Part First.

ORIGINAL COMMUNICATIONS.

ARTICLE I.—*On the Pathology of the Bronchio-Pulmonary Mucous Membrane.* By C. BLACK, M.D., Chesterfield, Bachelor of Medicine, and formerly Medical Scholar in Physiology and Comparative Anatomy in the University of London; Fellow of the Royal College of Surgeons of England, etc. etc.—(*Continued from page* 97.)

First Pathological Condition of Bronchitis involving the Submucous Tissue.

All the severe forms of simple bronchitis are referable to this variety, which, in many pathological points, resembles the epithelial variety of the disease, but which nevertheless differs from the latter in several very important particulars. These points of difference refer to,—

1. Respective duration.
2. Type.
3. The tissues involved.
4. Nature and composition of the discharge from the affected membrane.
5. Effects, both primary and secondary.

The differences thus specified may be briefly described. Epithelial bronchitis is always acute, whereas the above variety may be either acute or chronic. The former is always sthenic in type; the latter is either sthenic or asthenic. In epithelial bronchitis, the epithelium and basement membrane only are affected; in the above variety, the submucous tissue is likewise involved. In the former, the discharges from the affected membrane consist of epithelial scales or patches, and an excess of the natural secretion; in the latter, certain organic and inorganic bodies are added to the above.

The effects of epithelial bronchitis are epithelial desquamation or ulceration, and epithelial and basement hypertrophy. Of bronchitis involving the submucous tissue, ulceration, hypertrophy, bronchial abscess, and bronchial obstruction, as primary; and emphysema, bronchial dilatation, and collapse and atrophy of the pulmonary tissue, as secondary.

Now, in reference to simple acute bronchitis involving the submucous tissue, the first pathological condition of the bronchial mem-

brane is similar to the corresponding stage of epithelial bronchitis, except in these particulars; that the congestion is more intense, and that the phenomena arising out of it bear a corresponding aggravation. But this aggravation may either be accompanied by sympathetic fever, denoting power, or by an excessive action, coupled with vital prostration, showing a marked deficiency of constitutional power. With the former, or sthenic condition, there is a disposition in the bronchial membrane to prolong the duration of its first pathological stage; with the latter, or asthenic condition, the tendency is to pass rapidly into the second pathological stage. The explanation of these facts is readily found in the great amount of vital tonicity of the bronchial capillaries in the sthenic form of the disease, which, in the manner before described, successfully opposes exudation for a time; and of the small amount of vital tonicity in the asthenic form, which very quickly allows the pressure of the blood to overcome the contractility of the bronchial capillaries, and thus to permit an early exudation to take place. Under both the sthenic and asthenic forms of the disease, however, the pathological condition of the bronchial membrane is similar, not only in reference to each of these forms, but also to the first stage of epithelial bronchitis; therefore the phenomena immediately dependent on this condition must be essentially similar in all of them. But the expression of these phenomena will certainly be modified by the extent and intensity of the disease, and the amount of constitutional power which exists. Hence, in the variety of bronchitis now being considered, in which the bronchial engorgement is greater than in the epithelial variety, the sense of oppression and of straitened breathing is greater than in the latter disease. This aggravated feeling is partly due to the relative increase of blood in the bronchial capillaries, as compared with the epithelial variety, and partly to spasm of the muscular fibres of the bronchi, induced by the irritation resulting from the pressure of engorged capillaries, which two conditions diminish, in a proportionate ratio, the natural diameter of the tubes, and thus shut out a greater quantity of air than in the epithelial form of the disease.

For the same reason the sibilant rhonchi are more intensified, and partake more or less of a musical character; whilst the deep, sonorous rhonchi of the epithelial variety are, in this form of the disease, represented by rhonchi inclining to the sibilant character.

The percussion note is said not to undergo any alteration as a general rule; nevertheless, that it is occasionally rendered somewhat clearer than natural.[1] In the first pathological condition of the variety of bronchitis now under consideration, there is certainly never any increase of the percussion note; but, on the contrary, there is, if the disease is at all extensive, and more particularly if it affects the capillary bronchi, a slight, yet manifest, deadening of the percussion sound.

[1] *Vide* Walshe on Diseases of the Lungs and Heart, page 258.

The pathological condition of the membrane, the mechanism of respiration, and the relative effects of oxygen and carbonic acid gas on the contractility of the bronchial fibre, confirm the truth of the above statement, but in what way the following explanation will show.

