examination sought. This important duty will be henceforth performed by him. But the Council has thought it desirable, with the yearly increasing demand, to retain the services also of a skilled pathologist, as a consulting officer in cases of unusual doubt or difficulty, and for the purpose of making necropsies under peculiar circumstances when required. Dr. Sidney Martin, Professor of Pathology in University College, has accepted this office, and we are happy to be able to secure the services of so high an authority when required. I may also add that I have made it a rule, whenever the circumstances permit, to avail myself of the coroner's court, and am always glad to shift the responsibility of the inquiry to the local coroner in all needful cases.

But after the most complete arrangements have been made for the management of our own institution, the first and most important object of our policy, as already explained, is the reform of the registration of the death system: and it is still the most pressing one before us.

The second is the construction at no distant period, of a new and completely-equipped crematorium, probably on the "Siemens" principle, in the near neighbourhood of London, and possibly by means of a limited liability company.

The third is the consideration by the Local Government Board of the question whether cremation, while of course remaining optional for all in every ordinary case of death, should not sooner or later become imperative in all cases of death caused by contagious disease in its worst forms, such as small-pox, scarlet fever, diphtheria, and malignant cholera at least—at all events in the chief centres of population, the cities and large towns of the United Kingdom. And for these a question might arise as to the employment of crematoria set apart for the purpose, provided with special antiseptic precautions to ensure freedom from danger to attendants or others frequenting them. This may be secured simply and easily by well-known practical arrangements; and of course with the same attention to decorum and to religious rites as at the existing crematoria. I advocated this mode of dealing with all zymotic diseases in a paper read at a large meeting of the International Congress of Hygiene, held in London in 1891, and a resolution strongly approving the proposal was carried in a large meeting of sanitary experts and medical officers of health.

REFERENCE.

¹ Reports on Death Certification, 1893, Eyre and Spottiswoode, London (373, 472).

THE LUMLEIAN LECTURES

ON

BRONCHITIS, PULMONARY EMPHYSEMA, AND
ASTHMA.

Delivered before the Royal College of Physicians of London.

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LECTURE II.

ON THE NATURE OF PULMONARY EMPHYSEMA.

IN 1764 Dr. William Watson (who afterwards became a Fellow of our College and a knight) read before the Royal Society "an account of what appeared on opening the body of an asthmatic person." He described what he called an emphysematous state of the lungs, "a condition which he deemed to be due to an extravasation" of air into the lungs from the "bronchia and vesicular substance," and what was no doubt vesicular emphysema of the lungs in the sense which we give to that phrase. Pulmonary emphysema has been known in one of its results—namely, the barrel-shaped chest from the time of the ancient Greeks. The lesion of the lungs themselves was first noted by Valsalva and Ruysch in the earlier part of the last century. However, Laënnec was not far from the truth when in the first edition of his great work published in 1819 he said that "emphysema of the lungs, such as he had described it, seemed to him to be a disease hitherto unknown."

It is universally agreed that the note of pulmonary emphysema is to be found in enlarged capacity of the acini and lobules of the lungs beyond what can be produced in health

by the deepest inspiration. This change in the structure of the lung, the anatomical characteristic of emphysema, is pretty thoroughly known; but when we come to inquire into the process which leads to emphysema the case is very different. For we cannot know the process in the same way as we know the result; we cannot watch the lungs becoming emphysematous; we can only argue about the process. Deprived of the intuition of sense, the light of Nature, we must follow the light of reason, and beware lest it prove to be *ignis fatuus*. And unfortunately we can get no help from experimental pathology; we cannot set up progressive emphysema in the lower animals and mark its stages in the same manner as we can set up inflammation. Hence the many theories of emphysema and long discussions which, however, do no dishearten me from attempting the difficult topic once more; my only fear is lest I should weary you.

ACUTE EMPHYSEMA.

The emphysematous process, like all Nature's processes, may be assumed to be complex, and we shall do well to begin by endeavouring to resolve it into its simplest elements. Nature itself helps us much by offering to our consideration a form of emphysema which certainly is simpler than ordinary emphysema and which seems to consist in a mere over-distension of the lung. I allude to the acute emphysema first adequately described by Fauvel in his essay upon capillary bronchitis. You know the condition—both lungs distended with air, as if insufflated by the fullest possible inspiration, but some parts of the lung distended more than this, emphysematous, in short, and especially so in the upper lobes and their anterior portions. You know that this acute emphysema is especially met with in young children who have died from suffocating capillary bronchitis or from diphtheritic croup. Many of these children will have enjoyed the most perfect health until they are attacked by the bronchitis or croup, so that there is no question in them of any preceding disease of the lungs. The suffocating bronchitis will often prove fatal within a week from its beginning; croup will kill in a still shorter time, and there is a disease, very much less common, namely, hæmorrhage into the air passages, which may suffocate in a few minutes, acute emphysema being found *post mortem* in all cases.

Let us turn our attention to croup in the first place, inasmuch as it is often entirely free from one condition which might complicate the question, namely, collapse of the lung. For although fatal croup is usually associated with abundant puriform secretion into the small air-tubes of the lungs, yet in spite of this form of bronchitis there is sometimes no collapse at all even when the lungs have become highly emphysematous, so that collapse when present can be no more than somewhat contributory to the emphysema. In other words, the emphysema is but slightly, if at all, compensatory to any collapse. John Cheyne, in his book on the pathology of the laryngeal and bronchial mucous membrane published in 1809, frequently alludes to the emphysema and puriform catarrh of croup. For example, he says: "The lungs were so filled that they did not recede in the least when the thorax was opened; every surface which was cut poured out this puriform fluid from several points."

Setting collapse aside, the conceivable causes of acute emphysema are two, namely, dyspnoea and cough; that is to say, the movements of dyspnoea and of cough, not overlooking the fact that they usually concur. Here also the question is simpler in the case of croup. In capillary bronchitis both dyspnoea and cough are great, and therefore the question of the associated emphysema is complicated. But in croup the dyspnoea is extreme, while the cough is insignificant, or even absent, so that it may be left out of consideration, and all this in cases affording a high degree of insufflation and emphysema of the lungs.

Our analysis thus leads us to infer that dyspnoea is the cause of acute emphysema. Now, the movements of dyspnoea are forced movements, inspiratory or expiratory, or both. But before proceeding further with this part of our subject let us cast a short glance upon the movements of breathing in general.

From the earliest times the diaphragm has been regarded as the essential agent of respiration—we may say of respiration, and not of inspiration only, because expiration is little

more than a result of inspiration. Galen was the first to show that most of the intercostal muscles were ordinary muscles of inspiration. Guided by the researches of Duchenne we may disregard the exceptions which Galen made, and we may say that all the intercostals act simultaneously, and are employed in ordinary quiet inspiration. They not only raise the ribs, but keep the intercostal spaces tight during the descent of the diaphragm. When the intercostal muscles are paralysed but the diaphragm is not, the upper two-thirds of the chest are motionless.

The chest may be roughly compared to a bow, inspiration to a bow when pulled upon by an archer, and expiration is little more than the elastic recoil of the bow when the archer lets go the string. But the inspiratory muscles do not altogether let go when expiration begins, they continue to act as moderators of the expiratory movements, and hence expiration is more gradual than it would be were it a mere elastic recoil. In short, the muscular contractions of expiration are persisting contractions of inspiratory muscles unless the expiration be forced, and then extraordinary muscles of expiration come into play.

Physiologists seem to be agreed that although the outward pressure of the air at the mouth during expiration is greater than the pressure inwards during inspiration, yet the forces concerned in inspiration are decidedly much greater than those of expiration. These propositions at first sight seem to be discrepant, but we are not called upon here to explain the apparent discrepancy—we will only remark that they indicate a provision in the human economy for repressing the greater powers of inspiration. Let us especially note that the elastic retraction of healthy lung is not satisfied by the deepest expiration and that the lungs are provided with a considerable reserve of elasticity, so necessary is it to maintain sufficient deflation of the lungs, or in other words to keep them sufficiently empty of air. For if there be insufficient efflation there must be insufficient inflation.

In the forced breathing of healthy persons there is the same disproportion between the powers of the antagonist movements. But physiological experiments relate to a few forced respirations studied in a healthy subject. The forced breathing of disease introduces a new set of conditions and requires that we proceed to an analysis. The extraordinary muscles of inspiration are chiefly three: the sterno-mastoids, the scaleni, and the upper third of the trapezii. These muscles can be seen contracting during inspiratory dyspnoea. Other muscles there are which assist to some degree, but to enumerate them is not necessary. These extraordinary muscles act by raising the upper part of the chest and fixing it so as to enable the ordinary muscles of inspiration to act with extraordinary power. The fixity of the clavicles and first ribs enables the intercostals to contract with their greatest power, and to elongate the chest in a direction upwards at the same time that the diaphragm elongates it downwards. Observation of the living patient shows us that forced inspiration tells chiefly upon the front part of the thorax above Harrison's furrow.

