true light; and I venture to suggest that the adoption of a similar form of table by other observers when registering family history will be found convenient, and calculated to prevent mistakes as to the real influence of the hereditary and collateral tendencies of diseases.

It is necessary here to point out that the words "Asthma" and "Asthmatic," when used in reference to family history, are to be understood in their broadest popular sense; that being the nearest to the truth that can be obtained when depending for the accounts of past diseases upon the reports of non-medical persons, which must usually be the case. In these tables, then, "Asthma" must be considered to mean cough and short breath of a chronic kind, from whatever cause proceeding, except that the greatest care was exercised in making the enquiries, to include no cases under this head that could better be classed as "Consumption" or "Heart disease," for each of which a separate heading was provided.

The cases in which both parents were affected with the same disease have been kept distinct from those in which only one was affected, and every individual is marked according to sex. The brothers and sisters (collaterals) of the patient are marked as individuals according to sex, and also distinguished according to the case to which they belonged.

The total numbers of collaterals in each case are also distinguished according to sex. The sex of the patients



is also stated. It can therefore be seen at once in how many cases one or both parents were affected by the diseases mentioned; in how many cases one or more male or female collaterals were affected; and in how many cases one or both parents, and one or more male or female collaterals, *escaped* the diseases. And it can also be seen how many individuals of either sex—including the patient, the parents, and the collaterals—were affected by the diseases tabulated.

Table XVII. gives the details of the thirty-three cases of Emphysema and Bronchitis conjoined (White).

Table XVIII. gives the details of the eighteen cases of Bronchitis without Emphysema (Red), and Table XIX. those of the one case of Emphysema with neither present Bronchitis nor history of its previous occurrence (Yellow).

Table XX. gives the details of the six cases of Emphysema with history of previous Bronchitis, but no signs of present Bronchitis (Blue).

In these four Tables the details of each group may be seen separately.

Table XXI. comprises in one table a summary of the details as affecting the whole fifty-eight cases.

The facts contained in these tables are too numerous and complicated to be all discussed or even enumerated now, but I may mention the following as among the results which may be most readily obtained from them:—

1. In the 58 cases of Winter Cough, one or both parents were asthmatic in 29 per cent.

Neither parent was asthmatic in 71 per cent.

2. In the 52 of these cases in which there were collaterals, from 2 to 3 collaterals were asthmatic in 21 per cent.

No collateral was asthmatic in 79 per cent.



TABLE XVII.—*Family History in 33 Cases.*

WHITE.		Asthma (popularly so called).				Total Number of Collaterals in each Case.			
Sex of Patient.	Number of Case.	Parents.		Collaterals.		M.	F.		
		One. M. F.	Both. M. & F.	M.	F.				
F	2	1		...		1	0		
F	5		...	...		3	1		
M	9		...	...		4	3		
M	14		...	2		2	2		
M	15		...	...		1	1		
F	17		...	...		1	4		
M	22		...	...		3	3		
M	23		1 1		1	0	1		
M	24		...	...		1	1		
M	26		...	...		2	4		
M	27		...	...		0	1		
F	28	1		1	4	3	5		
M	30		...	...		4	5		
F	31		.	...		3	0		
M	33		...	...		0	0		
F	34		...	...		3	2		
F	35	1			2	1	5		
M	38		...	...		3	3		
F	40		...	1		4	2		
M	41		...	...		0	0		
M	42		...	...		1	4		
F	44		...	...		1	4		
M	46		...	...		2	0		
M	47		1 1	1	1	3	3		
M	50		...	...		0	0		
F	51		...	...		3	4		
F	52		...	...		2	0		
M	55		...	...		1	1		
M	56		...	...		2	4		
F	58		...	...		3	5		
M	59	1		...		11	9		
M	61		1	...		3	4		
M	62		...	...		0	0		
Persons .....		3	2	2	2	5	8	61	81
Cases .....		5		2		6		29	



TABLE XVIII.—*Family History in Eighteen Cases.*

Red.		Asthma (popularly so called).						Total Number of Collaterals in each Case.	
Sex of Patient.	Number of Case.	Parents.				Collaterals.		M.	F.
		One. M.	F.	Both. M.	F.	M.	F.		
F	3		...			...		2	1
M	10		...			...		0	0
F	12		...			...		6	7
F	13			1	1	1	3	2	5
M	16	1				..		1	2
M	18		...			6		6	3
F	21		...			...		4	3
M	25		...			1		4	2
M	29		...			...		3	2
M	32		..			...		3	6
F	36		...			...		1	2
M	37		...			...		6	2
M	39	1					2	4	2
F	43			1	1	...		0	2
M	45		1			...		1	4
F	49	1				...		11	0
F	54		...			...		3	7
F	60		...			..		1	3
Persons . . .		2	2	2	2	8	5	58	53
Cases . . . .		4		2		4		17	

TABLE XIX.—*Family History in One Case.*

Yellow.					
M	20	...		...	11 2
Persons . . .		...		...	11 2
Cases . . . .		...		...	1



TABLE XX.—*Family History in Six Cases.*

Blue.		Asthma (popularly so called).				Total Number of Collaterals in each Case.	
Sex of Patient.	Number of Case.	Parents.		Collateral.		M.	F.
		One. M.	Both. F. M. & F.	M.	F.		
F	1	1		...		3	2
M	7	1		4		4	2
M	19	1		...		7	5
M	48	1		...		0	0
M	53		...	...		2	4
M	57		...	...		1	1
Persons . . . .		3	1	4		17	14
Cases . . . .		4		1		5	

TABLE XXI.—*Summary of the Family History of 58 Cases of Winter Cough. For particulars of each Group (White, Red, Yellow, Blue) see other Tables.*

DISEASE.	Winter Cough.						Asthma (popularly so called).					
Description.	Patients. Total.		Parents. Total.		Collaterals. Total.		Parents. One. Both.				Collaterals	
Sex .....	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Persons ...	36	22	58	58	147	150	8	5	4	4	17	13
Cases .....	58		58		52 *		13		4		11	

\* No collaterals in six cases out of fifty-eight.

NOTE.—The total number of individuals concerned in this history is 471, of whom 241 were males, and 230 females.



3. In the 39 cases of Emphysema, with signs of present Bronchitis, or history of previous Bronchitis (White and Blue), one or both parents were asthmatic in 28 per cent.
4. In the 34 of these 39 cases in which there were collaterals, 2 collaterals were asthmatic in 21 per cent.
5. In the 18 cases of Bronchitis without Emphysema one or both parents were asthmatic in 33 per cent.
6. In the 17 of these cases in which there were collaterals, 3 collaterals were asthmatic in 24 per cent.

Or if, instead of calculating the percentage per number of cases, we calculate it per number of individuals concerned, we have the following results:—

TABLE XXII.—*Asthmatic tendency in 455 persons.\**

GROUPS OF CASES.	CLASS OF PERSONS.	Total number of Persons.	Number of Asthmatics	Per centage of Asthmatics to total number of Persons.
White and Blue Groups combined (Bronchitis and Emphysema)...	Patients ... 39 Parents ... 78 Collaterals 173	290	69	About 27 per Cent.
Red Group ... .. (Bronchitis and no Emphysema)	Patients ... 18 Parents ... 36 Collaterals 111	165	39	About 24 per Cent.
Totals .....	.....	455	108	About 24 per Cent.

\* The 16 persons included in the Yellow Group are omitted.



Of the 78 parents concerned in the White and Blue Groups only 13 were asthmatic, or nearly 17 per cent.

Of the 36 parents concerned in the Red Group 8 were asthmatic, or 25 per cent.

So far, therefore, as we can be guided by these cases, we are led to the conclusion that it is the Bronchitis, and not the Emphysema, which is the hereditary complaint; for we find, when we take the total number of persons concerned, including the patients themselves, their parents, and their brothers and sisters, that the percentage of asthmatics is nearly the same in the cases in which no Emphysema existed, as in those in which it was combined with the Bronchitis. And when we take the total number of parents concerned in each group, we find that the proportion of asthmatics is only about 17 per cent. in the cases of Emphysema; whereas, it is 25 per cent. in those of Bronchitis uncomplicated with Emphysema.

I am quite aware that it would require many hundreds of cases to afford statistics which would be perfectly reliable; but so far as they can be trusted, these results are very interesting. They accord with what our clinical experience and our knowledge of the etiology of diseases leads us to expect. We know that it is especially diathetic states which are apt to be hereditary; as for example, Gout, Rheumatism, Syphilis, Scrofula. And we know also how frequently these diatheses give a special tendency to Bronchitis and other catarrhal affections of the naso-pulmonary mucous membrane. It is, then, only, what we ought to expect, that complaints which are especially apt to occur in hereditary diatheses should prove, on statistical enquiry, to run in families. (See Treatment.)



You are aware that the commonly received opinion is, that Emphysema is the hereditary disease, and that in a very marked degree. Thus, Dr. Fuller says:—"Of 43 Emphysematous persons whom I examined, with a view to this enquiry, 26, or, in other words, 60·4 per cent., acknowledged an hereditary taint." "A very similar result has been obtained by Dr. Jackson, jun., of Boston, U.S., who reports that no less than 18 out of 28 Emphysematous persons (or about 64 per cent.) had either a father or mother, or both, afflicted with Emphysema; whereas, 3 only out of 50 non-Emphysematous persons (or 6 per cent.) sprang from an Emphysematous stock." (Note to p. 296, "On Diseases of the Chest.")

There is room in both of these statements for grave error. We have no evidence as to how it was decided that the parents were Emphysematous; and, knowing as I do the difficulty of getting exact information as to the nature of the diseases from which parents and other relatives of patients have suffered, I have little doubt that the nearest approach to the truth which either Dr. Fuller or Dr. Jackson was able to attain was, that the parents suffered from *some form of chronic Asthma*. It would have been impossible to be nearer the mark than this by anything less than a careful physical examination of the parents themselves. Now the popular expressions "Asthma" and "Asthmatic" include chronic Bronchitis, cardiac Asthma, and spasmodic Asthma, as well as Emphysema. By careful enquiry, spasmodic and cardiac Asthma may, perhaps, be eliminated; but I am convinced, not only from my experience with patients, but from the examination (at the "Briton," and "National Union" Life Offices) of many thousands of



applicants for life assurance, who are very closely catechised on these points, that what is popularly called "Asthma," must be taken to include chronic Bronchitis and Emphysema.

These statistics, therefore, which we are *supposed to have in evidence of the hereditary nature of Emphysema*, must be taken with considerable caution. They must, at least, be made to apply as much to the hereditary transmission of a tendency to Bronchitis as to that of Emphysema.

In my own cases I have shown that the family history of Asthma was about the same in cases of chronic Bronchitis without Emphysema as in those with Emphysema, while the per centage of Asthmatic *parents* was much the largest in cases without Emphysema.