The bronchial membrane is deeply congested, owing to which the natural diameter of the bronchi is diminished, the sentient nerves irritated, and a reflex motor impulse produced in the bronchial fibre, which, by its contraction, still further narrows the caliber of the tubes. The result of this is a partial admission only of the normal quantity of air into the pulmonary cells. The desire for more air, or rather the sensation of want of air, is excited in proportion to the deficiency, and the mechanism of respiration is taxed to overcome the obstacle which opposes the introduction of the usual quantity of air into the lungs. Hence arises a correspondent action of the supplementary aids of respiration; but notwithstanding the increased muscular effort thus excited, but little more air is admitted into the pulmonary cells, the walls of which are consequently not so tense as in the healthy inflation of the lungs. With a deficiency in the gross amount of air in the lungs, there must of necessity be a corresponding flaccidity of the cell-walls, and, as a consequence of the latter condition, a proportionate deadening of the percussion note. But it may be said, that the proper distension of the cells is maintained by the retention and rarefaction of the included air, owing to the impediment which is offered to expiration by the narrowing of the bronchial tubes. Such impediment does not in reality exist beyond the degree of narrowing produced by the mere turgescence of the bronchial membrane; because the carbonic acid gas, with which the expired air is surcharged, quickly destroys, for a time at least, the contractility of the bronchial fibre, thereby relaxing spasm, and favouring the egress of air from the lungs. But, admitting that carbonic acid gas exerts no such influence over the contractility of the bronchi, that consequently the diameter of these tubes is the same in expiration as in inspiration, and that the natural expiratory force is, under the above circumstances, insufficient to expel a proper quantity of air from the pulmonary cells, it is evident that the distressing sensation of the want of breath thus occasioned, would excite a forced expiratory act, which being one-third more powerful than the extreme of forced inspiration,[1] would more than counterbalance the latter effort, and thus cause the greatest amount of vesicular distension to depend, not on rarefaction of included air, but upon the inspiratory act. Again, on the other hand, oxygen excites the contractility of the bronchial fibre, and thus the impediment offered by spasm is added to that occasioned by bronchial turgescence, during the inspiratory movement. It is hence certain, that the impediments to inspiration are greater than those to expiration; that consequently

[1] See Reid's article on Respiration, Cyclop. of Anat. and Physiol., Part 32.

there is no inordinate retention of air in the ultimate cells of the lungs, and that no unusual amount of rarefaction takes place. With these conditions for free expiration, relatively considered, and with a deficient supply of air during inspiration, there must arise a defective inflation of the lungs, which must and does produce a corresponding diminution of the healthy percussion sound of the chest. It may, therefore, be truly asserted,—

That the pathological condition of the bronchial membrane, in the first stage of inflammation, has an invariable tendency to diminish the healthy percussion note of the chest.

But the amount of this diminution, and consequently its appreciability or otherwise, will depend on the extent and type of the disease, and on the particular bronchi affected.

In all cases in which the capillary bronchi are the chief seat of disease, the percussion note is lower than in bronchitis affecting the larger tubes; and, in like manner, the asthenic exceeds the sthenic type in this tendency to interfere with the natural resonance of the chest.

The indications of treatment deduced from the pathological details above given, are in the main similar to those of the first stage of the epithelial variety of the disease. Here, however, a full bleeding will, in the sthenic form of the disease, produce a decided effect in relieving the engorged condition of the bronchial membrane. But this condition will only be temporarily relieved, unless *cold air* be constantly applied to the affected membrane, so as to maintain the vital tonicity of the bronchial capillaries, and thereby to prevent their re-engorgement with blood, which would otherwise inevitably take place on re-action following the temporary depression produced by the loss of blood. Bleeding carried to the extent of producing a decided impression on the system, by lowering the heart's action, diminishes the supply of blood to the bronchial membrane, the vessels of which, by virtue of their inherent contractile power, force forward their contents into the corresponding capillary veins, and thus inordinate congestion is at once relieved. But the same loss of blood which thus lowers the contractile power of the heart, exerts a similar effect on the contractility of the capillaries, and thus the power of both is temporarily weakened in a corresponding ratio. In the bronchial capillaries, too, previous inordinate distension has contributed to exhaust the vital tonicity of the vessels, from which it is evident that, on the full resumption of the heart's action, their yet remaining tonicity would be unable to withstand the pressure of the blood on their walls, which would consequently yield to the distending force, and thus the previous condition of congestion would again take place. Hence, then, the absolute necessity of exciting and maintaining, by direct application of cold air to the affected vessels themselves, the vital tonicity of the bronchial capillaries, whilst the action of the heart is reduced to the lowest degree compatible with the safety and well-being of the patient. Hence, too, in the latter

point of view, the value of antimony, ipecacuanha, digitalis, and of other depressants during this stage. With a different object, yet tending to the same general result, purgatives, diaphoretics, and diuretics are indicated, amongst which mercury, salines, jalap, scammony, antimony, Dover's powder, Jacob's powder, acetate of ammonia, the warm bath, and the spirit of nitric æther, may be specified. In the use of these different remedies, it is necessary so to administer them, that their action shall be manifested in one and the same direction,—that is, that when a purgative effect is intended, all the remedies then employed shall have that tendency; and otherwise, as the necessity of the case seems to dictate.

In the treatment of the first pathological condition of asthenic bronchitis, the depressant, evacuant, and other remedies of the sthenic variety, cannot be employed with any degree of success. The vital tonicity of the bronchial capillaries is, in a great measure, already exhausted, and therefore unable to respond immediately to the direct application of any agent calculated to excite and maintain it. Cold air, therefore, could not produce that amount of contractility in the bronchial capillaries which would enable them, after their congestion had been relieved by bleeding, evacuants, and other remedies, to withstand the force of the current of blood propelled into them, and to prevent their again being inordinately distended. Hence it does not, in this variety of bronchitis, exert that immediate and purposive effect which I have repeatedly observed it produce in the sthenic type of the disease. As such is the case, and as bleeding, evacuants, and depressing remedies generally, would only further lower the vital tonicity of the capillary walls, and of the system at large, their use cannot be indicated in the treatment of this type of bronchitis. There are, however, different shades or degrees of debility accompanying and characterising it, in some of which a partial adoption of the remedies proper for the sthenic variety may be beneficially employed. But where such is the case, where local depletion and moderate purgation are ventured upon, great care should be taken to allow a corresponding reduction of temperature in the air breathed, and to maintain, at the same time, considerable warmth of the skin. Of all the means employed, the temperature of the air breathed, and of the skin, should be the last to receive but common attention.