The extraordinary muscles of expiration affect quite another part of the chest. In a patient coughing violently we see the muscles of the abdominal walls, especially the recti, contracting so as to straighten and depress the front part of the chest below Harrison's furrow. The diaphragm being relaxed is necessarily driven upwards. If the largest air tubes be obstructed by mucus or by partial closure of the glottis—as in the case supposed of coughing—it is very instructive to see how forced expiration dilates exactly the same part of the chest as that which is dilated by forced inspiration. Hence in diseases attended by cough or dyspnoea the unfortunate upper and front part of the thorax has no chance—forced inspiration and forced expiration combine to keep it distended. Nature provides that the result of ordinary inspiration shall be fully counterbalanced by expiration; Nature provides extraordinary muscles to reduce the distension of the lower part of the chest caused by forced contraction of the diaphragm, but Nature provides no extraordinary means by which the injurious effects of forced inspiration upon the upper part of the chest can be repaired by forced expiration. On the contrary, both movements concur to the same end, or, in other words, no muscular contractions can reduce over-distension of the upper chest, and if

the inherent elasticity of the lungs and rib cannot reduce it nothing can.

Before leaving this topic, let us repeat that we must be careful to distinguish between the voluntary forced breathing of a healthy person and the involuntary, instinctive, automatic forced breathing which attends dyspnoea. Voluntary excessive movements soon fatigue, but automatic breathing is not attended by a feeling of fatigue, and so the extraordinary movements of dyspnoea can be continuously kept up for hours, days, or weeks—may we not say, indeed, for months or years? Moreover, let us note that this remark applies to the movements of breathing only, but by no means to those of cough; cough must be intermitting.

We will now return to the consideration of the acute emphysema of croup. In the living patient a very few hours of dyspnoea suffice to produce a marked bulging on the front of the chest above Harrison's sulcus. After the patient's death we find that the corresponding part of lung is the seat of a high degree of emphysema. The whole lungs are insufflated or distended with air, but nowhere is the emphysema so marked as in the situation just indicated. This is the very part of the chest which is most expanded by forced inspiration, and which is expanded by forced expiration also in case of cough, but expanded, let us note, by a different mechanism in the two cases. In deep inspiration this part of the chest wall is expanded in the first instance and the expansion of lung follows, whereas in forced expiration and in cough this part of lung is expanded in the first instance and it drives the chest wall outwards. It will be hardly necessary to recall to your memory the mechanism by which cough produces emphysema here, according to the doctrine elaborated by our former distinguished President, Sir William Jenner. The glottis is closed in preparation for a cough or for any great effort. Forcible expiration follows; certain parts of lung are compressed from which air escapes, but not being able to pass the glottis it is driven into those parts of lung which are not compressed, especially the upper anterior parts so often mentioned. Expiration under these conditions can doubtless produce emphysema in certain places, but in the case of croup inspiration is by far the more potent agent, being incessant in its operation, whereas cough is only occasional, and sometimes there is no cough at all. Moreover, forced expiration cannot explain the highly insufflated state of the whole of both lungs, for the distension caused by expiration must be partial.

We are yet far from having reached a full understanding of the mechanics of acute emphysema. We have arrived at an extraordinary expansion of part of the lungs, but the essential cause of the expansion lies deeper than this. You will understand what I mean when I ask this question, Why does the expansion remain permanent? And I think that from what has gone before the answer will readily come, Because Nature has omitted to provide the thorax with muscles by means of which this acute emphysema could be prevented or overcome. The only means which Nature has provided towards this end is the elasticity of the lungs and ribs, especially of the lungs, and this means, unfortunately, is inadequate. It is vain to object that physiological experiments prove forced expiration to be more powerful than forced inspiration, because these experiments deal only with the lungs as a whole, and cannot tell us anything about the respiratory powers of different parts of the lungs, which is the very thing we want to know. Can it be thought for a moment that the expiratory power of the upper part of the chest is greater than the inspiratory? Certainly not.

The elasticity of the lungs, then, falls sadly short of what is required of it during an emergency of dyspnoea. How is this? Either the elasticity is defective absolutely or it is defective only in respect of the increased demand made upon it, and probably both conditions concur; that is to say, the elasticity is called upon to do more than usual at the very time when it is itself weakened and able to do less. In the first place, this unusual demand upon the pulmonary elastic recoil is due to the abnormal secretions in the air passages, and the expired air has to overcome the resistance of these secretions in addition to what is the only obstacle to expiration met with in health—namely, the atmospheric pressure. The elasticity has too small a reserve; it is equal to overcome the atmospheric pressure, but not much more. Inspiration, being the more important motor of the upper lung, can

draw air past the obstructing secretions, of which air expiration can expel only a portion, and thus much air comes to be imprisoned in the parts which are, or which become, emphysematous. In the second place, the elasticity of the lung is probably damaged. We have already compared normal inspiration to drawing a bow, and it does not seem a very far-fetched similitude to compare the result of an enormous number of forced inspirations to a bow which has been overdrawn, bent, and crippled. The elastic recoil of the lungs is weakened. So far as I know no careful microscopic examination of the pulmonary tissues in acute emphysema has ever been made.

And now to sum up the conclusions at which we have arrived. The feeble expiratory power of certain portions of the chest is a condition which predisposes the underlying portions of lung to suffer from acute emphysema in cases of croup. Given this predisposition, the aforesaid portions of lung become overdistended in consequence of forced inspiration or of forced expiration, or of both.

I have purposely avoided interrupting the chain of reasoning by allusion to the opinions of others, for it seems to me that the argument stands in no need of any such support, whilst as to opinions which are more or less contrary I hope you will agree with me that the limited time allotted to these lectures can be better spent than by engaging in polemics.

The acute emphysema of suffocating bronchitis will now be easily understood. This affection is referred to by Aurelian in his chapter upon peripneumonia, where he says that as the disease gets worse the chest becomes more protuberant. The causes of the emphysema are the same as in croup, but with this addition, that in peripneumonic catarrh more or less collapse of the lung is common, and tends to aggravate the distension of the rest of the lung, according to the mechanism explained by Sir W. T. Gairdner.

CHRONIC EMPHYSEMA.

Acute emphysema is a condition which often ends in complete recovery. Chronic emphysema, on the contrary, is a progressive disease. Although it was first adequately described by Laënnec, it had engaged the attention of physicians long before his day, not, however, as a lesion found upon the *post-mortem* table, but as a disease of the living subject. Being looked at merely from the clinical point of view, emphysema was usually called asthma; for example, Heberden, speaking of what must have been chronic bronchitis and emphysema, says: "When a constant cough has afflicted people in youth and adult life, in their old age they become asthmatic."

Laënnec tells us that there was a time when he thought emphysema to be a very rare disease, but that afterwards he became convinced that it is common enough. Virchow supplies figures intended to show that emphysema is not common. In ten years only 0.3 per cent. of all patients admitted into the Charité Hospital of Berlin were admitted on account of emphysema; in only 0.6 to 0.7 per cent. of the bodies examined *post-mortem* was emphysema the chief lesion found, and in only 5.5 per cent. was there any emphysema at all. The statistics of St. Bartholomew's Hospital yield these figures: in four years (1894-1897) 9,928 patients were admitted into the medical wards; 165, or 1.66 per cent., were believed to be emphysematous; 1,363 *post-mortem* examinations were made, and emphysema was found in 154 bodies, or in 11.3 per cent. Our figures justify Laënnec's opinion; a lesion found in 11 per cent. of persons dying in the medical wards of a hospital cannot be called uncommon.

The anatomical sign of chronic emphysema consists in actual loss or atrophy of the lung tissues, yet many pathologists of the first rank have expressed a different opinion—namely, that there is a hypertrophy of lung tissue, either a hypertrophy preceding the atrophy or a local hypertrophy simultaneous with local atrophy. Laënnec himself thought it highly probable that emphysema is usually accompanied by a certain degree of hypertrophy, finding that the lung feels firmer and less flexible when pinched between the fingers. Yet he recognised the associated destruction of tissue, as, indeed, such a man could not help doing when he examined emphysematous lungs in the manner which he recommends—namely, by inflating and drying them. Laënnec's contemporary, Louis, was less sagacious: he admits no exception to his universal affirmation that

emphysema implies increase or hypertrophy of lung tissue. When we ask how Louis detected the pulmonary hypertrophy we get no clear information; he seems to have done no more than adopt Laënnec's methods and to have examined the lung by pinching it and by drying it. These doctrines are not altogether incorrect, only we must express them a little more precisely. In the first place, emphysema in its earlier stages does not affect the whole lung or does not affect it equally. And next, the great and extensive atrophy of tissue cannot be gainsaid in the case of emphysema in its later stages, the lungs being examined according to Laënnec's method; they feel as he says, no longer firm and inflexible, but soft and downy, and the loss of tissue is most obvious to the eye. Induration is a better name than hypertrophy to give to the abnormal firmness and rigidity of the alveolar walls, and this induration is always very partial, and found only in the earlier stage of the disease.