A careful consideration of all the evidence on the subject, leads me to the opinion that there is a tendency in Bronchitis to be transmitted from parent to offspring, and to affect collaterals, and that this may be explained, as I have already suggested, by the fact that various diathetic states, known to be hereditary and to run in families, predispose to affections of the naso-pulmonary mucous membrane.

If, then, the tendency to Bronchitis and Catarrh is hereditary, and if Catarrh and Bronchitis are common causes of Emphysema, it is easy to see how naturally the mistake has arisen of concluding that Emphysema itself is hereditary.

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## LECTURE VI.

### TREATMENT.

WE now come to the all-important question of treatment. What are we to do when a case of Winter Cough comes before us in practice? All that I have told you, and attempted to make clear in the foregoing lectures, has had a definite connection in my mind with this question, and I shall have very much failed in carrying out my intentions if I have not put you in a favourable position to answer the question for yourselves.

Let us consider, then, what it is that we have to treat. First and most prominently thrust on our attention is the Winter Cough. That is clear enough, and it must, I think, be equally clear to us all that, if we direct our attacks upon the Cough, as though that were the disease, we shall make a fatal mistake, and shall most certainly be disappointed in our hopes of doing any permanent good.

What we have to treat, then, in the large majority of cases of Winter Cough is a combination of some or all of the conditions which I have already discussed, viz. :—

1. Dilated right heart.
2. Collapsed lung.
3. Emphysema.
4. Thickened naso-pulmonary mucous membrane, with narrowing of the air-passages.



5. Catarrh of the naso-pulmonary mucous membrane, of greater or less extent.
6. An undue susceptibility of the naso-pulmonary mucous tract.
7. Local and general conditions favouring or producing susceptibility of the mucous membrane.
8. Cough and short breath ; symptoms of the existence of the conditions already enumerated.

1. Dilated right heart. This is a secondary affection, produced by the persistence or frequent repetition of obstructed pulmonary circulation. It is a complication so sure to occur in a protracted and neglected case, and, once established, it exercises so important an influence over its future course, that we ought never to forget the tendency to its occurrence. During the whole course of any case of Winter Cough which comes under our care, we must remember congestion of the right side of the heart as a thing to be continually guarded against. If no organic change has yet occurred in the organ, we have to remember this as a thing to be prevented ; if the organ is already hypertrophied or dilated, or the tricuspid valve has already ceased to prevent regurgitation, we have to remember that all these conditions will be aggravated or kept in check in proportion as we guard against congestion of the heart. We have to remember that congestion of the right heart is the ever present cause of Dropsy, and that this miserable complication of a Winter Cough will come and go as we permit or prevent stasis of the pulmonary circulation. And I need not remind you of the damaged liver, and the damaged kidneys which, in time, result from the neglect of retrograde venous congestion.



It would be impossible in this lecture to enter into the details of the treatment of dilated heart. Let me simply warn you, in passing, that in every case of Winter Cough danger-signals must be placed upon the pulmonary circulation, the right heart, and the great veins.

2. Next on our list stands Collapsed Lung. In the course of a case of Bronchitis, Whooping-cough, or any other affection of the air passages, whenever a portion of lung is suddenly deprived of the power to be inflated, by the presence of a plug of secretion sucked into its main air passage, a set of symptoms is produced, the severity and importance of which will vary with the obstinacy with which the obstructing plug resists the attempts at its removal, and with the extent of lung-substance cut off from the pneumatic circulation.

Small portions of lung are frequently being temporarily blocked-off in this way during the fits of coughing which attend naso-pulmonary affections; but the plugs are so quickly removed and the admission of air restored, that, in the majority of instances, no damage remains. But every now and then it happens that the plug fails to be removed. It is just sufficiently dislodged by expiratory efforts to allow the air-cells behind it to be emptied, but is borne back into its place on the front of the inspiratory tide, and fixed in the threshold of a portion of collapsed and useless lung tissue.

Under any circumstances, however small the portion of lung thus put in peril, whether the plug is eventually removed or not, a very distressing convulsive cough is set up, which, as I have already stated, may cause Emphysematous distention of the air-cells; and when an important branch of the bronchi is in question, and a large portion



of lung tissue at stake, an unmistakable and most alarming set of symptoms attend the accident. It is not my purpose, however, to treat here of these symptoms further than to remind you that, in this way, the occurrence of collapse of portions of lung may happen to constitute an important feature in a case of Winter Cough. But what we have to consider here is the existence of a portion of lung, thus damaged and useless, in its position as one of the possible accompaniments of a Winter Cough, and to what extent, if at all, it need influence our treatment.

Now, in this capacity it may soon be disposed of. First, you have to take care when making your physical examination that you do not mistake it for a portion of hepatized lung, and thus waste your own and your patient's time in treating it as such; and, in the next place, having made up your mind as to its nature and the probable length of time it has existed, there are two courses to be followed:—First, if it is at all recent, cautiously to endeavour by rational measures, which will be chiefly gymnastic, to restore it to a permeable condition; and, second, if it is clearly of old standing, to let it alone. So great is the power of the organism to compensate such damages, that considerable portions of lung can be deprived of function without producing more than temporary distress.

But what I wish particularly to impress with regard to the discovery of a portion of collapsed lung is this,—to bear clearly in mind the mode of its production, to remember that what has happened once may happen again to the same patient,—that every time these violent convulsive coughs are set up, you run the chance of having a fresh set of air-cells deprived of their elasticity



by the over-distention of their walls during the expiratory shock,—and that the cause of all this is the naso-pulmonary Catarrh by which the plug was produced that stopped up the tube. Of this Catarrh I shall speak hereafter.

3. Emphysema. I have shown you in former lectures that Emphysema may come either before or after the first history of Winter Cough, although in the large majority of instances it comes after the cough. I have shown that Emphysema, *per se*, cannot be considered as the cause of the cough, because the Emphysema may exist without the cough, and the cough may exist just as much without as with the Emphysema. The only way in which the Emphysema can be considered as a cause of cough, is by its predisposing to affections by which the cough is produced. Practically, it rarely happens that Emphysema exists for any length of time without some circumstances arising which bring on a cough.

I have shown you that when the commencement of Emphysema has preceded the commencement of Winter Cough it may be—nearly always—attributed to one of the following causes:—

1. Violent expulsive acts.
2. Violent exertions of force in lifting or carrying weights.
3. Convulsive fits of cough; as in Croup, Whooping-cough, Laryngitis, and the like; in which the cause of cough is a temporary one, the cough ceasing but the Emphysema remaining.
4. Violent fits of sneezing and of nose-blowing, under peculiar conditions of obstruction.



It is evident that, in the fourth of these sets of causes, the conditions likely to cause the sneezing and the nose-blowing will usually be such that, if they continue for any length of time, they will become causes of cough; and thus the occurrence of Emphysema before the cough, instead of after it, is accidental.

In the first three sets of causes, it is evident that they may have ceased for any length of time after producing the Emphysema, and, except so far as the existence of Emphysema is concerned, may have no possible influence over the after history of the case. A man may, for example, have overstrained his lungs by lifting too great a weight twenty years ago; or a child may have had Whooping-cough, and overstretched the air-cells during one of the paroxysms; and the man and the child may have remained Emphysematous ever since, although the Whooping-cough had long been completely cured, and the heavy weight never again lifted.

In these cases, the Emphysema may be considered as a disease, *per se*, as long as it remains uninfluenced by superadded diseases. It is well, then, for our present purpose of correctly estimating what it is we have to treat, that we first consider what are the essentials of this simple Emphysema.

In the first place, it consists of a portion of lung, the air-cells of which have been *overstretched*; and it is important to recollect what this overstretching means when it exists in its smallest appreciable degree.

You know that in the healthy state the walls of the air-cells are elastic, and that it is the essential condition of perfect elasticity that a body shall, when stretched, have the power of recoiling to exactly the same position



in which it was before the tension was applied to it. In proportion as it loses this capability, it deviates from the standard of perfect elasticity. A perfectly elastic body will retain this property until it is so far stretched that it snaps; but most elastic bodies retain their elasticity within narrower limits, and lose the power of perfect recoil before they sufficiently lose cohesion to snap; and thus they may be stretched to some extent beyond the point at which they retain the power of perfect recoil. It is this which constitutes overstretching of an elastic body; and the elastic air-cells of the lungs are susceptible of a certain amount of this over-stretching without rupture.

The elasticity of the lungs is so nicely calculated to meet the requirements of respiration, that, in the normal condition, after the fullest normal inspiration, the lungs recoil by elastic force alone to a condition of fullest normal expiration, and yet retain an elastic power competent to recoil still farther; so that when full ordinary or elastic expiration is supplemented by extraordinary or muscular expiration, the lung is still recoiling before the contracting chest-wall. Thus, in the natural state, pressure of the chest-wall upon the superficies of the lung is unknown.

Upon the perfection of this degree of elastic recoil everything depends.

The smallest appreciable degree of *overstretching* of the air-cells may be taken to be such as shall leave their power of recoil intact to the extent of full *ordinary* expiration, but deprive them of that which should still carry them on before the contracting chest-wall in extraordinary or *muscular* expiration.



But small and trifling as this amount of overstretching and of impaired function may at first sight appear, it constitutes the first step in a most important series of changes, and, for that reason, must take rank as a very serious disease.

It is true that when the loss of perfect elasticity is thus limited, there is nothing in it which need at all interfere with *ordinary* respiration. The lung can expand when the chest-walls are expanded by inspiration, and recoil before them when they recoil. But all the normal calculations are deranged when any cause arises which requires *muscular* expiration to be put into force. Then, at once, a totally abnormal condition is discovered, in which the chest-wall has to exert pressure upon air-cells full of air, and to drive the air hither or thither, according to the amount of muscular force exerted on different portions of the lung-surface.

In addition to this, an elastic body, which has been so far damaged as to have lost the power of complete recoil, must have lost something of the stability of the *whole* of its elastic power; and thus is rendered more susceptible to further damage through loss of resistance, to an amount of tension which, before, it would have been competent to withstand.

In illustration of such changes as I have described, I may refer to cases, such as those already cited, in which persons received, many years ago, some overstretching of the air-cells, as in the shock of an attack of Whooping Cough in childhood, but suffered no material interference with respiratory power until some superadded affection placed an obstruction in the way of the expiratory tide; and thus taxed the elastic power of the air-cells, and



called for forced muscular expiration, or some change in the occupation of the person called for forcible expiratory acts performed with a closed glottis.

But in an advanced state of Emphysema the walls of the air-cells are found attenuated, their capillaries ruptured and obliterated, and their partitions broken through, so that several cells are thrown into one. Those bronchial tubes, which run among the distended and crowded cells, are subjected to undue pressure from without, and to diminution of their calibre, when muscular expiration is brought to bear upon the Emphysematous lung by which they are surrounded.