In cases not admitting even a partial adoption of the sthenic remedies, the indications are,—

1. To relieve the congested condition of the bronchial membrane, by derivation of blood to the skin, and occasionally to other organs, for the purpose of increasing their function.

2. To maintain the due aëration of the blood, and to give to the latter sufficient nutritive materials to support the general system, and to restore its deficient tonicity.

3. To gradually excite the vital tonicity of the capillary walls, by the constant inhalation of a cool air.

To fulfil the first of these indications, dry cupping, mustard poultices, or a terebinthinate epithem, should be applied to the chest. The extent of surface irritated should, however, be great; otherwise, the derivation of blood thereby produced would not be sufficient to relieve the pathological condition of the affected membrane. The maintenance of considerable warmth of the skin generally has already been noticed as necessary to this end. When purgatives are necessary, they should consist of those which are considered warm and somewhat stimulating, in order that the power of the system may be maintained, and the biliary and intestinal secretions excited, whilst the volume of blood is but scantily reduced. The temporary erythema thus produced in the intestinal mucous membrane, and the corresponding excitement of the kidneys under the action of diuretics, indicate the negative effect which they may be made to exert on the bronchial membrane in disease of the latter.

In carrying out the second of the above indications, a sufficient quantity of oxygen must, if possible, be admitted into the lungs, to effect the due aëration of the blood, and thereby to render it sufficiently stimulating to the different organs whose function is, to eliminate effete and morbid elements from the system, and thus to preserve or restore the integrity of the latter. But another important point indicated under this head is, to support the general system by proper food, in order that the inordinate debility which exists may be overcome, and the general power and tonicity restored.

With these means of treatment we must couple the third indication, and gradually excite the vital tonicity of the capillary walls, by the breathing of a cool air, which will now restore the proper diameter of the vessels, and thereby destroy the pathological condition of disease.

Second Pathological Condition of Bronchitis involving the Submucous Tissue.

When the bronchial membrane has assumed the second pathological condition, the particular and general phenomena arising out of it are essentially similar to those which characterise the corresponding condition of the membrane in the epithelial variety of the disease. These, therefore, do not require to be again noticed in this place; but they permit me at once to consider the pathological phenomena which are peculiar to this, as contrasted with the epithelial, variety of bronchitis.

In the latter, I have shown, from the absence of inflammatory exudation in the sputa, that there is sufficient presumptive evidence that the submucous tissue is not the seat of exudation, and that the disease consists in an excessive mucous cell-growth, coupled with epithelial desquamation; and that it is, therefore, confined to the bronchial epithelium and basement membrane. In the former variety of bronchitis, not only are these last-mentioned structures

affected, but there is invariably exudation into the submucous tissue, as an essential part of its pathological condition. This exudation takes place contemporaneously with that into the basement membrane, and in both instances it consists of the same elements of the blood,—namely, water, fibrine, albumen, and various salts. The ultimate destination, however, of the two exudations is not exactly the same, they being disposed of in the following manner.

The exudation into the submucous tissue coagulates by virtue of the fibrine which it contains; whilst the watery portion, holding in solution the different salts, is, in the generality of cases, re-absorbed into the vessels. The coagulated portion of the exudation now forms a nutritive matrix similar to the basement membrane in the healthy condition and action of that structure; and in it germinal centres arise from molecular aggregation of the fibrine, which possesses an inherent capability of being developed into cells. The necessary conditions for germination and growth being present in the heat and moisture of the part, these germinal centres begin to assimilate to themselves the intervening substance, and thereby to assume the shape and endowments of cells. As the disposition of the fibrine, during the process of coagulation of the exudation plasma, is to accumulate more or less in masses, and as each particle or molecule of such fibrine is endowed with the same inherent vitality, each of these masses must of necessity contain many germinal centres, all of which may assume an active development, and in this way produce masses of cells, as represented in the adjoining figure.

Fig. 8.

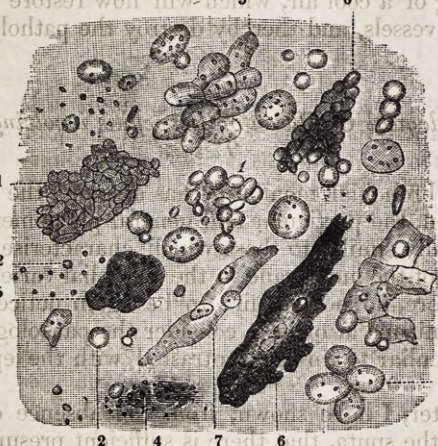

Fig. 8.—1. Exudation masses. 2 Exudation cells and molecules of fibrine. 3. Epithelial patch. 4. Superficial layer of basement membrane. 5. Entire thickness of basement membrane. 6. Mass of coaglated exudation, showing traces of cell development. 7. Bronchial cast.