The microscope has not yet disclosed any primitive tissue changes apart from mere atrophy, if such changes there be; and I need not refer to hypotheses such as that of Rainey, who deemed the primary lesion of emphysema to consist in fatty degeneration of the alveolar walls, or that of Villemin, who described a sort of granulation of the connective tissue which supports the capillary network of the lungs, for in all probability what both these anatomists saw were degenerative changes in the epithelium—changes secondary to a preceding failure of nutrition, whatever the nature or cause of that failure may be. Other pathologists have supposed a primary affection of the capillary blood vessels or of the unstriated muscular fibres or of the epithelium, but the truth is that the earliest changes are very imperfectly known.

We are thus led on to survey the great field of strife in the topic of chronic emphysema. There is rarefaction of lung and there is destruction of lung. Does one cause the other, and if so how? And this is not all; we desire to look deeper still, and we ask how the primary defect, whether it be rarefaction or destruction, is brought about—to what is it due? In other words, what is the essence of emphysema? We wish, if possible, to witness emphysema in the making, and inasmuch as morbid anatomy can show us no more than the final result, I invite you to discuss the subject with me from the clinical point of view.

First, we will inquire what means we have for discovering the existence of emphysema in the living subject, and we are met at the outset by the fact that most of these patients suffer from manifest bronchitis also. These complex cases are not suitable for our present purpose; we cannot disentangle the symptoms of the two diseases so as to be able to say this is due to emphysema and this to bronchitis, whilst as to the physical signs of emphysema there are none, excepting in advanced cases, which are always complicated with bronchitis. If we require instances of emphysema quite unattended by catarrh, such instances are not to be found. However, let us consider a case of marked progressive emphysema in which the indications of bronchitis and catarrh are the least possible. A man about middle life begins to suffer from slight shortness of breath on exertion; he is short of breath—that is to say, the patient feels that he cannot inspire so much breath as he seems to need, in a sense like that in which we say that a man is short of money. His breathlessness increases slowly and insidiously until his breath is always short, but no signs of dyspnoea, no laborious or extraordinary movements of respiration, are apparent so long as he is at rest, not until he makes some effort, such as that of talking or moving the body. In cases still worse dyspnoea persists even when the patient is at rest. The dyspnoea of emphysema, like most other kinds of permanent dyspnoea, is liable to paroxysms in which the shortness of breath becomes very distressing. These paroxysms are induced by any effort, such as hurrying, or even no more than straining at stool, raising the foot to enter a carriage, or lifting the arm to shave. A paroxysm will sometimes occur during sleep, and will suddenly awaken the patient, or will sometimes occur just as he is falling off to sleep. The mere taking of food may be enough to provoke an attack; so will any sudden change in the temperature of the air breathed, such as occurs on leaving a hot room. The paroxysm is sometimes very sudden and severe, and the patient's description of it sounds very much like that of an

attack of angina pectoris—indeed, the heart probably does participate, for I have known a pulse which was habitually about 80 fall to 60 or 70 during the paroxysm.

Now, suppose a patient suffering from progressive dyspnoea of this kind and from no other symptoms, no cough, no expectoration; and suppose we find by examining his chest that his lungs are enlarged, or, in other words, that he presents the physical signs of emphysema, we might well ask whether he were not the subject of protopathic emphysema. And some physicians say that they have met with such patients, and that therefore they believe in the occurrence of emphysema, pure and simple and uncomplicated. I cannot say that this has been my experience. I have never known this progressive dyspnoea and emphysema to be unattended by cough and expectoration.

The cough is a tickling cough (*tussis titillatoria*) brought on by a troublesome sensation referred to the larynx. A person affected in this manner and who, in the spirit of the age, diagnosticates the nature of his own disease goes to a specialist who snips off the tip of his uvula or discovers an irritable spot in the larynx, but fails to afford relief. The cough, like the dyspnoea, steals insidiously upon the patient until in the course of years he becomes liable to fits of coughing which are very severe, during which he becomes purple in the face and sometimes even suffers from giddiness—giddiness which is in any degree of intensity, from an attack so slight that the man declares that he has retained his sensibility throughout to an attack which amounts to total loss of consciousness, in which latter case the patient sinks to the ground and sometimes hurts himself in his fall, but even at its worst the coma does not last more than two or three minutes. The expectoration which attends the cough is very scanty and consists of pure mucus. Or it may be that there is no expectoration at all, or, at any rate, no expuition.

Aretæus describes a class of persons whom he designates "pneumodes," a word which Stephanus interprets to mean *pulmonis vitio laborantes* or *pulmonarii*, and which seems to have the same meaning as the French word *poitrinaires*. Now, if it were not for one particular in Aretæus's picture of the disease we could not doubt that his "pneumodes" were cases of progressive emphysema. The disease, he says, is a kind of asthma, and there is little difference in the symptoms of the two diseases; all patients labour under shortness of breath as in asthma, but they have this as peculiar, that they cough as if about to expectorate, but their effort is vain, for they bring up nothing; or if anything is forcibly raised from the lungs it is a small, white, round substance resembling a hailstone. The thorax is broader than natural, but not altered in shape; the lungs, though free from suppurations, are filled with humours; in some cases the affection terminates in dropsy about the lungs or in anasarca. But the pneumodic disease proves fatal within a year from its onset, and this is so grave an objection to our supposing that Aretæus was referring to cases of pulmonary emphysema, that I should not have alluded to his description were it not for the peculiar sputum which he depicts, and which possesses the same characters as those of the sputum expectorated in the affection which Laënnec denominated "*catarrhe sec.*"

All physicians admit a close connection of some kind between bronchitis and emphysema—a connection in many cases so obvious as to be past all doubt, but in other cases not so. Laënnec went so far as to say that emphysema is almost always the result of intense and extensive *catarrhe sec.* All writers on emphysema mention this opinion of Laënnec's; few if any, adopt it or even discuss it, but no opinion of that remarkable man can be passed over lightly. What does he mean by dry catarrh? He himself admits that the phrase is a contradiction in terms; to find appropriate names for diseases was not a strong point of his; witness his perversion of the meaning of the word "apoplexy" in his phrase "pulmonary apoplexy." But medical writings are full of this fault, and Laënnec is no worse than many other physicians. A better name than dry catarrh would be dry bronchitis (*bronchitis sicca*), for the word "*siccus*" as applied to pulmonary diseases denotes scantiness or absence of expectoration. Stoll speaks of "*pleuritis sicca*" and "*pleuritis humida*"; Hippocrates of "*dry orthopnoea*," and the phrases "*dry cough*" and "*dry asthma*" are colloquial.

In the lungs of persons who died in an early stage of dry

catarrh, Laënnec used to find the mucous membrane of the bronchia swollen and dull-red or violet in colour—conditions most notable in the smaller bronchia, which might be almost completely obstructed by the swelling. The extent of the swelling was not more than a few lines, the mucous membrane in the same tube above and below being natural. This local bronchitis was extensive in proportion to the duration of the disease. When the dry catarrh was universal, or even very extensive, it always ended by determining emphysema. He often found the partially obstructed tubes to be plugged by pellets of viscid mucus of the size of hemp or millet seeds, and it is these mucous pellets which make up the pearly sputa which such patients, even when they think there is little or nothing the matter with them, expectorate in small quantity every morning.

During life the dry catarrh often continues to a slight degree for many years, Laënnec goes on to say, or it may be altogether latent, the patient noticing no more than that his breath is shorter than that of other men, especially upon exertion. This dyspnoea increases gradually, and the patient becomes subject to attacks bad enough to deserve the name of asthma. The tendency to dyspnoea is accompanied by cough, which at the beginning of the disease is, like the dyspnoea, slight. The cough is attended by expectoration of what Laënnec calls pearly sputa, which closely resemble the hailstone sputa (*chalaixia*) described by Aretæus and Galen. Such is a brief summary of Laënnec's characterisation of his *catarrhe sec.*

Now, if we admit that a man in a state of perfect health ought not to cough or expectorate, and also that expectoration implies catarrh, it follows that at no period in the progress of emphysema is that condition unassociated with catarrh. Again, inasmuch as emphysematous patients always become in the advanced stage of their disease manifestly bronchitic or catarrhal, it is reasonable to believe that in the early stage also their cough and expectoration indicate slight bronchitis or catarrh. Lastly, the symptoms of progressive emphysema (as I have already attempted to depict them) and those of Laënnec's dry catarrh seem to relate to the same disease looked at from two different points of view.

