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* 202.)

Pulmonary Cellulitis.

BY the above term I propose to designate inflammation confined to the epithelium of the pulmonary cells,—a disease which is extremely prevalent amongst children, and often regarded as pneumonia, and which is not rare even in adults. It indeed bears the same relation to pneumonia as epithelial bronchitis does to the severe variety of that disease, inasmuch as the products of the inflammation, like those of epithelial bronchitis, appear on the free surface of the membrane; whilst, in pneumonia, intercellular exudation, which corresponds with the submucous exudation of severe bronchitis, occurs. Owing, however, to the anatomical arrangement of the pulmonary cells, and to the immense distribution of vessels on their walls, it is evident that epithelial inflammation in this situation must be more intense than in that of the bronchi; and that, consequently, exudation, instead of not exceeding the demands of an excessive cell-growth, is poured out in the same copious manner, and undergoes the same changes, as that on the free surface of the membrane, in the severe kinds of bronchitis.

There are in this affection, as in the varieties of bronchitis already detailed, two pathological conditions of the membrane, each of which is associated with particular phenomena, which it will be proper to notice under their respective heads. But before doing so, it may be observed, that cellulitis is more common in the lower than in the upper half of the lungs; in the posterior than in the anterior portions of the lungs; in infants and children than in adults; that it may occur suddenly and at once; or that it may supervene on either the epithelial or the more severe variety of bronchitis; that when it does so, it is owing to the extension of inflammatory action by continuity of membrane, and is associated with capillary bronchitis,

which has superseded, in a great measure, the bronchitis of the larger tubes; and that, when it occurs in the upper half of the lungs, it is generally dependent on tuberculous or other deposit in the pulmonary tissue.

First Pathological Condition of the Bronchio-Pulmonary Membrane in Cellulitis.

The first pathological condition of the bronchio-pulmonary membrane in cellulitis is, in every respect, similar to that of the corresponding stage of bronchitis, and consists of an extremely vascular engorgement, accompanied by total suspension of the accustomed secretion. When the capillary bronchi are likewise implicated, spasm of their muscular fibres is associated with the above-mentioned conditions of the membrane. The appearance of the latter is observed at this period to vary in colour from a deep reddish-brown to a bright crimson.

The degree of shading, however, is not equal throughout the affected pulmonary cells; the edges of their apertures, where the membrane is folded on itself, being of a more deeply reddish-brown or mahogany colour than their interior, which is of a brighter crimson hue. When the capillary bronchi are also affected, their lining membrane is similar in coloration to the interior of the cells, the depth of colour gradually diminishing until it is lost in the natural hue of the fourth or fifth gradation of the bronchi. The whole structure is turgid and puffy, the natural diameter of the pulmonary cells and minute bronchi is diminished, and the cilia of the latter tubes are erect, and are seen by the microscope to project at right angles from the surface of the epithelium on which they are set. This diminution in the capacity of the pulmonary cells and minute bronchi alters the breathing power of the lungs, which undergoes a corresponding deficiency, and hence the sensation of the want of breath is excited. Hence also increased action of the lungs, to compensate for the deficient inflation. Owing now to the dryness of the membrane, and consequently to the direct application of air, surcharged at times with floating particles of dust, to the sentient membrane, which is rendered doubly irritable by the pressure of its engorged capillaries on the terminal fibres of its nerves, cough is present. From the excess of blood in the affected part, a sense of weight, and sometimes of burning, is experienced by the patient. Owing, too, to the same cause, the percussion-note of the chest is reduced, and the conducting power of the lung increased.