Note.—The exudation-cells here figured are not of full growth; neither are many of them represented so round as they are observed in sthenic exudation.

Such structures are *invariably* present in the exudation during its early existence. They are, therefore, diagnostic of the existence of inflammatory exudation, and afford evidence, to a certain extent, of the degree of vitality which exists in the system, of which I shall hereafter speak. As these masses of coherent cells continue to grow, they assimilate and apply to themselves the nutritive matrix which binds them together; and after having thus exhausted it, they fall apart, and become distinct and isolated cells. Very many of such cells, however, do not originate in mass, but have, from the first, an isolated existence in separate and distinct molecules of fibrine. (Fig. 8, 2.)

During their growth, that portion of the nutritive matrix which cannot enter into the composition of the cell-wall passes, in a state of solution, into the cavity of the cells, which are thus kept in a state of moderate distension. When growth has reached its utmost limit, the endosmotic property of each cell permits more fluid to permeate it, and ultimately to distend it to such an extent, that the molecular attraction of its cell-wall is overcome, and disruption of the cell takes place. But this disruption does not consist in a mere rent, but rather of a molecular disintegration of the cell-wall, owing to the inordinate distension of the latter at every point of its surface, after it has ceased to be a living structure. In this way the exudation plasma undergoes a process of cell development and of cell decay in its removal from the system.

But sometimes other structures than these, to which the term pus-cell has been given, form a part of the exudation. They occur when the quantity of exudation is great, and the inflammatory action continues severe; or when a scanty exudation takes place, and continues to be influenced by a slow, persistent inflammation. They originate, like the exudation cells, in the fibrinous portion of the exuded plasma, and are in fact no other than such cells modified by the degree and persistence of the inflammation and the quantity of nutriment which is offered to them. The physical differences which they exhibit, consist in a slight difference of colour, to be afterwards explained, and in the presence of a number of granules, which are scattered over the inner surface of their walls, and which have the same chemical composition as the walls themselves. These cells generally measure from $\frac{1}{2100}$th to $\frac{1}{2600}$th of an inch in their diameter, contain from three to nine granules or nuclei, are rendered more transparent by the action of acetic acid, and are entirely destroyed by prolonged contact with liquor potassæ. Thus, in their chemical re-action, they manifest the same disposition as fibrine and albumen subjected to the same re-agents. Hence the identity of composition with these substances, in the distribution, as it were, of which the fibrine represents the cell-walls and nuclei, and the albumen the liquid contents. The conditions which determine their formation, may be thus explained. A severe inflammation affects the bronchial membrane, exudation takes place into the submucous tissue, but still the capillary engorge-

ment is maintained in all its intensity. None of the fluid exudation is therefore re-absorbed, neither is the temperature of the part reduced. The necessary stimuli of growth abundantly exist, the result of which is, that the inherent vitality of the exuded fibrine germinates, and rapidly develops cells. Owing, however, to the persistence of capillary engorgement, the nutritive elements offered to the cells are in excess of the demand which is necessary for the complete development of their walls. The overplus of fibrine, still in a state of solution, consequently passes by endosmosis into the cells, in which, from the force of homogeneous attraction between it and the cell-walls, it is precipitated in minute granules or nuclei upon the inner surface of the latter, thus constituting the plastic-corpuscles of Bennett. Such cells have now reached the utmost limit of development of which they, as isolated structures, are capable; and in this condition they either admit of being assimilated to the surrounding tissues, under the influence of the vital force which pervades the living organism, or they undergo a process of partial decay, during which they assume the particular character of pus-cells.

Now, this conversion into pus-cells is due to the action of oxygen on the structures of the plastic corpuscles, by which they undergo an *adipocerous* degeneration, similar to the well known effect produced on dead muscular tissue when exposed to moisture, and to a very partial contact with air. Hence the origin of the fat which invariably forms a chemical constituent of pus; and hence also the colour of the latter fluid as a consequence of the saponification of such fat by the alkalies present in the exudation. The oxygen necessary for this purpose is, in this particular instance, derived, at least in part, from the air taken into the lungs during respiration; but when maturation occurs at some distance from a mucous surface, or from the surface of the body, it (the oxygen) is supplied by the blood as it passes through the capillaries in the immediate neighbourhood of the affected part.

When a scanty exudation maturates, in consequence of a slow, persistent inflammation, the process is exactly similar to the one just described, except that in this instance the exudation first coagulates before cells are formed from it; whereas in copious exudation, accompanied by a continuance of capillary engorgement, cell structures, which ultimately assume the pus character, at once begin to form, without previous coagulation of the fibrinous portion of the exuded plasma. When, by exudation, the excessive engorgement of the capillaries is effectually relieved and does not return, the watery part of the exudation is re-absorbed into the vessels, whilst its fibrinous portion coagulates. This re-absorption of the fluid, and coagulation of the fibrinous, portion of the exudation, limit, to a certain extent, the conditions of germination and growth; but still, by the persistence of the capillary engorgement in a minor degree, sufficient fluid, impregnated with an excess of nutritive elements, is

supplied to the coagulated plasma to effect its gradual development into pus-cells.