Turning back, then, to the question with which I started—the question of treating this Emphysematous state of lung *independently as a disease, per se*—everything turns upon the elastic property of the air-cells, and whether or not it can be restored, when lost even in the smallest degree. Now we must remember on what the loss of elasticity depends. If it were upon damaged muscular tissue, either organic or inorganic, we should be able at once to turn to certain remedies and hygienic measures which are known to be capable of restoring lost function to muscular tissue and of increasing its contractile powers. If it were simply dependent upon elastic fibres snapped in two, we might hope, by the reunion of the snapped fibres, to restore the lost elasticity. But, unfortunately, the condition with which we have to deal consists of *elastic fibres overstretched*, and thus, by some change in the relation of their molecules, deprived of their essential physical property; and we have no positive knowledge of any remedy competent to effect the rearrangement of the molecules, and thus to bring back the normal function.



It seems then a very hopeless case ; and all that we know clinically of the progress of elastic tissues which have been overstretched, does not do much to reassure us. And yet there is some hope to be gathered from this source. We have in the skin the most familiar and easily observed specimen of a mixed fibrous and elastic tissue—a tissue, the elasticity of which depends upon the interweaving of elastic fibres with other materials, as in the air-cells ; and from observation of what happens in the skin, we may gather both discouragement and hope. We know how beautifully the skin recoils after tension under normal circumstances ; how it accommodates itself to a stretched condition under some circumstances ; and how, under others, as when tumours have disappeared over which it has been extended, it recoils, and obliterates the trace of the previous distortion. But we also know how very slow it is to recover itself if it has been overstretched beyond a certain point, and, especially, if that overstretching has been of long continuance, or often repeated, as in the pendulous folds which so long disfigure those who, having been fat for years, have become thin.

But still we know that, even in such cases, though the change may be slow, yet, under favourable circumstances, especially under the influence of vigorous, healthy assimilation, time will do much to restore the normal elasticity to the skin. By analogy we might well hope for such changes in the over-stretched elastic tissue of the air-cells. And theoretically it would appear that, if healthy assimilation could be kept up, the cause of the damage not repeated, and sufficient time granted, a healthy molecular condition might be restored to the damaged elastic tissue.



But how shall we secure "healthy assimilation" in a tissue withdrawn from the influence of volition, and deprived, by the very change we wish to remedy, of the exercise of its involuntary mechanical function? Are not these the essential conditions of degeneration rather than of healthy nutrition! Again, how shall we prevent the "repetition of the cause of damage" in a part whose damaged condition is itself the excitor of new sources of injury? And, lastly, Is not the "grant of sufficient time"—so precious when the other conditions are fulfilled—but an opportunity for degradation of tissue when they are withheld!

If, then, the restoration of over-stretched air-cells to their normal properties and functions is such an unpromising task, when the morbid change is once firmly and long established, let us consider whether we have any means of staying the progress of Emphysema when it first begins, or of depriving it of any of its evils when it is further advanced.

I am quite certain that I have seen air-cells, temporarily over-distended under peculiar morbid conditions, regain their normal state when those conditions were removed; and I have often seen cases of confirmed Emphysema, which had long been accompanied by the most distressing and ominous symptoms, deprived of all their serious import by the removal of thickening of the naso-pulmonary mucous membrane, although no remedial change had occurred in the Emphysematous condition itself.

This brings me to the fourth on the list of "conditions, with some or all of which we have to deal, in the majority of cases of Winter Cough," viz., "thickened naso-pulmonary mucous membrane, and narrowing of the passages which it lines."



I have already shown you that this is the most important of the causes of Emphysema. I have shown that this condition plays the principal part in causing variations in the degree and persistency of the short breathing, whether the Winter Cough is accompanied by Emphysema or not. I have shown that when Emphysema exists in a slight degree, it may be pushed on to any extent, by continued obstruction to the expiratory tide by narrowed air-tubes, and that its progress may be stayed by removing the obstruction. I have shown you how the Winter Cough itself is dependent, in the majority of instances, upon the condition of the naso-pulmonary mucous membrane. And I have shown you the way in which this thickening of the mucous membrane and narrowing of the air-passages, takes place. We have seen how various are the causes, both within and without the body, by which flushing of the mucous membrane may be excited. I have shown you that this flushing may be a very transient condition, or may run on into the production of serious and permanent changes in the chest.

The cases which I have laid before you illustrate all the stages of Catarrh of the naso-pulmonary tract, from a mere cold in the head to a severe and abiding disease of the whole bronchial tree. They show the way—the insidious way—in which Catarrh steals its marches on its victims; how simple in character and short in duration the first attacks may be; how they dispose the mucous membrane to fresh attacks; and how apt each attack is to involve a larger extent of surface than its predecessor; how often it happens that, when once the complaint has reached the finer ramifications of the bronchi, it lurks



there still—even when the larger tubes have been restored to temporary health—every fresh attack of Catarrh in the larger passages supplying the lurking enemy with reinforcements, and enabling it to advance from its fastnesses, and to encroach further and further upon the respiratory tract, until, at last, it needs but a breath of wind upon the lining of the nose or fauces to raise a storm of rebellion throughout the length and breadth of the naso-pulmonary mucous membrane. Thus have we seen how very much that is connected with Winter Cough, in a practical sense, centres itself in Catarrh.

It is to this point, therefore, that I wish to devote most of the time we have left for the consideration of treatment—the treatment of Catarrh, and of those changes in the mucous membrane and in the calibre of the tubes, which result from Catarrh.

If we could nip every Catarrh in the bud what a catalogue of ills we should prevent. And yet this is not such a very difficult thing to do, when we have a chance of trying it. But, unfortunately, colds are thought so lightly of by patients, that they seldom try to stop them till they have become severe, have lasted an unusual time, or have produced some complication. Nevertheless, I believe, they would do better in this respect if they had more faith in the possibility of stopping colds; if their doctors would impress upon them more the importance of stopping them; and, especially, if they knew that colds can be stopped without lying in bed, staying at home, or in any way interfering with business.

I shall, therefore, occupy your time for one minute to tell you my plan of stopping a cold. The plan will not answer if the cold has become thoroughly established; it



must be begun directly the first signs of Catarrh show themselves in the nose, eyes, throat, or chest—in fact, before any considerable amount of secretion has taken place. If employed at this stage it is almost infallible. The plan is as follows:—

1. Give five grains of ses-carb. of ammonia, and two minims of liq. of morphia in an ounce of almond emulsion every three hours.
2. At night give ʒiss. of liq. of acet. of ammonia, and a tumbler of cold water, after the patient has got into bed and been covered up with several extra blankets; cold water to be drunk freely during the night should the patient be thirsty.
3. In the morning the extra blankets should be removed so as to allow the skin to cool down before getting up.
4. Let him get up as usual, and take his usual diet, but continue the ammonia and morphia mixture every four hours.
5. At bed time the second night give a compound colocynth pill. No more than twelve doses of the mixture from first to last need be taken as a rule; but should the Catarrh seem disposed to come back after leaving off the medicine for a day, another six doses may be taken and another pill. During the treatment the patient should live a little better than usual, and on leaving it off should take an extra glass of wine for a day or two.

As everything depends upon the promptitude of the treatment, persons who are subject to Catarrh, especially if it inclines to the influenza character, should be provided with a prescription for the medicine, and full instructions how to manage themselves *when a cold sets in*. Many old Catarrhal patients of mine, who have been accustomed for several years to stop their colds in this way, have given their medicine the somewhat unprofessional title of



the "magic mixture," and would not be without it for the world, so often has it saved them from their old enemy. That, then, is, in my opinion, the best and simplest way of *stopping a cold*. It, in fact, leads to its cure by "resolution." An addition to this plan is needed in persons whose colds seize at once upon the bronchial mucous membrane. Besides the plan of proceeding I have described, they should put ten grains of extract of conium, ʒi. of compound tincture of benzoin, and ʒss. of sal volatile into a pint of hot water, temperature 170°, and inhale the steam for twenty minutes at bed-time each night; put a mustard poultice on the front of the chest one night, and between the shoulders the next; and, unless the weather is warm, should wear a respirator out of doors till all signs of the cold have quite passed off.

By these simple means, promptly adopted, an attack of Catarrh may generally be stopped, and thus all the troublesome and serious effects prevented which follow an established and protracted cold. I would particularly point out, that in Epidemic Catarrh or Influenza I have followed this plan of treatment, and it has proved most successful.

If these timely steps have been neglected, and a catarrh in the naso-pulmonary tract has become fairly established, a different plan of treatment is of course required, which must differ according to the severity of the attack, and the part of the mucous membrane principally affected. It may then of course become necessary to confine the patient to the house, to his room, or to his bed, and may involve all the treatment usual for acute Bronchitis, with which you are so well acquainted. With this I will not occupy your



time, therefore, further than to impress the great importance of completely curing each attack—of leaving no vestiges behind; we must only be satisfied when, by all methods of testing the respiratory powers, we cannot detect a lingering trace of disease in the naso-pulmonary mucous membrane.

But there is a form of Catarrh, common—I might almost say universal—among the children of the poor, of which we have very little chance of seeing the beginning, although we too often see the end. It begins so early in their little lives that they seem as though they were born to it, and it goes on summer and winter from year to year. It is, of all forms of Catarrh, that which most certainly leads to thickening of the naso-pulmonary mucous membrane and narrowing of the air-tubes. It begins in carelessness and folly, and continues through carelessness and folly. The children are not half-clothed from their tenderest years; the little money at the disposal of the parents is wasted on a few fine clothes, instead of being spent on a sufficient covering of wool next the skin. It is quite sad to see the children brought to this Hospital—martyrs to Catarrh—with tawdry feathers and smart ribbons, but not a scrap of flannel on their wretched little bodies. Besides these defects in dress, they are subject to be taken by their mothers from close, smoky rooms into the cold air, and to be exposed at the corners of draughty streets while their mothers are gossiping late into the evening. While these conditions remain, no medical treatment can be of any avail; and there can be no doubt that it is from this source that a very large number of the cases of narrowed air-passages, chronic Bronchitis, and Emphysema, are supplied to Hospitals.



We come, then, to the important question,—what plan of treatment is to be pursued in cases of confirmed thickening of the naso-pulmonary mucous membrane, with narrowing of the air-passages?

The number of such cases presented at this Hospital is enormous, and I have no hesitation in saying, that they are quite susceptible of successful treatment, although the difficulties in the way are often very great. In private practice it is much easier to cure these cases, because proper treatment can be more satisfactorily carried out.

I have already explained that in this state the mucous membrane is remarkably susceptible to fresh attacks of Catarrh, by each of which its morbid condition is aggravated. The first point in treatment, then, is to provide against such attacks. It is, indeed, the fact that the membrane will, in course of time—a long time, certainly—spontaneously recover its normal condition, if it can be *absolutely* protected from the recurrence of Catarrh. It is in this way that quite wonderful recoveries have been made by fortunate changes in climate. I use the word *fortunate* advisedly, because even the best-selected change is sometimes most unfortunate, in consequence of those vagaries in the seasons to which all climates are subject.