This dulness on percussion is confined to the seat of disease, which, as a general rule, is the posterior and lower half of the chest; whilst on its confines the percussion-note is not unfrequently of elevated pitch. If one lung only suffer, the opposite lung may take on a compensatory action, and its function will consequently be increased. The breathing is now deep; the ribs on the sound side are unusually

elevated and everted during inspiration; the intercostal spaces are swelled out, and the number of respirations is less than when mere activity of function is the compensatory mode adopted by nature. Where the above mode of compensatory action prevails, the rhythmic movement of the two sides of the chest is unequal, that of the diseased side stopping short of the degree of elevation of the sound side, with which, however, it descends in rhythmic order from its own point of elevation. But when mere activity of function is the compensatory mode adopted, the rhythmic inequality of the two sides of the chest is not so strongly marked; the elevation and eversion of the ribs, on the sound side, exceed but very slightly, and sometimes not at all, those of the diseased side; but the number of respirations is always increased. The particular type of the disease determines the particular compensatory mode of action, since we invariably find that the former method is associated with the sthenic, and the latter with the asthenic, form of cellulitis.

Although the condition of the bronchio-pulmonary membrane is, at this stage, such that it produces a manifest and palpable deadening of the percussion-note, a diminution, from the first, of the vesicular murmur, when the capillary bronchi are implicated, and the reverse, at the very commencement, followed very quickly by a diminution of the vesicular murmur, when the cells alone are affected; yet it never proceeds, like pneumonia, to the extent of producing the characteristic physical signs of perfect consolidation of the pulmonary tissue. Such is the diameter of the pulmonary cells, and such the anatomical structure of their walls, that the mere engorgement of their vessels is of itself insufficient to produce complete pneumonic consolidation. The vesicular murmur is consequently always present; nevertheless it is invariably diminished in intensity after the tonicity of the pulmonary capillaries is, in a great measure, destroyed by the continued pressure of the blood upon their walls. Until such is the condition of the capillaries, the capacity of each cell is sufficiently great to allow a quantity of air to make considerable pressure on its walls during inspiration, and the contractility of its walls is sufficiently active to oppose such expansile distension; consequently these two forces, acting in opposition to each other, generate an increased vesicular murmur, which the increased conducting-power of the lung readily transmits to the walls of the chest, and to the ear of an observer. With, however, the increasing exhaustion of the vital tonicity of the capillaries, there is such a reduction in the capacity of each cell, owing to excessive vascular engorgement, that but comparatively little air is admitted into it; whilst, owing to the lateral pressure of the capillaries on the individual fibres of the cell-walls, and to the partial exhaustion of the contractility of such fibres by previous over-action, and probably now by defective nutrition, the anti-expansive power of the cell-walls is diminished, so that a concurrent diminution of the two generating causes of sound gives rise, as a necessary consequence, to a diminished vesicular murmur.

When the capillary bronchi are affected, the air, during inspiration, is in consequence unable to enter the pulmonary cells in quantity sufficient to excite the contractility of their walls by distension; therefore, as the capacity of each cell is diminished, as the generating causes of sound are but feebly opposed to each other, the vesicular murmur is from the first below the standard of health. But the signs which take their expression more particularly from the absolutely physical condition of the lung, rather than from this condition in conjunction with the vital forces of the lung, undergo a steady increase in proportion to the increasing density of the lung. Thus, as a general rule, the vocal resonance of the affected portion of lung augments in proportion to the increase of vascular engorgement; consequently the vocal resonance in cellulitis is always increased whether the vesicular murmur be so or not. For the same reason, the percussion-note is always deadened; whilst tubular breathing and bronchophony are always present in the affected part, in which, however, they are modified by the extent and degree of vascular engorgement, by the particular portion of lung, and also by the relation of the bronchial tubes to the surface thereof.

It is certain that, when this pathological condition of the bronchio-pulmonary membrane is at all extensive, not only are the functions of the affected organ deranged, but the whole system will, in consequence of the many intimate and distant sympathies which exist between its different parts, feel the loss of equilibrium thereby occasioned. Hence the effect produced on the vascular and nervous systems, and through them, on the different secretions, appetites, propensities, and feelings.

As to the indications of treatment, they are in general so similar to the corresponding pathological condition of the membrane in severe bronchitis, that reference only needs be made to the treatment of the latter.

Second Pathological Condition of the Bronchio-Pulmonary Membrane in Cellulitis.