It is, then, on the persistence of capillary engorgement, in a greater or less degree, after exudation has occurred, that the formation of pus-cells depends; but the actual conversion of plastic into pus-cells is, as I have before said, due to the destructive action of oxygen on the former bodies.

In the application of the above facts to the exudation of asthenic bronchitis, we should naturally expect that, as the exudation in such cases is always copious, and as capillary engorgement subsequently exists in its original intensity, thereby preventing the re-absorption of the more fluid part, and retarding or preventing complete coagulation of the exuded plasma, there would be a strong disposition in the latter to develop pus-cells. Such, indeed, we find, from actual observation, to be the case; in proof of which I may add, that in every instance of well-marked asthenic bronchitis which I have had the opportunity of examining after death, pus-cells formed a part of the exudation into the submucous tissue. Their formation in such cases depends, not only on the conditions before named,—*i. e.*, persistence of capillary engorgement after exudation has already occurred, and the action of oxygen on the plastic corpuscles, which constitute the ultimate limit of organisation of which such exudation is capable,—but also, in part, on the deficient vitality of the exuded plasma, which neither permits plastic corpuscles of full growth to arise, nor enables them, when they have attained a diminutive growth, to resist the destructive action of oxygen, and consequently their conversion into pus-cells so effectually as similar structures in a healthy exudation. Hence, therefore, the inherent disposition of imperfectly vitalised exudation to assume the distinctive character of pus-cells. Such structures generally measure from $\frac{1}{2500}$th to $\frac{1}{4000}$th of an inch in their diameter; are not so uniformly round as healthy cells, but present more or less of an irregular or angular figure; and are mingled with numerous flakes and masses of undeveloped fibrine.

Fig. 9.

Fig. 9.—Pus-cells from imperfectly vitalised exudation.

Whenever, therefore, the characteristic structures of pus present the physical appearance just described, we acquire indisputable evidence that the fibrine, out of which they spring, was imperfectly vitalised, and that consequently the constitutional powers are defi-

cient—the deficiency being in proportion to the dimensional and figurative aberration of the cells from the standard of healthy pus.

As to the proofs that these so-called pus-cells are nothing else than plastic corpuscles in a state of adipocerous degeneration, and that the latter bodies are but the exudation-corpuscles in an advanced stage of growth, it may be observed, that the characteristic cell of inflammatory exudation, in its very first stage of development, is the exudation-corpuscle only; that subsequently, under a persistence of the inflammation, the plastic-corpuscle appears at the expense of a comparatively numerical diminution of the exudation-corpuscle; and that the plastic-corpuscle is superseded by the pus-cell, with the all but total disappearance of the exudation-corpuscle. This conversion of the one cell into the other may be watched in the fluid exudation of an abscess during its different stages of development. Again, the walls of all, and also the nuclei of both the plastic and pus cells, are similarly affected by acetic acid and liquor potassæ. Thus, by acetic acid, they are rendered more transparent; whilst by prolonged contact with liquor potassæ they are destroyed. These results quite accord with the previously described effect of such re-agents on fibrine; and they not only serve to show the chemical composition of the walls and nuclei of such cells, but also their common origin, and the ready convertibility of one cell into the other. Whatever difference is manifested between themselves, on the application of chemical re-agents, is due to the different nature of their contents, and not to the cell walls themselves. Thus the contents of the exudation-corpuscle consist of water, albumen, and the alkaline salts of the blood; of the plastic-corpuscle, the same, with fibrine in addition; of the pus-corpuscle, of all the last-mentioned substances, together with the addition of fat, the origin of which is due to the action of oxygen on the fibrine of the plastic-corpuscle, as before described. Hence the solution of the different effects of various re-agents on the above structures.

Having now traced the pathological changes which take place in the exudation poured out into the submucous tissue during the second stage of bronchitis, it is proper to consider, in the next place, the corresponding pathological condition of the epithelial surface of the bronchial membrane, and the modification produced in its secretion.

Contemporaneously with exudation into the submucous tissue, a similar exudation takes place into the basement membrane, which thus becomes surcharged with the elements of cell-growth, and which, by virtue of the excess of fluid present, furnishes a powerful condition for germination and development. The epithelium, which till now had been deprived of its due amount of nutritive fluid, in consequence of the engorged condition of the capillaries preventing the passage of even the necessary quantity, is now flooded, as it were, with such fluid, the result of which is a rapid development of mucus-corpuscles. Hence these bodies in excess appear in the first

portions of the sputa expelled; but with them we also find a great number of epithelial scales or patches, which, having been blighted during the total suspension of all natural exudation in the first pathological condition of the membrane, are now unable to assimilate the entire portion of basement membrane allotted to them, and are consequently cast off, together with such portions of membrane, in the process of decay and repair. There are, therefore, one portion of the affected membrane denuded of its epithelium, and another portion of it with epithelium undergoing a rapid development into mucus-cells. Hence a difference in the vascular shading of the affected membrane, which is even detectable by the unaided sight, but which is rendered very apparent by the microscope. Amongst the epithelial scales and patches thus shed, there are others, which consist of the most superficial layer of the basement membrane enclosing nuclei, and sometimes of the whole thickness of that structure itself.