We have now reached a position which enables us to resume our discussion of the nature of emphysema. Starting from the note of progressive emphysema—namely, the atrophy of lung tissue—the problem may be resolved into a choice between these alternatives. Is there a primary degeneration of lung tissue, rendering it apt to be permanently distended and also to be destroyed particle by particle? Or is the atrophy due to distension of the lung, and, if so, what causes the distension? In briefer language, is the atrophy primary or secondary? The easiest answer is that which assumes the atrophy to be secondary to distension. Laënnec asserts that "pulmonary emphysema is almost always developed in consequence of intense and extensive dry catarrh," and we have seen that clinical observation supports this opinion. But in speaking of the priority of catarrh we must understand it only as a priority of order and conception and not as a priority of time. Another argument in favour of Laënnec's opinion is this, that inasmuch as acute emphysema is undoubtedly dependent upon bronchial affections, it is probable by analogical reasoning that chronic emphysema likewise is so. I will not repeat Laënnec's explanation, which you all know, concerning the manner in which he supposes dry catarrh to lead to emphysema, and I will only remark that his hypothesis is not so easily set aside as some people think by pointing out that the air-current at the mouth of a healthy man is more powerful during expiration than during inspiration, for the conditions in pulmonary catarrh are obviously not the same.

Let us fix our attention upon the fact of the barrel-shaped chest in cases of emphysema. It is a chest more capacious than a healthy chest could be rendered by the deepest and most powerful inspiration. The distended chest is a sign of distended lungs, and this fact puts before us a disjunctive hypothesis: either the distended chest distends the lungs, or the distended lungs distend the chest. Now the latter hypothesis affords no explanation at all, because it leaves the distension of the lungs unexplained; and the cause of this pulmonary distension or emphysema is the very thing we are asking for. Wherefore let us consider the former hypothesis, namely, that the distension of the lungs is consequent upon

increased capacity of the thorax. In normal inspiration which fact precedes the other, dilatation of the lungs or dilatation of the chest? And by what powers can the chest be over-distended? Obviously by the inspiratory muscles, ordinary and extraordinary, and by no others. And if the chest be over-distended, what must ensue? Over-distension of the lungs, because the potential vacuum caused by extraordinarily deep inspiration is certainly not filled up in any other way.

In health the inspiratory distension of the lungs is exactly counterbalanced by the expiratory contraction. Why, then, in the barrel-shaped chest is this no longer the case? Obviously because the expiratory powers have been unequal to the task of restoring the chest to its natural shape and size. That is to say, the expiratory powers are no longer equal to the inspiratory. And is not this a sufficient reply to those who, relying upon spirometrical observations made upon healthy people, reject Laënnec's explanation of the mechanism of emphysema?

In emphysema, then, there is failure of expiratory power; nay, more, this is the essence of emphysema. Expiration being no longer able to rid the lungs of their exhausted air, ordinary inspiration cannot supply the necessary quantity of fresh air. And there is only one means by which the expiratory failure can be met, and that is by inspiratory movements extraordinary in power and amplitude. These extraordinary inspirations being repeated for weeks, months, and years, the chest becomes permanently over-distended, and hence the lungs become permanently over-distended, and this is emphysema.

We have no clear and certain knowledge respecting the nature of the expiratory failure. We may conceive it to be absolute or relative. Absolute—that is to say, the elastic and muscular movements of expiration are weakened: relative—that is to say, the resistance which expiration has to overcome is increased. 1. The notion of an absolute debility of expiratory movement must relate to the elasticity of the lungs only, because there is no reason for supposing the elastic or muscular movements of the chest to be primarily weakened. And thus we are led back to the question of degeneration of lung tissue, either primary or secondary to other disease—an obscure topic which we will again defer for awhile. 2. But the notion of a relative debility of expiration is that which lies at the bottom of Laënnec's doctrine, the resistance to expiration being increased by the presence of mucous plugs in the air tubes and by swelling of the mucous membrane. True, reply his critics, but these obstructions are equally great for inspiration as for expiration, and thus we are again met by the question concerning the relative powers of inspiration and expiration—a question which is answered by reiteration of the fact that Nature has provided us with very feeble means of reducing by forced expiration the distension of the chest and lungs produced by forced inspiration. The extraordinary muscles of expiration are inadequate, whilst as to the elastic tissues of the lung we may hazard the conjecture that if overstrained they do not easily recover, and it is only inspiration that can overstrain them.

Now, just to consider for a moment the question whether the over-distended chest can possibly be due to over-distended lungs. In other words, can we assume a primary over-distension of the lungs powerful enough to distend the whole thorax and to make it barrel-shaped? If so, what distends the lungs? Forced expiration is sufficient to distend certain portions of lung which are unsupported by bone or muscle, such as are the apices. But more than this is inconceivable; it is inconceivable that expiration, how forcible soever, can produce a shape of chest which is identical with that produced by forcible inspiration. Again, a favourite seat of emphysema is the undermost surface of the lung where it lies upon the diaphragm; how can coughing—an act during which the diaphragm is driven powerfully upwards—over-distend this portion of lung? Surely it is the powerful inspiratory descent of the diaphragm which produces this effect; and let us remember that the forced expiration of persons coughing, of musicians, or of horses tugging up a hill is rendered possible only by a preceding forced inspiration. In short, we cannot understand the occurrence of a primary over-distension of the lungs sufficient to distend the thorax.

Now to discuss and finally to dismiss the question of the degenerative lung changes. In the first place, it is probable that the elasticity of the lungs is injured if the tissues be much or often overstretched, and any such loss of elasticity must tend to increase emphysema by weakening expiration. In the second place, it is very reasonable to believe that the anæmia, which is the visible and undoubted result of over-distension of any portion of the lung, will cause degeneration and atrophy of the tissues thus imperfectly supplied with blood. In the third place, it is possible that there is such a lesion as primary atrophy of lung, analogous to the atrophy and perforations which we often observe in the omentum, falx cerebri, and valves of the heart. In the fourth place, it is not unlikely that the form of pulmonary emphysema in which the total size of the lung is not increased, "atrophous emphysema," is an atrophy of the second or third kind. And, lastly, there are good grounds for believing that even ordinary progressive emphysema with enlarged lungs is from its very beginning an atrophic process, even although to the mind's eye the destruction be secondary to the distension.

To end our inquiry by recapitulating the results at which we have arrived, I submit to your judgment these propositions: That pulmonary emphysema is chiefly due to forced inspirations, although forced expiration may to some small extent play its part. That the distension of the lung in so far as it is due to inspiration is secondary to distension of the chest. That the forced inspiration is rendered necessary by a feeling of dyspnoea. That the dyspnoea which occurs at the beginning of pulmonary emphysema, and which determines it, is consequent upon obstruction of the air passages. That in chronic progressive emphysema this obstruction depends upon bronchitis, either humid and attended with free secretion or dry with scanty secretion. That when once emphysema is set up, the dyspnoea and the necessity for forced inspiration are increased by the natural defect of expiratory power in the lungs and in certain parts of the chest, Nature having provided means insufficient for reducing excessive inspiratory distension to the normal, and for thus emptying the lungs of residual air. That the degeneration and atrophy of lung tissue are usually dependent upon preceding over-distension, although it is possible that pulmonary atrophy may to some extent be primary.

Allow me to conclude by appropriating the words of the most ancient of the Greek philosophical poets: "These opinions have seemed to me to resemble the truth."

LECTURE III.

ON THE NATURE OF ASTHMA.

I PROPOSE to devote this day's lecture to a discussion of the nature of asthma. The topic you will, perhaps, consider to be almost exhausted. However, I invite you to the discussion because even if we cannot predicate all that asthma is, yet the time has come, I think, when we should finally dismiss certain opinions which have for two centuries impeded the attainment of clear notions upon the pathology of the disease.

Before proceeding to the argument it is desirable that we should come to an agreement as to the meaning of the word "asthma." It was used in a simple etymological sense until the time of our distinguished Fellow, Dr. Thomas Willis, in the middle of the seventeenth century. Asthma before his time denoted any kind of panting, gasping, pury breathing, such as follows upon running or exertion. Hence asthma and dyspnoea were synonymous for most of the older physicians. A few, such as Celsus, signified by asthma the highest degree of dyspnoea, but this was all; asthma was never regarded as a special sort of dyspnoea. In reading the writings of the ancients we must always bear this in mind, and beware of importing into their generic use of the word the special meaning which it bears in modern times. Indeed, so far as my limited reading enables me to judge, I should say that what the ancient physicians have left us concerning asthma is of very little value. It is interesting to note that those most conservative of people, the illiterate, continue to use the word in the sense of Hippocrates and Galen.