But, even if we could always select an appropriate climate, and secure that it should not be fickle, it is not every one who can obtain change of climate, and scarcely any one who can do so just at the time it is needed. Unless, then, we are to confine our beneficial treatment to a very small number of favoured persons, we must find some substitute for change of climate; and this brings me to the question of the *use of respirators*.

But first let me not omit to speak of that very important



class of influences which I placed seventh on our list of the things we have to treat—those “general conditions, favouring or producing morbid susceptibility of the mucous membrane of the air-passages.”

When treating of the hereditary transmission of a tendency to naso-pulmonary Catarrh, I pointed out that this was due to the hereditary nature of certain diathetic states; and when treating in detail of the properties of mucous membranes, and the various modes in which they are affected, I pointed out that flushing, congestion, irritation, increased secretion, and all the phenomena of Catarrh, may be brought about by the presence in the blood of such impurities as proceed from mal-assimilation, imperfect digestion, rheumatic, gouty, syphilitic, typhoid, rubeoloid, and other poisons; and I pointed out the analogy in this respect between affections of mucous membranes and affections of the skin. I need not, therefore, go over this ground again; but it is in relation to treatment that these facts assume the greatest importance. We may as well expect to cure a skin-disease dependent upon mal-assimilation by external applications alone, to cure an attack of gout by poulticing the great toe, or to remove a syphilitic affection of the eye by the application of simple lotions, as to effectually treat an affection of the naso-pulmonary mucous membrane, dependent upon similar general causes, by remedies directed only to the catarrhal condition of the membrane. We may, indeed, in one case and in the other, produce temporary local amendment by such local and narrow-minded treatment; but we know that the local disease will recur again and again so long as we neglect the general condition. When I say, then, that the first thing in



treatment is to provide against fresh attacks of Catarrh, I must place first on the list of means of such prevention, the treatment of whatever general conditions we can discover which, acting from within, may favour or produce morbid susceptibility of the mucous membrane of the air-passages.

Everything that we learn from physiology and pathology ; all that we know of etiology, confirmed by our deepest clinical experience, conspires to hold up *the treatment of diathesis as the secret of therapeutic success.*



## LECTURE VII.

### TREATMENT—CONTINUED.

HAVING, then, attended to the diathesis, let us proceed to protect the air passages from causes of irritation acting from without.

What these causes are we have already learnt from the analysis of the cases which I have reported.

Fresh colds were the only causes which brought on or aggravated the cough in 72 per cent. of the cases, and the most frequent and potent causes of these "fresh colds," as stated by the patients themselves, were—

1. Sudden changes of temperature in 21 per cent.
2. Fogs and damp air in 19 per cent.
3. Draughts of cold air in 16 per cent.
4. Cold winds in 10 per cent.
5. Getting wet in 14 per cent.
6. Wet feet in 17 per cent.

And we find this same list of causes of fresh colds as the potent provokers of short breathing. This important list is no less striking for the power for evil which it is shown to possess, than for the remarkable simplicity of the evil powers themselves.

If then these unfortunate sufferers from Winter Cough could have been protected from sudden changes of tem-



perature, fogs, mists, cold winds, draughts, wet feet and wet coats and dresses, 72 per cent. would have been kept free from their coughs.

It would indeed seem strange if we could not find means of protection against such common-place influences. In truth, there is no deficiency of means of protection against them, and it is because of the very common-place character of those means of protection, and of the influences themselves that both are so much neglected and undervalued.

But 72 per cent. of the cases of Winter Cough which I have analysed, might probably have been prevented by attention to these common-place things. Let us then give a few minutes to their consideration.

1. Sudden changes of temperature.

This is the most difficult to avoid of any on the list. The occupations and amusements of all classes involve such changes, and we cannot stop these occupations and amusements, even were it desirable to do so. The workshop, the counting-house, the committee-room, the opera-house, the ball-room, must be warm when the outer air is cold, and changes from one to the other cannot be avoided. But very much could be done to prevent the body from feeling these changes. The first and most important is the complete envelopment of the body and limbs in wool next the skin, thus interposing a bad conductor of heat between the surface of the body and the outer air. It is surprising that even in the present day this simple and common-sense protection is neglected by so large a number of persons both of the educated and of the uneducated classes. It is not sufficient for the purpose in view that a little body-vest should be worn just big enough



to cover the thorax and abdomen, leaving all the extremities unprotected. It should be insisted upon by medical men that the arms and legs require to be protected from sudden transitions of temperature as much as the trunk. In fashionable life the greatest practical difficulty we have to encounter is the question of exposing the necks and shoulders of ladies in evening dress. It is useless to order body clothing of wool to the throat, and to expect that ladies will give up a fashion which has been followed and thought charming in all countries and all ages. The difficulty is, however, to be got over pretty well. Every lady in evening dress should carry with her, as invariably as she does her pocket handkerchief, a Shetland shawl or a mantilla of wool or fur, of a size and shape to cover all those parts not protected by woollen underclothing, and it should only be removed while actually within warm rooms, and should be kept at hand to replace on passing through passages, or if the rooms become cold, or if sitting in draughts.

The main source of protection then against sudden changes of temperature to the surface of the body, is to be found in a complete covering of wool next the skin. But besides this, a much greater attention than is common should be paid to putting on and taking off complete and efficient *over-clothing*, on going from hot to cold, and from cold to hot temperatures.\* This is particularly neglected by the working classes, and by girls and boys at schools. In fact, schoolmistresses and schoolmasters appear to be peculiarly neglectful and peculiarly ignorant of the grave

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\* See "A Manual of Diet and Regimen," 3rd Edition, by Horace Dobell, M.D. Churchill.



importance of these matters, as they appear to be of so many others, which not less vitally concern the physical welfare of those under their charge.

But when we have adopted all available precautions for avoiding transitions of temperature to the surface of the body, we shall entirely fail in our object of preventing catarrh, unless we also protect the naso-pulmonary mucous membrane itself. But of this I shall speak again by and bye.

What I have said with regard to sudden changes of temperature will apply equally to two other causes of fresh cold upon our list, viz., draughts of cold air and cold winds. Both are to be deprived of their sting by proper clothing of the skin and mucous orifices.

Getting wet, and wet feet, occupy a very serious place in our list, and there is no doubt that damp and cold applied to the general surface is the most efficient means of producing chill and vital depression, with congestion of the internal organs. It is necessary that cold be combined with moisture to produce this effect. Even if all the clothes on the body are wet, no harm will come so long as they are kept warm, and this suggests the very great value, to all persons liable to exposure to wet, of light waterproof overalls. They may either be put on to keep the underclothing dry, or, if the underclothing has become wet, either by weather or by perspiration, they may be put on to prevent too rapid evaporation and consequent reduction of temperature, especially when the person is about to remain still after getting warm with exercise. In this variable climate, therefore, school-girls, governesses, shop and factory-girls, and all women whose occupations call upon them to brave the weather, ought to



carry with them complete waterproof mantles made as light as possible, but extending from the neck to the ankles, and that can be put on or not as required; and boys and men similarly exposed, should carry waterproof overalls.

These are things easily obtainable in the present day, and within the reach of all classes; so that it only requires that their importance should be sufficiently impressed upon those who need them.

But if wet and cold to the surface of the body is a fruitful source of catarrh, wet feet—which means wet and cold feet—is a still more prolific source. There is no external influence which so surely produces congestion of the naso-pulmonary mucous membrane as wet and cold to the soles of the feet. There is nothing so universally neglected, and yet there is nothing so easy to avoid. Warm socks, horsehair soles, goloshes, provide efficient protection against wet and cold feet. It does not seem to be half enough understood that, although a shoe or boot may not be wet through, if the sole is damp, it will by evaporation most effectually conduct away the heat from the sole of the foot, and therefore ought never to be worn after exercise is over.

I should hardly have ventured to occupy so much of the time of a medical audience with these apparently simple and commonplace suggestions, were it not that they are so commonplace that their importance is apt to be overlooked.

We have still one item left on our list—viz., Fogs and Damp Air, which were the things most inclined to make the breath short in 24 per cent. of the cases, and the most potent causes of fresh colds in 19 per cent. I



have particularly remarked, that although the smoke and other irritating matters constituting fog are unquestionably very injurious, it is the moisture and cold of the fog which are the qualities most potent for mischief to the naso-pulmonary mucous tract. There is but one means of depriving a fog or mist of its injurious properties, and that is a respirator; and the same may be said of the changes of temperature, of which I spoke just now; a respirator is the only means of protecting the respiratory passages from the effects of transitions of temperature. It would be difficult to over-estimate the value of efficient respirators, in this climate, as a means of protection against naso-pulmonary catarrhs, if persons disposed to these affections would only carry respirators about with them in their pockets, ready to put on, if required, at a moment's notice.

I believe that any kind of respirator is better than none; but after experimenting with almost every kind that has been brought out of late years, I am quite convinced that there is none at all equal to Mr. Jeffreys' metal-wire respirator, or, as he now calls it, "pneumoclime." The "himalene," which he has just introduced, is also a most excellent instrument; but I object to it on account of the warm scarf in which it is concealed. Although it is quite proper to cover the neck lightly, I am decidedly of opinion that *warm wrappers* round the neck are objectionable; they produce congestion of the nasal and faucial mucous membrane, and thus dispose to the very complaints they are supposed to prevent. If Mr. Jeffreys would deprive his himalene of the scarf, or make the scarf of a much cooler material, I think it would be very useful for persons who feel the pneumoclime to be oppressive.



But before leaving this subject of sudden changes of temperature, I must not forget to speak of sleeping-rooms. It is quite astonishing what follies are committed with regard to the temperature of sleeping-rooms. On what possible grounds people justify the sudden transition from a hot sitting-room to a wretchedly cold bedroom, which may not have had a fire in it for weeks or months, it is impossible to say; but it is quite certain that the absurd neglect of proper warming in bedrooms is a fruitful source of all forms of catarrh. We cannot too much impress this upon our patients. It may often be almost as necessary for a delicate person to put on a respirator on going up to bed as when going out of doors, unless proper precautions are taken to assimilate the temperature of the sleeping-room with that of the sitting-room.

Such, then, are the principal means by which I would attempt to defeat the fickleness of climate, and to prevent the recurrence of those attacks of catarrh which keep up and aggravate the disease of the mucous membrane. And you will probably have observed that they all assume that the patient suffering from Winter Cough is to lead an active and an out-of-door life—not to be confined to his bedroom, or his sitting-room, or even to his house.

This is a point in the treatment which I consider of very great importance.

Shut up your patient month after month, and perhaps winter after winter, in warm rooms, with little exercise, and you need not be surprised if you add fatty degeneration to his Emphysematous air-cells; fatty degeneration to his heart, the muscular strength of which is so important in keeping up his pulmonary circulation; biliary con-



gestion to his liver, already disposed to be overcharged with blood; fat to his omentum, to impede the free action of his diaphragm—so essential to his easy respiration; dyspepsia to his digestive organs, the vigour of which is so important in keeping up healthy nutrition in his tissues;—in fact, if you adopt the “shutting-up system,” you need not be surprised if, after a dreary hypochondriacal life, your patient should become prematurely old, and die of apoplexy, paralysis, or dropsy.