They measure, in general, from $\frac{1}{800}$th to $\frac{1}{1700}$th of an inch in their longest diameter, and are distinguished from each other by their relative degree of transparency; those patches of basement membrane which consist of its superficial layer only being much lighter and more transparent than those composed of its whole thickness. (Fig. 8, 4.)

Those portions of the membrane which have lost their epithelium in patches, and also those parts of it in which the basement membrane is almost or entirely cast off, afford sufficient outlets for the fluid exudation beneath, which now transudes through the basement membrane, fills the vacuities occasioned by its superficial or entire destruction, coagulates upon the denuded surfaces left by the shedding of the epithelium, and thus acquires a *locus standi*, and the necessary conditions for its germination and growth. Cell-growth, therefore, immediately commences, and results in the partial development of masses of exudation-corpuscles, which, however, are frequently detached by the effort of coughing, and consequently do not attain mature development. (Fig. 8, 1.)

These structures supersede the epithelial and basement patches, which disappear from the sputa shortly after expectoration has been established. If they are not detached by cough, or any other accidental circumstance at this stage of growth, they reach their full development as exudation-corpuscles; after which, if the elements of nutrition continue to be supplied to them, they (the elements) are precipitated as granules on the inner surface of their walls, and thus the modified exudation-corpuscles become plastic-cells. They have now reached their utmost limit of growth as living structures; and, in obedience to the universal law of change which presides over every living structure, they must either enter into structural relation with the containing tissue, or perish, become subject to chemical laws which govern inanimate matter, and be cast off as dead and waste material. In the last-mentioned condition they

assume, in the way before described, the character of pus cells, and as such they now appear in the sputa.

Fig. 10.

Fig. 10—1. Exudation-corpuscle of full growth.
2. Exudation mass, showing the development of one of its cells into a plastic-corpuscle.
3. Plastic-corpuscle of full growth.
4. Pus-corpuscle of full growth.

It has been observed, that before either exudation, plastic, or pus cell appears in the sputa, the latter have contained an unusual quantity of epithelial and basement patches, which leave the membrane denuded at certain points. If, in this condition, the membrane be carefully dissected away and examined by the microscope, it will be found that these denuded parts are covered by coagulated exudation, whilst the surrounding portions of the epithelium are quite free. It is, therefore, evident that these points are the only outlet for the exudation, and that epithelial desquamation is an essentially preparatory step to the formation of either exudation, plastic, or pus, cell, on a mucous surface. Hence the doctrine, that pus can be formed on the free surfaces of mucous membranes, *without there being any breach of structure*, is opposed to microscopic observation, which teaches—

That every case of pus from mucous surfaces is invariably preceded by epithelial desquamation, and not unfrequently by ulceration of the basement membrane itself.

In addition to the structures already described as products of inflammation appearing on the free surface of the bronchial membrane, other bodies, which present neither definite figure nor structure, are found in the discharged sputa. They are, in general, too opaque to transmit the light; but occasionally, here and there, are spots, through which light can pass, and from which we discover that such bodies contain cell-germs, as shown in Fig. 8, 6.

By prolonged contact with liquor potassæ, the whole of these bodies is destroyed. Their chemical composition, therefore, seems to indicate their fibro-albuminous nature; whilst the explanation of their origin is, that after epithelial desquamation has taken place, exudation occurs upon the denuded surface of the basement membrane, that it coagulates, and that it is subsequently expelled by cough before it has had time to undergo cell-development. Occasionally, in bronchitis affecting tubes from the third gradation in size downwards, the exudation poured out is so copious that, on coagulating, it forms a perfect cast of the bronchus, in the same way as renal

casts are formed in the uriniferous tubes of the kidney.[1] I have, however, observed, that such bronchial casts are more frequent in subacute and semichronic, than in acute attacks of bronchitis.

They often occupy their position for some time, owing to their glutinous nature, to the inability of muscular effort to dislodge them by cough, and, in some instances, to a probably too deficient sensibility in the affected membrane to take cognisance of their presence. They resemble dirty-white or yellowish-white gelatinous, worm-like bands, which measure from $\frac{1}{200}$th to $\frac{1}{2000}$th of an inch in their longest diameter, and which include granules, exudation and plastic corpuscles, together with an occasional admixture of pus-cells. (Fig. 8, 7.)