The second pathological condition of the bronchio-pulmonary membrane in cellulitis is characterised, as in the different forms of bronchitis, by complete exhaustion of the vital tonicity of the affected capillaries, and by exudation. The membrane has now lost its dryness, and to some extent its puffiness and deep coloration. A bright pink or lake-red hue has superseded the deeply reddish-brown or mahogany tint of the previous stage. The interior of the pulmonary cells, their angles, and the terminal bronchi generally, when the latter are implicated, exhibit more or less of a uniform depth of colour.

The occurrence of exudation produces an alteration in the auscultatory signs of the chest, which, in reference to the resulting rhonchi, are moist in their character, as in the second stage of bronchitis.

Their mode of production, although the subject of much difference of opinion, is readily deducible from the pathological condition of the pulmonary cells. These (the cells) are more or less occupied by a fluid, their free surface is slightly irregular from the coagulation of exudation, whilst their walls are surrounded by vessels more or less engorged with blood. It is therefore evident that the air, on entering the pulmonary cells, has to encounter the resistance of this fluid and of the cell-walls, to which may be added whatever resistance the irregularity of the free surface of the cells can offer. The resistance offered by the fluid is overcome by the entering air insinuating itself between its particles, which envelop it in a thin film, constituting a minute bubble, which bubble, on reaching the surface of the fluid, encounters the resistance of the cell-walls, by which it is broken, and sound is emitted. The quantity of fluid in the air-cells, its viscidity, and the mode of breathing, influence the character of this sound, the differences in which are expressed by the terms crepitant and subcrepitant. If the quantity of fluid is sufficient to fill the cells at the extreme of expiration, if it is very viscid in its nature, and if the breathing is hurried rather than deep, the bubbles formed are small and numerous, the bursting of which keeps up a succession of fine sounds or cracklings, similar to that produced by slowly drawing the back of the closed hand over a shortly bearded chin. This is the crepitant rhonchus—a sound but seldom heard in cellulitis.

If, on the other hand, there is the same quantity of fluid present in the cells possessing the same viscidity; but if the breathing, instead of being hurried, is somewhat deep, the bubbles formed are larger and fewer than in the previous instance, and thus, by their bursting, a coarser sound than the crepitant is emitted, to which the term subcrepitant has been given. This is the sound most frequently present in the second pathological stage of cellulitis; but the conditions of its production are not always those above stated. They, on the contrary, rather consist in the presence of a quantity of moderately tenacious fluid, accompanied by slightly hurried and forcible breathing. These conditions of the fluid and of the breathing are, as a general rule, present in the second stage of cellulitis; therefore the subcrepitant rhonchus is the generally characteristic moist sound of this pathological stage of the membrane. If, however, with this condition of the fluid contents of the cells, the breathing, instead of being hurried, is either hurried and deep, or the latter only, larger bubbles than are necessary to constitute the subcrepitant rhonchus are formed in the pulmonary cells, in which, by their bursting, a still coarser sound than the latter is produced, to which the term submucous rhonchus is given. This sound is heard in the sinking stage of the disease, in which it is not only formed in the pulmonary cells, but also in the minute bronchi, owing to the accumulation of fluid in the latter tubes.