But when we have taken all these precautions—when we have taught the patient how to stop fresh colds; when we have altered those general conditions of his system which predispose to his complaint; and provided him with the means of avoiding the recurrence of Catarrh;—there is still much left for us to do, and which we can do successfully, to promote the recovery of a normal condition in the naso-pulmonary mucous membrane.

The principal agents in such treatment are:—

1. Medicines introduced into the stomach.
2. Medicines introduced into the air-tubes by inhalation.
3. Counter-irritation.

I do not believe in the possibility of adapting the exact details of treatment to particular cases, without taking into consideration and carefully balancing all the circumstances of each case, to an extent which it is impossible to do in lectures and books or in any other way, if the patient is not before us. I will not pretend, therefore, to direct the exact cases in which this or that remedy or combination of remedies is to be used. To do this is, in my opinion, very much like the folly sometimes perpetrated by Governments, of issuing from their offices



at home, orders for the exact mode in which their generals abroad shall conduct their battles. It has always ended in defeat.

Having, therefore, put you in full possession of the principles upon which your treatment is to be based, all that I shall further attempt to do is to call attention to those medicinal armaments at our command, which I consider most important in carrying out the principles of treatment indicated in these lectures. The exact disposal of the forces in any given case must be left to the judgment of the man who takes the responsibility of conducting the battle.

1. Of medicines given by way of the stomach, I would particularly call attention to ses-carbonate of ammonia. When treating the naso-pulmonary mucous membrane, we must not look upon ammonia simply as a stimulant; it has a most marked and important action upon the mucous membrane, as it has upon the skin in Erysipelas; probably this is due to its influence on the blood and on the capillaries. It assists more than any one other drug in restoring a healthy condition to a mucous membrane affected with catarrhal congestion. It may be variously combined with other medicines under varying circumstances. Thus, if there is a high state of recently excited injection, tartarised antimony may be given with the ses-carb. of ammonia with the best effect. If there be great irritability of the membrane, morphia in small doses may be given with the ammonia, as I have already directed, to stop a fresh cold.

With regard to antimony, it is important to bear in mind that it ought never to be continued long. The good it can do is soon done, if done at all, and directly it is accom-



plished the medicine does harm. Of ammonia I would also say that whereas, when first administered, it acts as a stimulant, it very soon loses this effect and becomes a depressant, by its action on the blood. It should never, therefore, be too long continued. It is better to withdraw it for a time and give it again, than to keep on with it too long at once. With morphia it is necessary to be very watchful that it does not stop secretion, when free secretion is the best means of relief to a congested membrane, and that it does not stop cough, when cough is an indispensable means of clearing tubes choked with secretion. But these are matters with which you are, no doubt, perfectly familiar.

Of ipecacuanha and squills I need hardly speak, their effects are so well known—one as a relaxing and soothing promoter of secretion—the other as an irritating expectorant, which assists in clearing the membrane of secretions already produced, and stimulates the mucous glands to contract and cease to secrete more. Ipecacuanha, therefore, may do harm in one way, and squills in another, if too long continued.

Senega irritates the cough. If there is too little cough, in proportion to the secretions requiring removal, it is an invaluable medicine, but I think its value is restricted to producing this effect. If the cough is already frequent, it does harm.

Ammoniacum acts like senega in irritating the cough, and thus assisting expectoration; but beyond this it has a further effect in stopping secretion, and afterwards producing excitability of the mucous membrane, which may become excessive if not watched.

I consider olibanum a much more valuable drug than



ammoniacum, and I am sorry to see it so little used. It has a remarkable effect upon the intestinal mucous membrane, arresting chronic dysentery and diarrhœa, and leaving a soothed condition of the bowel. I have seen the same effect produced upon the mucous membrane of the air-tubes—morbid secretion checked and altered without that irritation which is caused by ammoniacum. The olibanum may be used both by the stomach and by inhalation. It is probable that its topical effect is the more important.

Benzoin is another valuable remedy in restoring a healthy condition to a thickened naso-pulmonary mucous membrane. It may also be used by the stomach or by inhalation.

Spasmodic contraction of the bronchi indicated, as I have already pointed out, by the high pitch of the respiratory sounds, is a very awkward complication; and if not kept in check, it very much interferes with the restoration of the mucous membrane to a state of health. Stramonium, administered by the stomach in quarter grain doses twice or thrice in twenty-four hours, becomes a very useful adjunct to other treatment under these circumstances; or it may be used by inhalation, or some of the other medicines named under the head of inhalations may be employed instead.

I must not omit to remind you of the facility with which one part of the naso-pulmonary mucous membrane is able to act for the relief of another, as illustrated by the case I related of the gentleman whose spasmodic asthma was often carried off by sneezing, and who suffered from dyspnœa when a discharge of mucus from the nostrils was suddenly stopped. We may often take advantage of this property of the mucous membrane



with the best effect. Congestive tumidity of the bronchial membrane may be relieved by giving iodide of potassium in sufficient doses to produce the symptoms of a free coryza; and errhines may be used for the same purpose. Iodide of potassium, however, requires to be given with caution; for if it does not succeed in producing a free discharge from the nares and sinuses, it simply inflames the mucous membrane; and this inflammation may run down and aggravate the affection it was intended to relieve. Unless there was a syphilitic taint, I have never been satisfied that iodide of potassium acted beneficially upon a thickened naso-pulmonary mucous membrane in any other way than as a derivative, in the manner described.

I can speak much more satisfactorily of the influence of saline aperients acting as derivatives on a different tract of mucous membrane, and free, therefore, from the chance of exciting a tumid or inflamed condition in the diseased one. I know of no form of saline for this purpose so generally useful as the Friedrichshall water (which can be easily obtained in this country at the Mineral Waters Company, 27, Margaret-street, W.).

There is, however, a very nice form of aperient mixture which I often use, consisting of sulphate and carbonate of magnesia, with a small quantity of iodide of potassium, and a full dose of nitric æther, and which appears to suit some cases better than the Friedrichshall water,—producing a freer and more watery discharge.

In some cases of considerable and long-standing thickening of the mucous and submucous tissues, it may be necessary to give small doses of bichloride of mercury for a considerable time. Colchicum, too, which acts so bene-



ficially upon some affections of the skin, is often of great use in treating the respiratory mucous membrane.

There are many other remedies which I might enumerate, if we had more time at our disposal. But I am anxious not to leave the subject of medicines given by the stomach without mentioning tonics. Nothing can be more important in the treatment of winter cough than to improve the tone of the whole system of the patient by every means in our power. Zinc, iron, and quinine are the most important of such medicines; and I think their value may be taken to stand in the order in which I have enumerated them. Strychnine has been highly recommended in winter cough complicated with Emphysema; but as it especially acts upon the muscular system, exciting the muscular fibres to spasmodic contraction, I see no reason to expect any good from it in the case of diseases of the air-passages and cells; for, in the first place, we wish to diminish and keep off spasmodic muscular contraction of the air-tubes; and, in the case of the overstretched air-cells, they are not muscular but elastic fibres which we wish to strengthen; and there is no possible reason for supposing that strychnine acts upon elastic tissues. If it does good, it can only be through its effect upon digestion. In this way, like many other medicines that might be mentioned, it may do good service; for we must ever bear in mind, that, in order to prevent degeneration of the Emphysematous air-cells, of the walls of the air-tubes, and, especially of the heart, every available means must be employed for keeping up the vigour of the nutritive functions; so shall we not only keep off degeneration of tissue, but promote the healthy repair of the diseased parts.



Under the head of the general conditions predisposing to Winter Cough, I have already intimated the importance of correcting all forms of mal-assimilation and digestion. The internal medicines and diet necessary for this purpose must, of course, differ with the case; and I cannot do more in this place than remind you never to forget this part of the treatment. (See Appendix.)

I have already shown, when analysing the reported cases under the head of Colds and Coughs, that in 45 per cent. of all the cases of Winter Cough, the cough left in summer weather, and the short breathing was relieved in summer weather in 29 per cent.; and we found this relief to short breathing and to cough was really due to the removal of the irritability and thickening of the nasopulmonary mucous membrane, under the influence of *warm soothing inhalations in the form of summer air*. These are very important facts to bear in mind with relation to treatment, not only as indicating the importance of wearing respirators, to which I have already referred, but as pointing to the use of inhalations as a means of restoring the mucous membrane to a healthy condition. Inhalations may consist of fumes, vapours, or atomised fluids.

My own experience of atomised fluids is not at all satisfactory, except as a means of applying lotions to the nares and fauces. I object to their use for affections below the glottis, in which it is necessary to allow the patient to respire during the operation. They have the great disadvantage of conveying too large a quantity of cold moisture into the air-passages, and are thus apt to produce all the evils of severe damp and fog.

Fumes and vapours have not this objection, and are



among the most valuable of our means of acting upon the naso-pulmonary mucous membrane. Nitrate of potass is of great service when used in this way. It appears to act upon the respiratory mucous membrane, when introduced in the form of fumes dispersed through atmospheric air, much as it does on the pharyngeal mucous membrane when applied in the popular form of "sal prunella balls." It refrigerates and causes resolution of that flushed and tumid condition which I described when speaking of Catarrh and Asthma. It is in this way, I think, that it so signally relieves some cases of spasmodic Asthma. You will find that the fumes of nitre paper, and those of datura tatula, stramonium, and chloroform, relieve different classes of cases, or the same case in different phases. When the prominent mischief is vascular injection and tumidity, the spasmodic contraction being only excited by the excess of these conditions, nitre fumes give the most relief. When, on the other hand, the nervous element is most prominent, the tendency to spasmodic contraction being so great that it is set up by a comparatively slight amount of flushing of the mucous membrane, datura tatula, stramonium, and chloroform are the most potent remedies. The influence of the fumes of nitre upon the bronchial spasm is only a secondary effect, its most important influence being that it removes congestion and tumidity of the naso-pulmonary mucous membrane.

For the inhalation of vapours I am accustomed to order Nelson's inhaler, to be used without the sponge; a pint of hot water being put into it, the temperature of which should not exceed  $170^{\circ}$  when inhalation begins. The patient should inhale for about fifteen minutes at a



time, and should not go out of doors the same day that the inhalation is used without a respirator. It is often convenient, therefore, to restrict the use of the inhalation to the evening.

The materials which I principally use to medicate the vapour are—

1. Compound tincture of benzoin.
2. Compound tincture of iodine.
3. Carbonic acid.
4. Creosote.
5. Camphor.
6. Chloric æther.
7. Extract of conium.
8. Hydrochlorate of ammonia fumes, produced by adding a few drops of hydrochloric acid after liq. of ammonia has been mixed with the hot water.
9. Liq. of ammonia or aromatic spirits of ammonia.
10. Tincture of myrrh.
11. Tincture of lobelia.
12. Tincture of stramonium.
13. Turpentine.