Thomas Willis was the innovator upon the doctrine of the ancients. His opinions, in their mature form, are found in the second part of his *Rational Pharmaceutic*, published in

1675, after his death in the same year. He says, truly enough, that the ancient physicians and also most moderns up to his time had admitted only one kind of asthma, that which he calls pneumonic asthma, and which had always been deemed to be the result of the air passages of the lungs being obstructed or not open enough. It would be a more accurate expression to say that the ancients regarded all cases of asthma as pneumonic and dependent upon bronchial obstruction. From this pneumonic asthma Willis distinguishes a second kind which he calls convulsive asthma, the primary fault being in the motive organs of the lungs, to wit, in the moving fibres or muscular coats of the air vessels or in the diaphragm and muscles of the chest or in the nerves of the lungs and chest or in the origin of those nerves in the brain. There is also, he says, a mixed asthma, both pneumonic and convulsive, and inveterate asthma is usually of this kind.

You will observe that Willis's doctrine is very comprehensive, and that he vaguely anticipates all the theories of asthma which have been propounded since his day. His new notion of convulsive asthma was soon laid hold of; the term "asthma" came to be reserved for the exclusive denomination of that form of the disease which was believed to be spasmodic; and this is the sense in which the word is still used by most persons even in our own day. Willis's influence upon this question of pathology has been so great that the present lecture is to a large extent an endeavour to determine whether he were the discoverer of an important truth or the parent of a delusion. A satisfactory definition of the word "asthma" will be the end of our discussion if we succeed in attaining a clear and distinct notion of the nature of the disease, for, as Milton, writing in the spirit of both Plato and Aristotle, says: "Definition is decreed by logicians to consist only of causes constituting the essence of a thing." But meanwhile we may perhaps be able to agree upon a sort of provisional definition logically less exact. Thus, some diseases are defined accurately enough by means of what are called their anatomical characters, or the attendant lesions of structure, and some diseases are defined less accurately by their symptoms or the attendant lesion of function. Let us examine asthma from these two points of view.

First, what is the morbid anatomy of asthma? Here I must ask your attention to a very curious and remarkable fact. You may look through a large book upon asthma, an elaborate treatise deserving the repute it has gained, and you will find no chapter dealing with morbid anatomy. Yet surely some asthmatic patients die uncured, and some of their bodies are examined, and in all the bodies examined material lesions are found. What are they? Now many of these lesions are found in some bodies only, and therefore cannot have anything to do with the immediate cause of asthma. Some of these inconstant lesions are those which are apt to occur in the decline of life; and it is noteworthy that Rostan, the discoverer of softening of the brain, and who practised at the Salpêtrière in Paris, the great hospital for old and infirm women, after asking the question, Whether the asthma of old people be a nervous affection? replies as the invariable result of his *post-mortem* examinations—That the asthma of old people is a symptom of an organic lesion, and especially of atheroma of the aorta and of hypertrophy of the heart. Most physicians of our day would probably agree that Rostan's opinion is correct so far as its general principle is concerned—namely, that organic lesions in the chest are found—but he errs in his particulars by being far too exclusive; several morbid changes are much more frequently met with than those which he mentions. Again, such lesions as tuberculous affections of the lungs, phthisis (active or retrograde), and acute miliary tuberculosis, though often seen in persons who are not old, cannot be deemed essential to asthma. But signs of bronchitis and pulmonary emphysema are always found; and though no one would call them the anatomical characteristics of asthma, yet the fact that they are constant demands that they should not be passed over or dismissed with the remark that they are mere complications, an obvious begging of the question. That the bronchitis of asthma should be regarded as an accidental complication and by no means as an essential element of the disease is due to the fact that, since Willis's time there has always been a strong tendency to look upon asthma as something mysterious and its cause as supersensual. However, you will probably assent to the proposi-

tion that either bronchitis and emphysema constitute the morbid anatomy of asthma or it has no morbid anatomy. In either case we must reluctantly conclude that structural lesions cannot help us to a definition.

Secondly, definitions based upon symptoms or disordered functions observed in the patient during life are seldom satisfactory. In the case of asthma, any definition of this kind would involve so many explanations, reservations, and distinctions that it would be nothing less than a full description of the disease. I will not attempt to succeed where all nosologists have failed, and I fear that we must confess that symptomatology cannot help us to a definition.

So we will proceed to our investigation in the belief that, in the case of a given patient most physicians would agree whether he were suffering from asthma or not, and that we have a pretty unanimous though an indefinable notion of the disease. Yet we must admit that the notion has been inconstant, that the extent of meaning of the word has been growing narrower and narrower. Much of what Willis called pneumonic asthma has long since ceased to be called asthma at all; much of what, by way of exclusion, was formerly deemed to be convulsive asthma is now known to be dyspnoea due to disease of the heart, blood vessels, kidneys, to poisoned blood, and other conditions which need not be enumerated. Thus there is a risk lest asthma should become a mere *caput mortuum*, a kind of dyspnoea left when all known causes of dyspnoea have been separated, the definition of the word being purely negative, indicative of what asthma is not. But asthma is not a mere *caput mortuum*.

Inasmuch, then, as our most trustworthy guides in all the physical sciences, our senses, seem insufficient to enable us to explore the essence of asthma either in the living body or the dead, we must try to grope our way by the help of reason. And we will recur to the rival hypotheses already mentioned and will debate the question which remains much as Willis left it, namely, whether asthma is a pure affection of the nervous system or whether it is all pneumonic, due, that is to say, to gross material changes in the lungs, or whether it is a combination of the two.

I. In the first place, let us discuss the nervous, convulsive, or spasmodic hypothesis.

(1) That the air tubes possess muscular coats or moving fibres was well known to Willis. That these fibres will contract under appropriate stimulus so as to diminish the capacity of the bronchia, has been demonstrated many times by experiment, the vagus being the motor nerve. But farther than this our knowledge does not extend. From the best and latest works upon human physiology we cannot learn what the use of these fibres is, nor under what conditions they contract, nor whether they contract quickly or slowly, nor whether their contraction is momentary or enduring, yet surely we ought to be able to answer these questions before we can deduce a pathology of asthma from perverted function of these fibres. The prevailing feeling is that they help the bronchial walls to resist any unwonted pressure from within—that they oppose distension of the air tubes. But even supposing this opinion to be true we are no nearer to understanding the part which these fibres have been assumed to play in the production of asthma. We will admit that it is conceivable that their contraction may cause dyspnoea, but we must make the reservation that there is no proof of this assumption. And one thing seems to be certain—namely, that if there be such a disease as bronchial spasm, pure and simple, it must be a very rare condition, because it is inadequate to explain the catarrh which manifestly attends most, if not all, asthmatic seizures. Willis himself was far too sagacious a man not to have seen the meaning of this fact. He writes thus: "Although an asthma is sometimes simple from the beginning, namely, either merely pneumonical or convulsive, notwithstanding, after either disease hath for some time increased, for the most part it gains the other to itself, hence it may be concluded every inveterate asthma to be a mixed affection." But we have already seen good reason for doubting whether this doctrine of Willis's goes far enough. The whole subject of bronchial spasm is involved in so much obscurity that we cannot admit more than that it perhaps takes a share in the production of asthma, but is seldom, if ever, the chief or only cause. And we will dismiss this topic by adopting the words of an able physician of the last century, Robert

Whytt, "that a true nervous or spasmodic asthma, without any other fault in the lungs than uncommon delicacy or irritability of their nerves, is a disease which we seldom meet with."

(2). Let us come to the muscles of respiration. When I began to lecture on medicine at St. Bartholomew's Hospital it seemed necessary that I should expound some theory of asthma. I had been much struck by observing in bad attacks of asthma the fixed immobility of the chest in a state of the fullest possible inspiration. About the time I came across a paper by an experimental physiologist who had found that centripetal excitement of the vagus, divided below the junction of the superior laryngeal nerve, produced tetanic contraction of the diaphragm, intercostals, and other inspiratory muscles, a state of things which is necessarily accompanied by dyspnoea, and which, in this respect, resembles the asthmatic paroxysm. And so for a year or two I taught that the fit of asthma, at its beginning at least, was due to centripetal irritation of the vagus. But maturer experience soon showed me that this fixed inspiratory distension of the chest is by no means a constant attendant upon asthmatic dyspnoea, that the inspiratory movements are sometimes free and ample enough, and thus I lost all faith in the doctrine of protopathic inspiratory spasm. Moreover, what is the evidence of inspiratory spasm during the attack of asthma? No doubt the chest is in a state of inspiratory distension, dilated to its utmost capacity and fixed in this position. But it is more likely that this state of things is due not to inspiratory spasm but to expiratory debility. Air is imprisoned in the lungs, and no amount of forced expiration can empty them. In the last lecture I showed what a very partial process forced expiration is; over many portions of the lung it has no power. If the sufferer could empty his lungs he could fill them again; it is the deflation which is at fault, and the extreme inflation is due to incessant forced inspiratory efforts to make the most of what little room for fresh air can be provided by forced expiration. This fact is very well expressed in a tale told by Dean Swift of a farmer who, when dying of asthma, said: "Well, if I can get this breath once out I'll take care it shall never get in again." And thus to conclude this topic I submit that inspiratory spasm is not the cause of the asthmatic paroxysm.