Clinical observation further shows, what pathological deduction

seems to confirm,—namely, that the quantity of fluid in the air-cells influences the duration of the particular rhonchus present. If the quantity of fluid is small, it, in consequence of the provision made by the contractility of the fibrous tissue, favoured occasionally by position, of the cells, for the outward tendency of fluids, accumulates at the apertures of the cells, owing to which the entering and emergent air only is enveloped in films of fluid, and thus the rhonchus is limited to the commencement of inspiration and the end of expiration. But the inspiratory rhonchus is certainly more intensified than the expiratory, owing to the inward current of air encountering greater opposition than the outward current, and having, as a consequence of such opposition, to overcome the resistance of the cell-walls, which thus furnish a condition for the production of sound. This sound is, in the above-mentioned condition of the cells as to fluid, composed partly of a moist and partly of a dry rhonchus in the following manner. The air, on entering the pulmonary cells, encounters the small quantity of fluid collected at their apertures, on passing through which a moist rhonchus is produced; but the cells not yet being distended to their full extent, the inspiratory air lifts their walls above the surface of the contained fluid, in doing which it encounters the passive resistance of such cell-walls free from fluid, and also whatever resistance the irregularities of their free surface, caused by inflammatory exudation, can offer, whence arises a dry, rough, and harsh murmur, as the extreme of inspiration, but as this only. On the contraction of the cell-walls in expiration, the only resistance offered to the emergent air is by the fluid situated at the apertures of the cells, and here, therefore, sound is produced, which, owing to the pathological condition, and to the situation of the parts, is of a crepitant or subcrepitant character, and limited to the last third of the expiratory movement. On a careful analysis, however, of the sounds heard during the movements of the pulmonary cells in such instances, it is frequently observed that, whilst the rhonchus, produced by the air entering the cells through the fluid collected at their apertures, is of a subcrepitant character, that heard as the air passes out of the cells, through the same fluid, is distinctly crepitant. This difference in the quality of the two rhonchi is owing to the difference in the force with which the air enters and emerges from the pulmonary cells. The entering column of air, moving with greater force than the emergent column, produces larger bubbles than the latter, and therefore gives rise to a coarser (subcrepitant) rhonchus.

If a greater quantity of fluid than that which has already been stated, occupies the cells, the rhonchus is heard during two-thirds of the duration of the respiratory movements; but if the whole cavity of the cells is engaged, the sound is limited to the extreme of inspiration and the commencement of expiration, at which periods the bubbles, rising to the surface of the contained fluid, come into contact with the cell-walls, by which their disruption is occasioned. It

is, however, seldom indeed that anything approaching to complete distension of the cells occurs in cellulitis; therefore the rhonchus heard has not the limited duration named; but, on the contrary, it is generally present during two-thirds of the inspiratory, and the latter half or third of the expiratory movement. The cause of this partial distension of the cells only, seems to be, in a great measure, owing to the particular nature of their contained fluid, which being less viscid and tenacious than the sputum of pneumonia, is more readily propelled into the minute bronchi, in which it furnishes a condition for the production of a submucous rhonchus. But, although complete distension of the cells with exudation never occurs in cellulitis to the extent of producing perfect consolidation of the pulmonary tissue, yet we find, on microscopic examination of the sputa, that perfect casts of individual cells are frequently present, and that such a condition has existed, although not in that degree which produces the physical signs of perfect consolidation.

As to the resonant property of the lungs, in this pathological condition of the bronchio-pulmonary membrane, we find, that the continued presence of an excess of fluid in the pulmonary cells and minute bronchi, still keeps the percussion-note below the standard of health; and, at the same time, so far muffles and chokes the conduction, to the surface of the chest, of the vibratile motions originating in the production or propagation of sound from the subjacent bronchi, that bronchophony and tubular breathing are but indistinctly heard. These sounds, however, together with a diminution in the vesicular murmur, a harshness in the quality of the latter during inspiration, particularly forced inspiration, and a degree of prolongation of the sound of expiration, exist for some time after the total cessation of the more manifest signs of disease; and this they do, because the basement structure of the mucous lining of the pulmonary cells is hypertrophied by inflammatory deposit, which causes the increased thickness of the cell-walls to encroach on the normal capacity of the cells, to impede their contractile power, to render the pulmonary tissue a better conductor of vibrations than it is in health, and to modify, as above stated, the static and dynamic properties of the chest. I have found that, in such instances, the hypertrophy of the basement membrane gives to the latter a diameter varying from $\frac{1}{10000}$th to $\frac{1}{16000}$th of an inch; that, under a steadily progressive tendency to a restoration of the normal condition of the structure, such hypertrophy requires weeks, and not unfrequently months, for its complete removal; and that it occasionally leaves a degree of permanent thickening of the cell-walls, which thickening, in estimating the breathing-power of the lungs, is appreciable in proportion to the extent of surface involved, to the consequent reduction of capacity in the pulmonary cells, and to the degree of compensatory power which is manifested by the uninjured portions of lung, in comparison with the deficient action of those portions, the breathing-power of which has been injured by disease.