By so obstructing the bronchi that the air cannot enter the pulmonary cells to which the former lead, they give rise to pulmonary collapse, so ably pointed out by Dr William Gairdner, to whom pathological science is greatly indebted for several important advances connected with this and other subjects. Whilst, therefore, I refer all those who are interested in the subject of pulmonary collapse to his published work, "On the Pathological Anatomy of Bronchitis," I have pleasure in confirming the majority of his positions, by reference, not only to the human subject, but also to the inferior animals, and particularly to the ruminantia, rodentia, and digitigrade carnivora. During the present year (1852), I have examined the diseased lungs of eighteen animals, belonging to the above-mentioned classes, in fourteen of which I found certain bronchi obstructed with inflammatory exudation, together with collapse of the corresponding portions of lung. In eleven of these cases the collapsed portions of lung corresponded to the posterior and inferior lobes of the human lung, and were associated with different degrees of emphysema in the unobstructed portions of pulmonary tissue. In most of these cases the tissue of the collapsed lung was of a deep lake-red

[1] I may here remark, in reference to uriniferous casts in acute and chronic desquamative nephritis, that, according to my observations and investigations, hereafter to be published, the conditions of their formation are exactly similar to those which determine the formation of bronchial casts,—namely, 1. Vascular engorgement of the capillaries surrounding the uriniferous tubes, arresting for a time the natural transudation of fluid into the basement membrane, with which they lie in contact; 2. Exhaustion of the vital tonicity of these vessels, exudation as a consequence, and desquamation of those portions of epithelium which have perished during the stage of congestion, and the consequent suspension of all natural transudation; 3. The escape from the denuded surfaces of the basement membrane of an exudation, which, when copious, fills the corresponding uriniferous tubes, coagulates, and thus forms a cast, but which, when scanty, remains for some time attached to the basement membrane, on which it coagulates, germinates, and undergoes the same process of cell-development as characterises the products of bronchial inflammation. I therefore agree with Dr Christison, that the presence of uriniferous casts in the urine is pathognomonic of renal inflammation; whilst, with respect to their origin, I am compelled to differ from Dr Johnson, who refers them to transudation of fibrine through the walls of the congested Malpighian capillaries.

colour; and in all it manifested the firmness and consistence of liver. When incised, the cut surface presented a smooth, non-granular appearance, thus showing that the consolidation was not owing to inflammatory deposit. A fine horizontal section presented semi-dark linear shadings, from the falling together of opposite cell walls, whilst the fibrous tissue of the cells had evidently undergone a degree of atrophy, and the capillaries a certain amount of thickening, probably as the result of an unusual degree of contraction of their walls, induced by the pressure of surrounding collapsed pulmonary tissue.

The mean of sixteen analyses of the sputa of acute sthenic bronchitis gave, of—

	In 100 parts.
Water, ...	96·75
Organic matter, ...	2·15
Alkaline sulphates, „ phosphates, Chlorides of sodium and potassium, Sulphate and phosphate of lime, ...	1·10
	100·00

On comparing the above analysis with that of the healthy secretion of the bronchial membrane, it will be found that there is a considerable increase of solids in the sputa of sthenic bronchitis. In the asthenic type of the disease the difference is not so great, the average increase of solid constituents being, according to the mean of six analyses, from 1 to 1½ grains in 100.

To determine how far the organic matter agreed with fibrine and albumen under the action of acetic acid and liquor potassæ, as before detailed, a quantity of mucus was evaporated to dryness over a sand-bath, after which the solid residue was triturated in a mortar, and then digested in warm distilled water for some time. The whole was afterwards poured on a filter, and the fluid portion, holding in solution the soluble salts, passed through. The residue left on the filter was collected, dried, and divided into three portions. One portion was digested in cold acetic acid, a second in liquor potassæ, whilst a third was boiled with the acid. The first portion increased somewhat in bulk, became slightly more transparent, but was not in the least degree dissolved at the end of seventy-two hours; the second dissolved at first rather rapidly, afterwards more slowly, and imparted to the liquor potassæ the well-marked tinge of a weak solution of burnt sugar; whilst the third portion, in by far its greater part, underwent solution in forty minutes.

The result of these experiments seems to leave no doubt as to the chemical composition of the organic matter, which in acute sthenic bronchitis bears the proportion of upwards of 3 to 1 as compared with the organic matter of healthy mucus; whilst the salts of the latter are as 1 to 10 of the former. If, again, we compare the relative proportion of salts to organic matter in both healthy mucus

and the sputa of acute sthenic bronchitis, we find that in the former the salts are to the organic matter as 1 to 5·66; in the latter as 1·10 to 2·15. It is, therefore, evident that inflammation of the bronchial membrane produces a greater relative increase of salts than of organic matter in the sputa, the reason of which appears to be the following:—

After the coagulable portion of the exuded plasma has solidified on the denuded surfaces of the basement membrane, a continual supply of alkaline fluid from the blood is required to effect its resolution, before it can be assimilated in the growth and development of cells.

This supply of alkaline fluid regularly taking place for some time after all inflammatory exudation has ceased, must necessarily lead to a relative increase of salts, as compared with the organic matter of the sputa, which, according to the analyses before given, is actually the case.

If we take into consideration the absolute increase of organic matter and salts, as well as the greatly increased quantity of sputa, in acute sthenic bronchitis, we can appreciate the drain which is continually being made upon the blood by this cause alone; and if we further regard the waste produced by all the other secretions, and occasionally by the effects of medicines, as well as the negative result of almost total abstinence from food, we can readily understand how and why the bulk and weight of the body rapidly decrease in disease.

Looking at the pathological condition of the bronchial membrane in the second stage of bronchitis involving the submucous tissue, it may now be asked,—What are the indications of treatment which this condition affords? The vital tonicity of the capillaries is exhausted; exudation has occurred into the submucous tissue, as well as into and upon the surface of the basement membrane; and the free surface of the latter structure is in part denuded of its epithelium.