(3) Again, Willis thought that convulsive asthma might sometimes proceed from irritation of the pulmonary or thoracic nerves, or of the origin of these nerves in the brain. This is one of those recondite propositions which can be neither maintained nor refuted. Contraction of the bronchial muscular fibres or of the respiratory muscles, if it play any part in the asthmatic paroxysm, may doubtless be due to irritation of appropriate nerves; but the muscular contraction itself has not been proved to occur, and this must be done before we need discuss its causes. The influence exerted by the nervous system upon secretion, nutrition, and the blood supply is well known as a general fact, but any particular application of this fact to explain the pathology of asthma is at present beyond our power. Thus, we have no evidence to show that asthma takes its origin from the nerves or brain.

II. But if asthma be not convulsive, we are thrown back upon the hypothesis of pneumonic asthma, for we have no third explanation before us. Let us review the argument in favour of pneumonic asthma. This argument is wholly analogical. There are several diseases which resemble asthma in certain respects, more or less. Perchance we understand the pathology of these diseases somewhat better than that of asthma. If so, we may inquire whether the nature of asthma can be assimilated to that of the diseases which it resembles. It is obvious that such an argument as this is far enough from deserving the name of induction, but we will be comforted by the saying of a wise man, that to us probability is the very guide of life. Let us enumerate and illustrate the diseases referred to.

1. *A Peculiar Form of Acute Bronchitis*.—"Last summer I went out of town to see a little boy, 7 or 8 years of age, whose life was very precious to his family. He was thought to be dying of inflammation of the lungs. I found him raised up in bed, supported by his nurse, and breathing with all his might. His skin was hot, his face flushed, and his chest heaved, and his nostrils quivered frightfully. There was no croupy sound. Whatever the disease was it was all within

the chest. I percussed the chest; it sounded well in every part. I listened; the air entered freely, and reached every cell and vesicle of the lungs, but there was not the least perception of the natural respiratory murmur; a shrill sibilus had taken place of it altogether. Wherever you applied your ear to the chest you might fancy you heard the piping and screaming of a nestful of unfledged birds." Probably many of you will have recognised the hand of Dr. Peter Mere Latham in the somewhat rhetorical character of the description I have just read. I have set before you his description of the disease because it serves as an emphatic text and not because the affection is very uncommon; it is alluded to by many of the best authors, and I myself have seen a considerable number of cases. Latham goes on to say: "I am speaking of a disease which must be distinguished from asthma according to the usual acceptation—a disease not habitual to the individual, and of which, perhaps, he has never suffered a previous attack." But of course an asthmatic must have his first attack, to which Latham's criterion of habit cannot be applied, and unless he had told us that his patient had never before suffered from a similar attack and never had one afterwards, his argument that the disease was not asthma falls to the ground.

Latham's patient certainly suffered from acute dry pulmonary catarrh, to use the phrase of Laënnec; but in this form of disease the catarrh is not always dry; on the contrary, small mucous rales are sometimes heard all over the chest. I saw a girl, 4 years old, who one morning had a little cough, but was quite well in all other respects. In the afternoon she suddenly became much worse, and her state alarmed everybody. In the evening her distress, dyspnoea, and lividity were great; the upper part of the chest expanded considerably on inspiration, but there was at the same time much depression of the roof of the neck, of the epigastrium, and of the base of the chest. All over the lungs small mucous rales were heard. Her voice and cry were natural, so that there was no reason for suspecting laryngeal disease. Her temperature was below the normal. I was told that she had suffered from several sharp attacks of what had been called "bronchitis" and "congestion of the lungs;" that she had been subject to eczema all over the body; and that her father's father and brother had been "asthmatics" until they reached maturity. The very same night she improved much, and next day she was comparatively well; recovery was rapid.

The chief characteristics of this form of bronchitis may be thus enumerated: The rapidity with which the dyspnoea becomes extreme; the attendant fever, little or none; the whole attack seldom lasting more than two or three days; and the great tendency to recur. In the intervals most of the patients are free from pectoral symptoms, but in some cases emphysema occurs, and I have seen it well marked in a patient only 3 years old subject to this disease. Most of the children have suffered from eczema, and most of them come from families prone to asthma, bronchitis, and gout.

When we inquire into the previous history of patients who during youth have been liable to undoubted asthma, we find that in a certain number of them their disease dates from one or more attacks of this kind. And surely these attacks were asthma from the very first, although they were not deemed to be so at the time, but were called in Latham's language "acute bronchial inflammation putting on a peculiar form and affecting a peculiar course." The bronchitis does not cause the subsequent asthma, but it is asthma; the two diseases are identical in nature, and the asthma is bronchitis, although a peculiar form of bronchitis.

2. *Spasmodic Croup*.—This is an old phrase now seldom used, yet I do not know that we have a better. It is not a good phrase, because it implies a hypothesis which is probably untrue. The disease is characterised chiefly by acute dyspnoea; indeed, most of the cases described by Millar in the last century under the name of "acute asthma" were cases of spasmodic croup. Spasmodic croup is not laryngismus stridulus, for the dyspnoea of laryngismus is momentary, whereas that of spasmodic croup lasts for several hours and on and off for days. Spasmodic croup is not quite the same thing as laryngitis stridula—a disease which is characterised more by dysphonia than dyspnoea. Dr. Benjamin Rush, of Philadelphia was the first to apply the epithet "spasmodic"

to the disease—an indication probably that he as well as Millar recognised a resemblance to spasmodic asthma. I enter into these details because there still exists, as there always has existed, much confusion in the use of these names. Millar's description of the disease, published in 1769, is vitiated by the fact that he does not distinguish the different kinds of acute laryngeal disease which cause dyspnoea in children. Rush's description, published in 1789, is much more precise, perhaps because more concise. He says that "the cynanche trachealis spasmodica is known (1) by coming on suddenly, and that generally in the night; (2) by frequent and perfect intermissions of the symptoms for hours, and in some instances for days, without the least sensible discharge from the trachea; and (3) by yielding to anti-spasmodic remedies, particularly to the warm bath." Rush had examined the body of a fatal case and had found no marks of a membrane or even of mucus in the trachea; the lungs and trachea appeared to be in a sound state.

Many persons have believed in a close analogy between spasmodic croup and spasmodic asthma. Some have even gone so far as to say that "children who have been the subjects of croup or stridulous laryngitis in their earlier years are prone to suffer from true asthma in later life," but, judging from the facts of my own experience, I am disposed to think that this sequence is not common. Unfortunately the pathology of spasmodic croup is not well understood. I do not know that the glottis has ever been seen by means of a laryngoscope during the attack of dyspnoea. Could this be done, it would go a long way towards clearing up the darkness which hangs over the subject. Moreover, patients when attacked by spasmodic croup seldom die, and thus we are deprived of the information which minute *post-mortem* examination might afford. Nobody doubts that the disease implies an element of acute laryngitis, or of croup—for these two terms are almost synonymous, croup being the acute laryngitis of children. The evidence of laryngitis depends mainly upon these two facts: first, that the attack of dyspnoea is preceded for a few hours by hoarseness and croupy cough, and perhaps by other signs of catarrh of the respiratory mucous membrane; secondly, that the interval between the attacks is not free from the same symptoms. Admitting, then, that there is a fundamental ground of laryngitis, we must inquire in the next place into the cause of the dyspnoea, so sudden and so temporary, and here lies the difficulty. The epithet "spasmodic" begs the question by assuming that spasm of the glottic muscles is superadded to the laryngitis. But when we compare spasmodic croup with what is universally admitted to be a glottic spasm, that is to say, with laryngismus stridulus, the difference between the two diseases appears to be so great as to make us doubt whether the dyspnoea of croup can be spasmodic. In laryngismus the dyspnoea sets in very suddenly and lasts only a few seconds, for if it last longer the child will die. At any rate, if the croup be spasmodic we must assume that the spasm is very incomplete and that it lasts several hours—assumptions which seem improbable. It is improbable that the spasm should be always incomplete, and should never become a thorough laryngismus and thus kill the patient, whereas deaths from spasmodic croup are so uncommon that we may almost say that they do not occur. Moreover, the catarrhal swelling of the laryngeal mucous membrane would render any spasm much more likely to prove fatal, and we must never forget the small size of the glottis in children when we speak of their laryngeal diseases.