You will see on the table an ingenious instrument, invented by Mr. White, surgeon, of Finchley, who has so long devoted himself to pneumatics, and very skilfully carried out by Mr. Norman. If we wish to immitate and to sustain the natural effects of summer weather upon the air-tubes, this instrument appears to be admirably calculated for our purpose; for, by its means, a patient may go about all day long, and continue all night long, if necessary, inhaling air at almost any temperature, and in almost any state of moisture, dryness, or medication that may be thought advisable. From its portability Mr.



White has named this instrument the *vade-mecum* inhaler. But as I see Mr. White and Mr. Norman both in the room I will leave it to them to demonstrate to you, after lecture, all the uses to which their apparatus may be applied. I have no doubt that, in suitable cases, it will prove of very great service.

There is no class of complaints in which counter-irritation gives such unquestionable and unqualified relief as in affections of the respiratory tract of mucous membrane. The relief to the oppressive dyspnœa and to the irritability of tubes long narrowed by thickened lining, which speedily occurs under the influence of decided counter-irritation, is delightful to witness. In severe cases I am in the habit of ordering, at the commencement of treatment, three blisters, one for the front of the chest, and one for each side, between the scapula and the breast, to be used in succession; each blister to be allowed nearly to heal before the next is applied. The application of the first I *insist* upon, the other two I leave to the patients' judgment to apply or not as they choose, after having found what relief is obtained by the first. It is very rare indeed to find that they fail to apply all three—much more often they are so pleased with the effect as to volunteer to put on more if necessary. No form of counter-irritation is at all equal to a well-managed blister. Great caution should be taken not to leave the blisters on long enough to produce deep sores troublesome to heal. They should be quickly removed, and a warm linseed poultice applied before the vesicles are cut. Decidedly the nicest form of blister, and that which gives the patient least trouble, is Brown's cantharidine tissue and the tissue dressing sold with it.



When the tracheal and laryngeal membrane is that most affected, little strips of vesication may be produced along the course of the trachea and at the sides of the neck, in the rear of the thyroid cartilage. It is better not to apply blisters over the larynx. When the posterior nares and pharynx are most affected, the back of the ears or back of the neck are the best seats for external applications.

After the more decided effect has been obtained by blisters, iodine lotion, and camphorated ammonia, and turpentine liniments, may be employed with advantage; but if the case is severe or of old standing, and the patient will allow it, never fail to start with the blister. One caution, however, is needed, viz., not to blister while the skin is hot and dry, and the patient suffering from active inflammatory excitement. In some stages of congestion of mucous membrane, especially where the tumidity is great and there is great reluctance to secrete freely, hot fomentations applied as near the affected part as possible give great relief, and, by promoting secretion, put the membrane in a better position to be benefited by other treatment. It is surprising how much good may be done in a short time by repeated hot fomentations and poultices to the back and sides of the neck in affections of the nasal, pharyngeal, and laryngeal mucous membrane.

By a combination of such medicaments, appliances, and hygienic regulations as I have now briefly mentioned, we may hope to remove the susceptibility of the nasopulmonary mucous membrane, and to reduce it to its normal thickness, so that the air-tubes may be restored to their natural calibre, and the obstructions to the expiratory tide removed. The importance of accomplishing these



ends you will perfectly understand from what we have already seen respecting the causation of Emphysema, and the circumstances which aggravate it when it exists and give poignancy to all the sorrows which it inflicts.

Now, you will perhaps be inclined to charge me with having altogether shirked the main question with which we are concerned, viz., the *Winter Cough*—with having discussed anything rather than the cough, and directed treatment to everything but the cough; and my answer to the charge is, that if everything is attended to which we have discussed, and everything satisfactorily treated that we have spoken of, there will be no cough left for us to treat.

Nevertheless, a word must be said about the cough *per se*. We have seen that frequent cough, and especially convulsive cough, is one of the causes—and a very potent cause—of Emphysema. It will be very important, therefore, to allay the severity of the cough by some direct means while the more soundly curative treatment is being pursued.

This may be most effectually done by the sedatives and anti-spasmodics of which I have already spoken, and they are especially useful when applied in the form of inhalations. But I have not yet mentioned three very valuable remedies—Indian hemp, lobelia, and bromide of ammonium. They have all the great advantage of acting speedily and decidedly. If they do not have a decided and speedy effect—if they do not, as it is popularly expressed, “act like a charm”—they do no good, and it is better to throw them aside; at least that is my experience.

Indian hemp is rather a troublesome remedy, from the difficulty of getting it of a reliable and equal strength;



but Mr. Morson has prepared a liquid alcoholic extract which is quite constant in strength and properties, and by specially ordering this we may rely upon our agent. Indian hemp is most likely to do good when the cough is accompanied by tendency to fitful Bronchial spasm, or when it occurs, not in long convulsive fits at distant intervals, but in a never-ceasing wearing and tearing bark.\*

Ethereal tincture of lobelia, on the other hand, stops those convulsive fits of coughing which occur, almost like Whooping-cough, with distinct intermissions. It is sure to aggravate an irritable cough, but it often acts quite magically upon those distressing and exhausting attacks of convulsive cough which are, of all others, the most likely to produce Emphysema. It is in this same description of cough that bromide of ammonium often proves useful.

I must just mention bismuth as another direct means of stopping cough, though it acts by an indirect route. If you find that the cough always comes on when the stomach is empty, bismuth will stop it after everything else has failed—that is, everything else except food—and a patient cannot be always eating for the sake of keeping off cough. There can be no doubt that the bismuth in these cases acts through the gastric fibres of the pneumogastric nerve. Hydrocyanic acid will often have a similar effect, but the bismuth is a safer and more persistent remedy. Digitalis is of great use when the cough is kept up or excited by a too rapid and forcible action of the heart. The less important, but still useful means of allaying

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\* See *Medical Times and Gazette*, September 5, 1863, "On some effects of Cannabis Indica," by Horace Dobell, M.D.



cough are very numerous; but I must not detain you by enumerating them, as they are most of them familiar to us all.

In concluding these details of treatment, I wish to draw your attention to a very important practical point, about which there is apt to be great misapprehension, viz., the season of the year in which it is most necessary to pursue treatment for the radical cure of a Winter Cough.—IT IS NOT IN THE WINTER, BUT IN THE SUMMER.—It is not in seasons of rebellion that the institutions of permanent peace can be best established. It is as much as can be reasonably expected in such times, if human life is protected, serious catastrophes averted, urgent necessities provided for, and the various elements of rebellion scattered and repressed. It is in the more quiet after-time that wounds must be healed, differences adjusted, the under current of disaffection eradicated, and securities obtained against the recurrence of disasters. In a severe case of Winter Cough, the first winter in which we are consulted must often be occupied by a succession of skirmishes with fresh attacks of Catarrh, and with a more or less successful battle with a chronic pulmonary rebellion. If we succeed in dispelling the cough before the warm weather comes to our assistance, we must often consider ourselves fortunate. However this may be, we have seen by the cases I have reported, and we know by daily experience, that, in a large number of instances, when the long-looked for warm weather comes at last, the cough does go, and the patient thinks himself well. Both we and our patients are then apt to be so pleased at the change, that we at once withdraw our forces, glad to believe that peace is firmly re-established. But if we



turn for a moment to our report, we shall see how exceedingly insecure is this summer-weather peace.

From a careful analysis of the circumstances attending cases of Winter Cough in which the cough left in summer weather, and of those in which it did not leave, we found that the essential difference between them consisted in the degree to which the affection of the mucous membrane had extended deeply into the ramifications of the bronchi. I then showed you that, on comparing the cases, there was found to be a gradual assimilation between those of Bronchitis without Emphysema and those with Emphysema, in respect to the way in which they were affected by colds, and that this took place in proportion as the Bronchitis had become a deeply-seated and abiding affection. We found that the necessary condition to determine colds to fall at once upon the chest, was the existence in the bronchi of a semi-inflamed condition—a condition of imperfectly eradicated Bronchitis—which kept up a constant irritability and susceptibility to a renewal of the half-cured disease immediately an exciting cause was presented. The only connection we were able to trace between the presence of Emphysema and the tendency of colds to fall first upon the chest, lay in the fact that, in such cases, the Bronchitis had extended deeply into the bronchial ramifications, and had become so permanently lodged in the smaller bronchi that it remained but partially cured there, even when the soothing air of summer upon the larger bronchi and upper parts of the naso-pulmonary tract had allayed irritation and relieved or taken away the cough. And we found that what applied to the relief or non-relief of cough by summer weather applied also to the short breath. I must repeat here, therefore, under the head



of Treatment, what I said when analysing the causes of colds and coughs—that our great attention must be directed to the importance of leaving no means untried which can give a chance of radically and permanently removing every lurking trace of Catarrh, or Bronchitis, or of their effects upon the mucous membrane, before we let our patient consider himself safely cured. We must only be satisfied when, by all our methods of examining and testing the respiratory organs, we cannot detect a lingering trace of disease, and when, after this, we have instructed our patient by what means he can best maintain this restored condition till it has become habitual.

It is in the summer that this part of the treatment can be best commenced, and, if possible, it must be carried out through the succeeding winter. We cannot consider our case complete till a winter has been passed through without cough or short breath.

This is the time when we should fall back upon that “great gun”—change of climate. I have reserved it till the end of the lecture, as in practice I reserve it till the end of the treatment, because I am convinced that this is the time when, as good generals, we should bring it into action. When we have conducted our patient safely through the first winter in which we are consulted, and guided him through the succeeding summer; when we have done all that is possible to restore a healthy condition of his respiratory organs, and brought him to the middle of autumn with no cough and no short breath; when he begins to look at the approaching winter with dread, lest his enemy should return and call for a renewal of his last winter’s regime; when he is tired of restrictions, and turns sick at the thought of goloshes, water-proofs,



respirators, physics, inhalations, and all the rest; when even his gratitude to his physician is beginning to be overbalanced by the longing to forget every reminder of an invalid's life—then is the time, in my opinion, to send our patient for the winter months to some delightful climate where he may continue his summer liberty without risk, and substantiate his cure by a prolonged freedom from Catarrh, and by the invigorating influence of an active out-of-door life upon his general health.

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## APPENDIX.

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### POST-NASAL CATARRH.

THE following observations extracted from a Paper read by me in 1854, to the Abernethian Society of St. Bartholomew's Hospital, have been confirmed by my subsequent experience :

Among the diseases which come before the physician, rather as sources of discomfort than as causes of death, the one I am about to describe under the name of Post-nasal Catarrh is particularly worthy of attention :—1st, from its frequency ; 2nd, from the great inconvenience it occasions ; 3rd, from the serious effects of which it is occasionally the indirect cause ; 4th, from its being accompanied by the symptoms of other diseases (and thus misleading the patient, if not the medical man) ; 5th, from the obstinacy with which it often resists treatment.