The condition of the membrane itself is very similar to that which accompanies the corresponding stage of the epithelial variety of the disease; therefore the treatment must be based upon the same principles. But there are those differences in the pathology of the two varieties of bronchitis already described, which must necessarily modify, to a certain extent, the treatment. In the more severe variety there is exudation into the submucous tissue, as well as upon the denuded epithelial surface of the basement membrane; in the epithelial variety neither of these conditions occurs. Again, bronchial obstruction, from inflammatory exudation into the tubes, is apt to occur in the more severe kinds of bronchitis; but in the epithelial variety such is never, or very rarely indeed, the case. These differences must necessarily modify the indications of treatment; but, in the main, they are the same as in epithelial bronchitis.

To procure the removal of the exudation from the submucous tissue, its tendency to cell-development must be favoured, and its subsequent disintegration procured. Heat and moisture—the natural stimuli to cell-growth—are already present in the affected part; but the liquefaction of the coagulated portion of the exudation, and the subsequent molecular disintegration of the resulting cells, will be greatly facilitated by the exhibition of the alkalies. Were it possible to exhibit these in large quantities, without injury to the red globules of the blood, and to the fibro-albuminous structures, by doing so the exudation might readily be disintegrated, and its absorption procured, without its undergoing the process of cell-development. For the removal of the exudation from the epithelial surface of the basement membrane, for the liquefaction of inspissated mucus, and for mucus generally, and also for the disintegration of bronchial casts, the alkalies are by far the most efficient. But in the case of inspissated mucus and of bronchial casts, so great may be the extent of bronchial obstruction and of pulmonary collapse produced by them, that the consequent insufficient aëration of the blood may require that the breathing power of the lungs shall be immediately improved. Sufficient time, therefore, cannot be given to the alkalies to effect the gradual disintegration of the causes of obstruction through the route of the circulation, in which case the use of emetics is indicated, for the purpose of effecting a mechanical dislodgement. But it must be borne in mind that some emetics, as antimony, are much more prostrating in their effects then others; and that the condition of the system induced by their exhibition would be accompanied by a corresponding diminution in the vital tonicity of the bronchial capillaries, which would again favour congestion and exudation, and thereby render the beneficial effects of such emetics very temporary indeed. Hence the importance of exhibiting emetics which produce the least depressing effects on the system, amongst which the sulphate of zinc, a warm solution of common salt, and mustard, deserve to be specified.

Sometimes, however, expectoration fails in consequence of sheer debility; and it is in these asthenic cases that expectorants, strictly so called, are indicated. All real expectorants (amongst which I do not include antimony, mercury, ipecacuanha, and the alkalies) exert more or less of a stimulating effect on the bronchial fibre and capillary circulation, which degree of stimulation is unnecessary and even injurious in sthenic bronchitis, or in cases in which sufficient power remains to dislodge the mucus by an ordinary degree of cough. If, by such an effort, the sputa are not dislodged, it is certain that the cause is owing to the tenacity of the sputa themselves, for which the appropriate remedy is not an expectorant, but a liquefacient, as potash, soda, or a mercurial preparation. But when manifest debility is present, and when expectoration fails as a consequence of such debility, expectorants are clearly indicated, and of

these senega, squill, ammoniacum, and the different balsams and oleo-resins are in vogue. The pathological condition of the membrane, however, indicates, in addition, the necessity for the exhibition of a generous diet, in order that the vital force of the system may be increased, the power of expectoration maintained, and a reparative process instituted and sustained by the affected membrane.

Other important indications, alike applicable to this and epithelial bronchitis, are expressed in the treatment of the second pathological stage of the latter variety, to which the reader is referred. Other, yet minor, indications will in most cases arise out of the urgency of individual symptoms, as cough, all of which should receive the judicious attention of the physician.

(To be continued.)

ARTICLE II.—*Report of an Attack of Colic, which occurred as an Epidemic, among the troops at Newera-Ellia, Ceylon, in the commencement of* 1852. Communicated to the Superintendent of the Army Medical Department, by Assistant-Surgeon ALEXANDER SMITH, M.D., Her Majesty's 37th Regiment.

SIR,—In reply to your letter of the 16th November 1852, I beg to forward, for your information, the following notice of an epidemic of colic, which prevailed among the troops at Newera-Ellia, in the island of Ceylon, between the 12th February and the 20th June 1852.

The period, during which I was in medical charge of the troops at the above station, extended only from the 5th May to the 31st July of the present year, but included the period in which the epidemic prevailed with greatest severity; and, previously to the 5th May, I had an opportunity, at Kandy, of witnessing several of the more severe cases sent from Newera-Ellia to the Head-Quarter Hospital. From the observations I was then able to make, as well as from subsequent examination of the Medical Records, I have every reason to believe that the epidemic maintained the same character throughout; so that, without impropriety, the following may be considered a description of it in its whole course.

Newera-Ellia is situated in the interior of the island, within about six and a half degrees of the equator, at an elevation of 6000 feet; and, from the latter circumstance, it has a mean annual temperature under 60°. It consists of a small native village, the barrack, and European residences—all widely scattered over a space of ground several miles in extent, and which may be described as an irregular table-land, intersected by water-courses. The