But if we give up the notion of spasm are there any other explanations which we can fall back upon? Two have been offered. One was put forward first, so far as I know, by Felix Niemeyer. He supposes that viscid mucus accumulates in the glottis while the child is asleep; that this mucus becomes dry, and so glues up the rima glottidis—an ingenious explanation, not wanting in plausibility, but which can be neither proved nor disproved. The chief argument against this hypothesis is found in the great resemblance between spasmodic croup and the peculiar form of acute bronchitis which we discussed just now. In both diseases, the same slight premonitory catarrh, the same sudden onset of alarming dyspnoea in the afternoon, the same speedy abatement of the dyspnoea, the same freedom from fatal results, and the same tendency to recur. Now, there is no reason for thinking that

the bronchitic dyspnoea is due to accumulated mucus, still less to dried mucus; and hence we may well doubt whether spasmodic croup is due to these conditions. But yet, until we can bring our senses to bear directly upon the glottis, until we can see what actually takes place, we may admit that there is possibly some truth in Niemeyer's hypothesis. How often are we tempted to exclaim, with the surgeon in the old novel, "A fig for reason, I laugh at reason; give me ocular demonstration."

The other explanation seems more probable. It supposes a sudden and temporary swelling of the mucous membrane of the larynx, such as sometimes occurs in the pituitary membrane during acute coryza when one nostril becomes suddenly blocked, and not by mucus, so far as can be made out, or at least no mucus can be expelled from the affected nostril. In the case of the nose this sudden swelling can be easily understood, for anatomists tell us that the veins of the mucous membrane over the turbinate bones constitute a cavernous structure, and that the membrane in this situation is erectile. No structure of this kind has been detected in the mucous membrane of the larynx, and hence the argument from analogy is weakened in a corresponding degree. In short, the opinion that spasmodic croup is due to sudden swelling of the mucous membrane depends mainly upon reasoning by way of exclusion, there being fewer objections to this pathology than to the hypotheses of spasm or of mucus obstruction.

Leaving these dry catarrhs of the respiratory tract, let us turn our attention to certain catarrhs of a character exactly opposite, being attended by abundant secretion of mucus, for these catarrhs also have a close affinity with asthma.

3. *Paroxysmal Coryza*.—In the first place we will consider paroxysmal coryza or "paroxysmal sneezing," as it is sometimes called. And let me set before you a vivid picture of the disease drawn by a sufferer, a married lady, aged 29 years. I copy the exact words of a letter she wrote: "Severe cold in the head every morning for two or three hours after waking; continual running of the nose, sneezing, watering of the eyes; pocket-handkerchiefs are of no use, I have to use towels. Frequent fits of violent sneezing, perhaps repeated a dozen or twenty times. Continual rising of phlegm into the mouth in the morning on waking; often an accumulated mass of phlegm has to be got rid of. Loss of taste and smell and impaired voice in singing. The above symptoms have been present for five years, though they have increased of late. In addition the following have occasioned trouble during the past nine months: a wheezing in the chest, difficulty in breathing, or in taking a deep breath. At times this prevents me from lying down at night; at least I go to sleep in the usual way, but am disturbed three or four hours after by a feeling of suffocation, and am obliged to spend rest of night sitting in a chair. This feeling sometimes comes on without any warning. Last week, when returning from a dinner party, I suddenly became oppressed in breathing, and had quite to struggle for breath for ten minutes or so. Violent exercise such as running, and I think swimming, also hearty laughing, produces much the same effect, namely, a wheezing and difficulty in breathing freely. Voice in singing much fallen off. Damp weather and cold winds seem to increase the tendency." Thus far the patient's own report, to which the following facts may be added: There was a tendency in her family to asthma, eczema, and gout; she herself never had eczema, but it had appeared in several of her relatives, and also in her only child, 15 months old.

The connection between paroxysmal coryza, asthma, and eczema is remarkable. A girl, 11 years old, had suffered from frequent attacks of asthma since the age of 2 years. Moreover, she had always been liable to fits of bad sneezing with running from the eyes. This sneezing was sometimes followed by asthma, sometimes not. When a baby she had eczema. In her father's family there was a tendency to eczema and gout, but not to asthma; in her mother's family there was probably a tendency to asthma. She had a younger brother who also was asthmatic, and who suffered from eczema in infancy. The girl, the brother, and the mother were all of a dark complexion. A second girl, sister of the patient, was of a fair complexion and had shown no tendency to asthma.

A boy, 2½ years old, became eczematous at 6 months of age.

The eczema cleared off at 12 months, and then he began to suffer from asthma. And mark that his attacks of asthma began with sneezing and running from the nose, and that an evidence of the chronic nature of the coryza was afforded by the ill-developed condition of the bridge of his nose. But there was no obstruction to his breathing in the nose itself.

At the risk of wearying you let me give you a brief outline of another case which shows the connection between this recurrent coryza and spasmodic croup as well as asthma. A lady, 30 years old, was subject to spasmodic croup when a child, and had been liable to bronchitis all her life. Since the birth of her first child three years ago she had become asthmatic—that is to say, she was continually rather short of breath, but more especially soon after arising in the morning or upon any sudden effort; her breathing would sometimes become very bad, she would turn blue, and the whole attack would last two or three hours; it had occasionally recurred three or four times in the day. Moreover, for two years she had been subject to attacks of sneezing almost daily, with copious distillation from both nostrils, as bad in winter as in summer. There were no objective signs of disease in the chest, nasal fossæ, or pharynx.

It is no part of my plan to discuss the relation which exists in some cases between these paroxysmal disorders and permanent disease of the nasal and post-nasal regions. But with regard to the actual attack of coryza let us observe that it is more than a mere defluxion of mucus. The mucus membrane of the nasal fossæ is swollen, especially the cavernous tissues over the turbinate bones, which swelling may be enough to make the opposite surfaces of the mucus membranes touch, and to the tickling sensation which ensues upon this contact much of the sneezing, and much, in all probability, of the distillation are due. The fact that in some cases asthma very quickly follows coryza suggests the notion of a rapid spreading of the catarrhal affection downwards along the respiratory tract; what in the upper part is a paroxysmal coryza becomes in the lungs paroxysmal dyspnoea, or, in other words, asthma.

4. *Paroxysmal Bronchial Flux.*—Closely allied as paroxysmal coryza and asthma undoubtedly are, there is an obvious unlikeness between them in one respect—namely, in the amount of mucous secretion. But there is a peculiar form of what Laënnec calls acute pituitous catarrh of the bronchial tubes, which more exactly resembles recurrent coryza in the abundance of the flux, its short duration, and its tendency to recur.

"A woman, aged 32, corpulent and of fair complexion, had suffered for some months from an obstinate cough, when one evening she was attacked by a complaint which very much resembled the paroxysm of spasmodic asthma, except that it was much more violent than first attacks of that disease usually are, and that its remission was attended with a very copious discharge of frothy serum from the bronchial tubes, which was thrown up by a slight, though almost continual, cough." On the next morning she was much better and she soon recovered. Some months afterwards she had another attack of the same kind, and about eighteen months after the first attack she had a third, much more violent. "She was suddenly seized with dyspnoea, attended with a convulsive kind of cough, which in less than five minutes from the time of attack increased so as to produce the appearance of suffocation. Her senses forsook her, her face became livid, her extremities were cold, and the action of the heart was so much diminished that no pulsation could be felt in the wrist, and but very little in the left side. She remained in this state nearly two hours, during which time a very large quantity of frothy serum tinged a little with blood was discharged without any visible effort by the mouth and nostrils. About the end of that time some very faint and involuntary efforts to cough came on, which gradually increased, and with every effort large quantities of the frothy serum were thrown off; perhaps the whole quantity might amount to 3 or 4 pints. About three hours after the beginning of the attack the difficulty of breathing became very sensibly diminished, and her senses were observed to return." In a few hours she had recovered so as to be much the same as she was before the seizure. After a somewhat longer time she had attained the condition of a patient in the best state of health. This woman was a patient of Mr. Thomas Baynton, of Bristol, and her case is narrated in a book published by the well-known Dr. Thomas

Beddoes, of the same city. You will have observed that the profuse expectoration is said to have been "serous," but this word is no doubt used in a loose sense, for Beddoes, who was a good chemist, expressly says that the fluid was ascertained by careful inquiry to be mucus, and not saliva, as he at first suspected. In my first lecture on bronchitis I spoke of acute pituitous catarrh, always a serious disease; and Baynton's case was an instance of this affection, but peculiar from its tendency to recur, and in this respect closely resembling paroxysmal coryza and asthma.

5. *Hay Fever.*—In this place I might refer to the fact that hay fever, which usually assumes the form of coryza and lachrymation, sometimes becomes an asthma. The paroxysms of hay asthma and of what we call ordinary spasmodic asthma differ in no particular. But this topic has been so fully discussed by our late distinguished President (Sir Andrew Clark), whose opinions are doubtless well known to you all, that I need not allude to them here. I will only repeat that the cause, whatever it be, of hay fever will produce ophthalmia, coryza, or asthma according to the portion of the mucous membrane which is affected.