Notwithstanding the obvious existence of Post-nasal Catarrh as a common complaint, it has not, so far as I can learn, ever been described as a distinct affection. I have, therefore, ventured to fill this gap by a brief description of the disease.

Post-nasal Catarrh may be acute or chronic, but it is much the more frequent in the chronic form, and is rather to be classed among the 'vestiges' of disease than among primary affections.

The acute form is caused by recent catarrh super-added to the chronic disease, the latter being the characteristic affection. I have, therefore, selected the chronic form for description. The symptoms are as follows :—

1. A sense of fulness deeply seated in the back of the nose, with a constant stinging and tingling sensation about the uvula, soft palate, and posterior part of the hard palate. This sensation is much aggravated after sleep, so that the patient wakes every *morning with a sore throat* ; but on examination of the throat, no inflammation, ulceration, or swelling is detected.

2. Short tickling cough, coming on at intervals, especially night and morning, or if long without food or drink ; but on examination of the chest no morbid sounds are present.



3. Frequent hawking and spitting of small pellets of mucus, which are not unfrequently of an orange-brown colour and very tenaceous.

4. On examining the pharynx, shreds of stringy mucus may often be seen hanging down from behind the velum; or the back of the pharynx is coated with brownish adhesive mucus, and sometimes, but not always, the mucous follicles are enlarged and red.

5. The morning sore throat is not relieved until after much forcible blowing of the nose, which is usually dry, a few pellets of inspissated mucus are removed. This mucus is often orange-brown from the admixture of a very small quantity of blood. The excretion of these pellets, which is a very troublesome process, gives immediate relief. But a certain amount of stinging and tingling soon returns, and continues through the day, to be aggravated as before by the succeeding night's rest.

6. In order to relieve the uneasiness about the fauces and posterior nares, the patient is constantly annoyed by an almost irresistible desire, either to draw the mucus down the throat, by a forcible inspiration, or to force it into the nose by an opposite effort; and, therefore, those who suffer from post-nasal catarrh acquire a habit of making a peculiar noise in the nose and throat, which is pathognomonic of the complaint. It is produced by inspiring by the mouth, closing the glottis, and then, with the tongue pressed against the hard palate, suddenly opening the glottis and jerking a gust of air up the pharynx through the posterior nares into the nose.

7. The usual symptoms of coryza—watery discharge from the nose, nasal voice, feverishness—are absent. If by any cause these symptoms are produced, those of Post-nasal Catarrh are for the time somewhat relieved, but only to be aggravated afterwards.

The history of the case will generally show that the post-nasal affection has been left behind as a vestige of one or more severe attacks of Influenza or Coryza, or of many slighter Catarrhs coming in quick succession.

The seat of this affection appears to be in the sphenoidal and posterior ethmoidal cells. The following considerations lead to this opinion:—



1. The deep seat of the sensation of fulness.
2. The absence of nasal voice, and interference with nasal respiration, except when the secretion has been voluntarily forced into the nose, or when ordinary Catarrh is superadded.
3. The great difficulty in dislodging the secretion, and of bringing it within the range of a sneeze.
4. The slight interference with the sense of smell, and the tendency of the secretion to accumulate and inspissate during the time the patient is in the recumbent posture.
5. The tendency of the secretion to flow down by the posterior nares, rather than by the anterior; which corresponds with the direction of the superior meatus, into which the sphenoidal and posterior ethmoidal cells discharge themselves.
6. The stinging and tickling of the uvula, and hard and soft palates, unaccompanied by any constant morbid appearances in them; which corresponds in position with the distribution of the spheno-palatine branches of the superior maxillary nerve, and may thus be accounted for as the transferred impression of irritation in the sphenoidal cells.

The duration of the complaint, when once established, appears to be quite indefinite, unless removed by treatment.

It seldom occurs before the period of puberty, which corresponds with the development of the sphenoidal cells.

The relief to the cough by taking food or drink is simply due to the removal, by these means, of the mucus clinging to the back of the fauces.

Among the serious effects of which this affection is sometimes the indirect cause, may be mentioned the production of hernia, the rupture of blood-vessels, strains to the lungs, and injuries to the internal ear from violent nose-blowing.

True Post-nasal Catarrh must be distinguished from the post-nasal discharges so common with children, which are of quite a different nature. They appear to be simply due to the difficulty which children have in effectually blowing their noses. Hence the nasal secretions, when augmented by cold, escape by the posterior as well as by the anterior nares, and during sleep almost entirely by the former. This discharge into the fauces, as in true Post-nasal Catarrh, gives rise to troublesome cough, which, through neglect of auscultation, I have often seen mistaken for Bronchitis, and much time and treatment wasted under this impression.



## ON SOME PRINCIPLES OF DIET IN DISEASE.

*A Lecture delivered to Advanced Students, at the Royal Infirmary for Diseases of the Chest, April, 1865,*

BY

HORACE DOBELL, M. D.,  
PHYSICIAN TO THE INFIRMARY.

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THE subject which I have chosen for to-day's lecture is one upon which it would be very easy to write a large book, but which is very difficult to treat in a short lecture. I must content myself, therefore, with condensing what I have to say into a few statements of opinion, avoiding all attempts at discussion, and if I appear to deliver my views dogmatically, you will, I hope, understand that it is not because I feel at all inclined to dogmatise, but because I am pressed by the extent of the subject, and the narrow limits of our time.

In my "Manual of Diet and Regimen"\* I have given "the Essentials of a Normal Diet" for health. The question now is, how ought these to be modified in disease? You will observe that these "Essentials of a Normal Diet" provide for the maintenance of healthy nutrition in a "healthy adult man of average stature taking moderate exercise," and, for simplicity sake, it will be best to make all our remarks apply to this "adult man of average stature" *under altered circumstances*.

The first and leading principle of diet in disease is, to provide for the maintenance of healthy nutrition, under the peculiar alterations of circumstances attendant upon disease.

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\* "A Manual of Diet and Regimen," p. 36, 3rd Edition.—Churchill.



In other words, the diet of disease should be as nearly that of health as the altered condition of the nutritive functions, and the altered conditions of the patient's life will allow; the grand object being to keep up healthy nutrition of the whole organism.

The true appreciation of this first principle, in its various bearings, will save us from running into either of those extremes which at different times have disfigured medical practice. We shall not be led to starve our patients upon water-gruel, when they are craving for natural food; or to stuff them with beef-steaks and porter, when every instinct of their nature rebels against it. In fact, you may take it as a very safe rule, that it is better cautiously to supply a patient with the kind and quantity of food that his stomach calls for, than to deny it to him without an unquestionably good reason for so doing.

This may seem to you very much like letting a patient eat and drink what he pleases. But that is not at all what I intend to recommend. All I mean is this, and I wish particularly to impress it, that if we mean to interfere in the subject of diet, we must take care that we thoroughly understand what we are about; and in order to do this it is necessary to keep well up in the following subjects:—

1. The physiology of healthy nutrition.
2. The composition of food, and the essentials of a normal diet.
3. The physiology of disease.

If we keep these matters well before the mind, and at the same time keep our wits about us in watching the case under treatment, it is surprising what an immense deal of good may be done by interfering with the diet; but not otherwise.

We start, then, with this as our first principle, never to be lost sight of—that healthy nutrition is to be maintained, if possible, under all circumstances. And we assume that to do this in a healthy adult man of average stature, taking moderate exercise, the essentials of a normal diet, as given in my “Manual,” must be supplied.

It must be borne in mind that the proportions and quantities of the different elements of this normal diet are arranged to meet the requirements of the different functions of the organism when in a healthy state of activity; and it



therefore follows, that if the activity of any of these functions is altered, the requirements will be altered; and hence, the second general principle is this:—To alter the quantities and proportions of the elements of a normal diet to correspond with any alterations in the conditions of life. Thus, when a man is overtaken by sickness, and confined to his room or bed, the adult man taking moderate exercise becomes an adult man taking no exercise; and the ingredients of his diet which were proportioned to his moderate exercise must now be proportioned to his no exercise; and other alterations must be made in like manner to correspond with other altered circumstances, in addition to any that may be specially required by the nature of his disease.

But I must remind you that, even when a man is confined to his bed, and precluded from taking any kind of exercise, he is still necessarily undergoing a considerable amount of muscular exertion, which must be provided for in his diet. For example, so long as life remains, such all-important muscles as those of respiration, and the heart itself, continue to act, and to require that their healthy nutrition shall be provided for by a supply of plastic materials in the food.

We come next to the long list of alterations of function which may be involved in the term "Sickness." And the third principle is—To alter the forms, quantities, and proportions of the elements of a normal diet, to meet the altered relations in the activity and condition of organs consequent upon disease. It is evident that, in order to carry out our first principle of maintaining healthy nutrition under all circumstances, it may be necessary, under some conditions, to reduce the quantity of every element of diet; and also, under some circumstances, to alter the proportions of the different elements. This we see demonstrated in some of the lower animals by the phenomena of hybernation. When an animal gives itself up to its winter sleep, every vital function is reduced to its lowest degree of activity; and the animal is able to maintain healthy nutrition for a long period without taking any food at all; but as respiration has to be kept up more actively than the rest of the functions, a special store of carbon for this purpose is laid up beforehand in the body. Now, supposing a man to suffer from any state of



disease which should place him in the position, as regards his functions, of an animal during hybernation, it is clear that, while his whole diet must be reduced to a very low scale, the respiratory elements must be supplied in quantities out of the normal proportions as compared with the rest; because no supply of carbon has been stored up in preparation for his illness, as it is in the hybernating animal in preparation for its sleep.

We see conditions, in many respects similar to these in some stages of fevers, in which absorption, nutrition, and every vital function is at its lowest point consistent with life, respiration being the only one sufficiently active to call for any considerable supply of food. But here, of course, we must not lose sight of an element in the case not present in hybernation—viz., the existence of a poison, which by some means, natural or artificial, has to be eliminated or destroyed, and which may be keeping some functions in activity, the requirements of which must be met. The precision with which we are able to do this in any given case, will depend upon the correctness of our knowledge of the nature of the poison, and of the organs concerned in the restorative process. Here, no doubt, we are often obliged to act in the dark, and to supply many ingredients which may not be needed, in the hope of furnishing among them that which is required, but which our ignorance prevents us from identifying. And we had far better, whenever our knowledge is at fault, act in this safe manner and supply much that may be useless rather than run the risk of withholding that which may be essential to life. But, in the majority of cases, our knowledge will be sufficient for the emergency, if we keep in mind the general principles of action.