And to sum up the argument in favour of the opinion that asthma is (in Willis's phrase) pneumonical, as distinguished from the convulsive form of the disease, the assumed bronchial obstruction being due, not to any sort of spasm, but to what he calls an afflux of humours upon the air passages. We have seen that there are five or six affections of the respiratory tract which are closely allied to asthma, and which may be said to constitute, together with asthma, a special class of diseases, characterised as follows: They set in suddenly, they speedily reach their highest point of severity, their duration is short, they are apt to recur, and in the intervals the patient is more or less free from signs of disease until the catarrh becomes chronic. The tendency to the disorders is hereditary and runs in families; many patients manifest a tendency to two or more of these complaints. If the force of these analogies be admitted, we may carry the argument farther and observe that all these diseases are catarrhal. Now, catarrh implies two conditions—namely, increased secretion and swelling of the membrane; but, as a matter of fact, neither of these conditions is constant. The defluxion or increased secretion may be absent, or at most may be very small, and this constitutes Laënnec's dry catarrh. The swelling is more constant, at least in acute catarrh; in paroxysmal coryza, which has so close an affinity with asthma, the swelling can be seen; moreover, both defluxion and swelling occur with great rapidity. The two concomitants of catarrh will explain the most important symptoms of all the disorders in question, including asthma.

If we consider the catarrhal tendency to be due to an inherent delicacy or vulnerability of the mucous membrane, a tendency usually inherited, we seem to be able to understand the reason of the fact that asthmatic persons during childhood are so often liable to eczema, which may be deemed to be indicative of a similar condition of the skin.

In the earlier part of the lecture we arrived at the conclusion that the doctrine of a spasmodic asthma is very imperfectly supported by facts, and now the argument has led us to believe that all asthma is merely a peculiar form of bronchitis, and that there is no need for any hypothesis of spasm in order to explain the phenomena of the disease. Should it be objected that bronchial spasm may possibly aggravate the effects of catarrh, we may appeal to the philosophic law of parsimony—*Entia non sunt multiplicanda præter necessitatem*. You know that this is not a new doctrine. Many years ago it was maintained very ably by Beau. Sir Andrew Clark arrived at the same conclusion by a different path—by noting how one and the same cause, hay fever, produces sometimes coryza, sometimes asthma, and sometimes both together. Which element of the catarrh, defluxion or swelling, obstructs the tubes is a question which naturally arises. The interesting observations of Lefèvre, published more than 60 years ago, upon the expectoration of peculiar mucous cylinders, which he compared to cooked vermicelli, towards the end of an asthmatic attack render it very probable that both secretion and swelling concur to obstruct the bronchia. But there remains another and much deeper question which concerns the asthmatic tendency—in

what does it consist? Why are some persons prone to asthmatic catarrh and others not? Why does the disposition run in families? What is the bond which in so many cases connects the several diatheses of asthma, eczema, and gout?

For the solution of such problems as these, and for the advancement of all medical learning, may a succession of men like Harvey and Willis never be wanting to our venerated College.

IS PETROLEUM EMULSION OF ANY NUTRITIVE VALUE?

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THERE can be no doubt that in recent years petroleum emulsion has crept into use as a substitute for cod-liver oil in the case of patients who are unable to take the latter. Now, petroleum belongs to the paraffin series—that is to say, to a set of substances which are characterised by the great opposition which they offer to chemical change. One would not, therefore, expect on *a priori* grounds that petroleum would be capable of assimilation in the body. In order to put the matter to practical test I recently carried out some experiments on the absorption of petroleum emulsion by man, the results of which entirely confirmed the suspicions which one had entertained on chemical grounds, and lead to the conviction that petroleum is of absolutely no use at all as a substitute for cod-liver oil. It is the object of this paper to describe these experiments and the conclusions to be drawn from them.

I employed a well-known and widely advertised emulsion of exceedingly pure petroleum. It was found that 30 c.cm. of it, when mixed with charcoal and evaporated to dryness, yielded on extraction with ether 7 grams of semi-fluid petroleum; 30 c.cm. represents about 3 dessertspoonfuls, which is the usual daily dose of the emulsion in question. I next proceeded to administer this dose to a healthy man with the object of ascertaining how much petroleum one could recover from the fæces. In the first experiment the subject was placed on a constant diet containing a moderate amount of fat. The fæces were collected daily, mixed with animal charcoal, and evaporated to dryness. The product was then extracted with ether in a Soxhlet's apparatus and the extract weighed. The results are contained in the following table:

First Experiment.

	Weight of Ether Extract.
Total fæces of three days without petroleum	7.1 grams.
Total fæces of three days on 30 c.cm. petroleum emulsion per day	32.7 „
Difference ...	25.6 „

That is to say, the administration of three dessertspoonfuls of petroleum emulsion daily had increased the ether extract of three days' fæces by 25.6 grams, although the diet was exactly the same as on the three previous days. The appearance of the extract was also quite different for the two periods. That of the first three days was solid and waxy, while that of the three petroleum days was semifluid in consistence.

In order to recover petroleum free from fat the ether extract was saponified by boiling with alcoholic potash solution. The residue was diluted with water, shaken up with ether in a separation funnel, the ether evaporated, and the residue weighed. The total 32.7 grams yielded on this treatment 21.6 grams of semifluid petroleum. Now 7 grams of petroleum had been administered on each day, so that 21 grams had been given, and 21.6 were recovered. The apparent excess found was probably due to the presence of traces of soaps.

In the second experiment no attempt was made to regulate the diet. Thirty cubic centimetres of the emulsion were administered on one day, and the fæces of the three succeeding days extracted with ether, and the extract saponified as above.

Second Experiment.

	Ether Extract.
First day (no petroleum)	3.5 grams.
Second day	4.1 „
Third day (30 c.cm. emulsion)	4.3 „
Fourth day (no petroleum)	7.6 „
Fifth day	6.9 „

On saponification, the extract of the fourth day yielded 4.5 grams of petroleum. The extracts of the third and fifth days yielded together 2.6 grams. The total amount of petroleum recovered was thus 7.1 grams, while the amount contained in the 30 c.cm. of emulsion given was 7 grams. The results of this experiment, therefore, entirely confirm those of the first.

From these experiments it may reasonably be concluded that petroleum is not absorbed in the human intestine and I consider, therefore, that it can in no wise be regarded as a food or a substitute for cod-liver oil.

Nor do I suppose that petroleum has any remote action, say, upon the lungs. It is conceivable that if crude petroleum were employed, some of the volatile substances contained in it might enter the blood and be excreted by the mucous membrane of the air passages, but the purer the petroleum used the less chance is there of any such occurrence.

Whether petroleum may have any value as a local application in intestinal diseases must be left undecided. One can imagine that there may be conditions of the intestinal mucous membrane in which the administration of petroleum might be of value in the same way as the application of vaseline to the skin, by forming a sort of pellicle on the intestinal surface.

In one sense, also, it may be regarded as an artificial intestinal mucus, and it might in that way have some value in certain forms of constipation. It is also worth considering whether it might not be a useful vehicle for the administration of intestinal antiseptics. Carbolic acid dissolves in it, and if the petroleum prevented the absorption of the acid it would also bring the latter into intimate mixture with the intestinal contents, and act as a sort of internal "carbolic vaseline"; but that part of the subject would require special investigation by experiment. My only object at present is to point out that petroleum, even when given in the form of emulsion, is not absorbed at all, and as a consequence can have no nutritive value.

CARCINOMA OF THE TONSIL.

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CASES of primary epithelioma of the tonsil are comparatively infrequent, but they are not so rare as was at one time supposed. The following case seems to me to have some points of interest.

Mrs. W., aged 60, consulted me in June, 1898. There was a well-defined swelling about the size of a pigeon's egg at the angle of the jaw, and extending up behind the ramus on the right side. On examining the pharynx, the right tonsil was found enlarged and its surface ulcerated; the uvula was pressed upwards and to the opposite side, and the right anterior pillar and adjoining surface of the soft palate were hyperæmic and swollen. There was no involvement of the tongue.

She gave the following history: In March a swelling at the angle of the jaw on the right side was noticed, and shortly afterwards she accidentally discovered that the right tonsil was inflamed and ulcerated. There was no difficulty in swallowing and no complaint of pain. She stated that though never a strong woman, she had suffered from no definite ailments. It may be noted that her mother died of "cancer of the breast," and a brother died of a "tumour on the right side of the chest" at 40 years of age.

As the swelling was regarded as malignant, a portion of the tonsil was removed and sent to the West of Scotland Clinical Research Laboratory, where the diagnosis of its malignancy was confirmed.

The report states: "Sections were cut vertical to the