The fourth principle is this:—To obtain rest for every organ while it is suffering under active disease, by removing from the diet such elements as increase its functions. These are conditions which it is not always easy to fulfil without deviating from our first principle. For example, in the case of diseased kidney, the healthy nutrition of this organ requires a supply of albuminoid materials, while its function is increased by any surplus of these materials in the organism; and when the function is interrupted by disease, a proportion of albuminoids in the diet, necessary to the



healthy nutrition of the organism generally, will be tantamount to an excess as regards the function of the kidney, and the accumulation of retained excretory matters will press injuriously upon the affected organ. In such a case other medical aids than diet must be brought to bear; and while the albuminoids in the food are reduced as low as is consistent with healthy nutrition, some auxiliary organs which are not damaged must be stimulated for the time, to save the diseased part from undue pressure upon its functions.

A simpler, but still important principle, may be stated as the fifth, viz.:—In all alterations of diet, to avoid any unnecessary reduction in the number and variety of the forms in which food is allowed to be taken. This becomes especially necessary to be borne in mind when dieting the dyspeptic, who are often still engaged in the active avocations of business and of society while under medical treatment. To treat such cases by cutting off from the daily bill of fare first one article and then another, till the food consists only of two or three permitted forms, is to destroy the appetite and the digestive powers by monotony of diet, and to depress the spirits of the patient by a constant series of petty denials. This plan of dieting can only be regarded as the resource of ignorance; because an enlightened view of the case will discover some particular defect in the function of digestion or assimilation which will at once indicate the form or element of the food which is to be avoided; and thus it will be only necessary to cut off those articles which specially represent this element, or simply to alter the forms in which they are presented to the stomach.

The sixth principle is also of great importance, viz.:—When it is necessary to remove from the food any of the essentials of a normal diet, to aim at selecting that which will answer the desired end with least danger to the nutrition of the vital organs. For example, if it is necessary for any special purpose to diminish the respiratory elements of the diet, it is safer to remove the carbo-hydrates than the hydro-carbons, because the latter not only supply carbon for respiration, but are essential to the nutrition of the nervous system, and of the albuminoid tissues generally.

The seventh and last principle which I shall give to-day is of very general application:—When it is desired to *increase*



the normal nutrition of a tissue or organ, we must not only supply it freely with the special materials requisite for its development, growth, and repair, but at the same time call upon it for the performance of its normal functions—over-fed idleness insures morbid nutrition, not healthy life.

In the next place I will give you a few *rules* which may assist you in carrying out these general principles—

Rule 1.—When the power of appropriating any essential ingredient of a normal diet is lost to the organism, the lost function must be substituted by some artificial process, or the ingredient in question must be withdrawn from the diet till the normal function is restored. In obedience to this rule you see me at this hospital administer pancreatic emulsions of fat to patients, who have lost the power of assimilating fat without this artificial assistance, while I adopt all practicable means of restoring the normal function.

Rule 2.—Is inseparable from the first, and it is this :—No essential of a normal diet must be withdrawn without an attempt being made either to supply to the organism in some other way the ingredients of which it is deprived, or to suspend those functions which call for a supply of this ingredient. Thus, to take a simple illustration :—Suppose the power of digesting meat to be lost through a deficient secretion of gastric juice, meat must be withdrawn from the diet till the lost function is restored, or else an artificial digestive fluid must be introduced; or if it is impossible by these means to maintain the digestion of meat, the physiological ingredients of meat must be supplied in the form of some albuminoid solution; or, finally, if this cannot be done, then those functions which principally waste the albuminoid tissues of the body must be placed as far as possible in a state of rest, muscular action must be suspended until the function is restored.

Rule 3.—If an undue waste of any elements of normal nutrition is found to be going on in the organism, and the means remain of appropriating those elements from the food, they must be supplied in the food, in quantities as much in excess of those proper to a normal diet of health as will be sufficient to supply the waste until it is stopped. This also may be illustrated by a very simple example. In Bright's disease of the kidney there is no loss of the power to appro-



priate the albuminoids from the food, whereas a constant loss of albumen is going on through the kidneys, which must be met by proportionate increase of the albuminoids in the diet. But in following this rule, in this particular case, it will be necessary to observe the precautions which I mentioned when speaking of the fourth general principle.

Rule 4.—When through any defect in the organism, the elements of a normal diet are lost to nutrition if presented in the usual forms, those forms must be changed; but care must be taken that in the altered form all the essential elements of a normal diet are supplied in their proper quantities and proportions. Nothing can illustrate this better than the use of milk as a substitute for solid or mixed foods in diarrhoea or sickness.

Rule 5.—Has to deal with more complicated difficulties. If such a defect exists in the organism that some of the essentials of a normal diet are misappropriated, so that the organism is deprived of some of the normal elements of nutrition, and at the same time a disease is constituted out of the misappropriated food, then we have a double duty in interfering with the diet. First, the source of the disease must be stopped by withdrawing that part of the diet out of which it is constituted; and, secondly, the elements of nutrition thus removed must be supplied by some other means or in some other form.

Thus, in Diabetes the saccharine and amylaceous elements of the diet are misappropriated; they do not serve their normal function of supplying carbon for respiration, and by passing off through the kidneys they constitute an exhausting disease. It is necessary, therefore, to stop the source of this disease by cutting off the saccharine and amylaceous ingredients of the diet till normal nutrition is restored. But, in the meantime, as carbon must be had for respiration, it is taken from the fat stored up in the body so long as that lasts, and when it is gone, from the albuminoid tissues themselves, till the whole organism is disintegrated; unless, at the same time that we cut off the starch and sugar, we increase the quantity of *fat* supplied in the food as much above the proportion proper to a normal diet as shall fully supply the demand.

The modern treatment of Diabetes may also be taken as a good example of the way in which increased knowledge of the nature of disease and of the physiology of food enables



us to act under what I have called the fifth principle of diet, viz., to avoid any unnecessary reduction in the number and variety of the forms in which food can be taken. In former days the poor parched diabetic was forbidden to drink water lest he should increase his flow of urine; now we are able to let him quench his thirst as much as he pleases, so that he takes nothing which contains starch or sugar; and again, by preparing his articles of food in such a manner as to exclude the injurious ingredients, and by selecting those which are known to contain them in the smallest quantities, or not to contain them at all, we are able to present the diabetic with a fairly tempting and varied diet, so that he is able to keep to it for months and years with comparatively little difficulty.

In conclusion I shall venture to give you a sixth rule, which I call the "golden rule," as it relates to the carrying out of the first of our principles throughout all the difficulties involved in the rest. The "golden rule" is this:—Never let a patient become weak through a defective diet if you can prevent it by any possible contrivance; and if a patient has unavoidably become weak, never let him remain so, through any defect of diet, one hour longer than it is absolutely impossible to prevent.

Other principles and rules I must reserve for future occasions, when treating of special cases as they come before us. But I hope that you will find, when you come to put them to the test of practice, that the few which I have given you to-day will serve both your patients and yourselves many a good turn, if you will only bear in mind what I have endeavoured to impress; that in order to carry them out satisfactorily you must keep up your knowledge of your profession generally, and especially of the various subjects which I have enumerated, as essential to a proper understanding of the alterations of diet required in disease.



FORM OF  
ENQUIRY INTO THE HISTORY OF WINTER COUGH.

BREATHING.	When did the breath first begin to be short on going upstairs or hills ?
	In what other way were you ill when the breath first began to be short ?
	What sort of health had you before the breath began to be short ?
	Since your breathing first became short, has it ever been otherwise than short ? If so, when ?
	What do you think most inclined to make your breathing short ?
COUGH.	When did you first have an attack of cough ?
	What sort of attack was it ?
	What else was the matter at the time ?
	How often and when have such attacks returned ?
	How has the breath been between the attacks of cough ?
	Have you ever been quite free from cough since the first attack ? If so, when ?
	When you catch a cold, does it affect first the chest, the throat, or the nose ?
	Describe the symptoms of the attacks of cold which leave a cough.
	What gives you cold most easily ?
	What gives you cold most often ?
	Is your cough much worse at times from any other causes than fresh cold ?
PAST HISTORY, &c.	What illness have you had within memory not already stated ?
	Do you attribute your complaint in the chest to either of those illnesses ?
	If you do, to which do you attribute it, and what reason have you for doing so ?
	If your mother is living, ask her these questions, and state what she thinks.
	If your mother is living, ask her whether you were a strong and healthy, or a weak and delicate child, and give her answer.



FAMILY HISTORY.

MOTHER.—If living, what age and what health ?  
 If dead, what age at death, and the cause of death ?  
 What health had she during life ?

FATHER.—If living, what age and what health ?  
 If dead, what age at death, and the cause of death ?  
 What health had he during life ?

BROTHERS.—How many living ?  
 What are their ages, and what health have they ?  
 How many dead ?  
 What were their ages, and the causes of death ?  
 What health had they in life ?

SISTERS.—How many living ?  
 What are their ages, and what health have they ?  
 How many dead ?  
 What were their ages, and the causes of death ?  
 What health had they in life ?

OCCUPATION.

What is your present occupation, and how long have you followed it ?  
 What other occupation have you followed, and at what periods ?  
 What are your hours for business and for meals ?  
 In what sort of place do you live by day ?  
 In what sort of place do you live by night ?

DIET HABITS, &c.

Do you take meat, and vegetables, and bread ?  
 Have you always done so ?  
 What fermented liquors do you drink, and what quantity per day ?  
 Have you always taken the same ?  
 Do you live regularly, and are your spirits usually good or bad ?  
 Do you smoke ?  
 How long have you done so ?  
 Has it any effect on your complaint ?  
 Has anything you do any effect on your complaint, and if so, what ?



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4. Apoplexy of the lung after coagulation.
5. Healed cavities, fibro-cellular cicatrices, and chalky concretions.
6. First stage of pneumonia.
7. Second stage of pneumonia.
8. Second stage of pneumonia passing into the third.
9. Medullary cancer.
10. Hard cancer.
11. Melanoid cancer.
12. Indurated lung, the effect of pneumonia.

## LIQUEFACTIONS.—

13. Second stage of acute bronchitis.
14. Chronic bronchitis.
15. Third stage of pneumonia.
16. Second stage of tuberculous disease.
17. Apoplectic coagula in the lung, softening.
18. Apoplexy of the lung before coagulation, with and without laceration.

## EXCAVATIONS.—

19. A large empty tuberculous cavity, with reflecting walls, free bronchial communication, and consolidated surrounding lung.
20. A similar cavity containing secretion.
21. A very superficial cavity, full of air.
22. Small tuberculous cavities, beneath a stratum of normal lung.
23. A large tuberculous cavity, beneath a thick stratum of densely-consolidated lung.
24. Emphysema (vesicular).
25. Bronchiectasis.
26. A gangrenous cavity.
27. A pneumonic abscess, partly emptied.

## PLEURISY, PNEUMOTHORAX, ETC.—

28. Normal lung, showing the interior of the bronchial tubes.
29. First stage of acute bronchitis.
30. First stage of acute pleurisy.
31. Second stage of acute pleurisy.
32. Third stage of acute pleurisy, compressed and displaced lung.
33. Pneumothorax.
34. The pathological causes and effects of pneumothorax.
35. Emphysema and pneumothorax.



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