With respect to the physical appearance of the sputa discharged in cellulitis, and to the pathological conditions on which they depend, it is found, that the very first portion discharged, presents to the naked eye the same colour and consistence as the first portion of sputum expelled in bronchitis; and that its microscopic appearance shows it to consist of the epithelium of the pulmonary cells, of well-developed mucus-corpuscles, and of a few points and masses of coagulated exudation, suspended in a fluid menstruum. (Fig. 7). The first pathological step, then, towards the establishment of a discharge from the affected membrane, consists, in cellulitis as in bronchitis, of the partial shedding of the epithelium, of rapid development of those portions of epithelium which retain their vitality into mucus-corpuscles, and of the coagulation, on the denuded portions of the basement membrane, of the fibrinous parts of the exudation, some of which are detached by the efforts in coughing, and consequently appear in the first sputa expelled. If the inflammation is confined to the pulmonary cells, the next quantity of sputum discharged is more consistent, viscid, and tenacious, presenting to the naked eye a yellowish or greenish-yellow colour. This kind of sputum has been described as a more frequent sign of pneumonia than even the rust-coloured expectoration; but, according to the pathological views here adopted, such is not the case. On the contrary, it, in conjunction with the physical signs before named, is characteristic of cellulitis; whilst pneumonia proper—*i.e.*, inflammation involving, by exudation, the whole substance of the cell-walls, and of the intercellular spaces and connecting tissue—is all but invariably accompanied by rust-coloured sputum during the first half of the expectorant stage. Blood, therefore, is an accidental constituent of the sputum of cellulitis; but as a general rule, a constant, and therefore a necessary, one of that of pneumonia.

Under the microscope, the former kind of sputum is seen to contain fewer epithelial patches and mucus-corpuscles than that first discharged. The epithelial rather now give place to basement patches, which are commingled with numerous exudation masses and corpuscles, and a few casts of individual pulmonary cells. (Fig. 11.)

The intensity of the inflammation is represented by the quantity of exudation masses and corpuscles present; whilst the vital energy of the system is determined by the shape, and, to a certain extent, by the degree of development which these corpuscles have attained. In sthenic cellulitis they are perfectly spheroidal in shape, and exhibit a development in size proportionate to the length of time which the matrix, in which they arise, has been attached to the denuded basement membrane. Hence they vary in size from $\frac{1}{1800}$th of an inch to a diameter but little larger than the nuclei from which they arise.

If capillary bronchitis is associated with cellulitis, there are, in addition to the above-mentioned structures, casts of the minute

bronchi, which may measure from $\frac{1}{235}$th to $\frac{1}{1800}$th of an inch in their longest diameter, and which are commingled with a greater

Fig. 11.

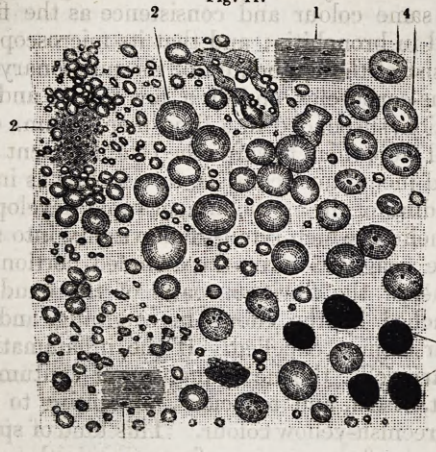

Fig. 11.—1. Portions of basement membrane.
2 Exudation masses and cells.
3. Casts of pulmonary cells.
4. Mucus-cells.

number of mucus-corpuscles than is present in cellulitis only. It is in cases of this kind that the lesion termed "bronchial abscess" is apt to occur. After desquamation of the epithelium of the affected bronchi and pulmonary cells, exudation is poured out in quantity sufficient to effectually obstruct the former tubes, and consequently to prevent the air from gaining access to the corresponding pulmonary cells. A complete or partial collapse of the latter takes place accordingly, as they are free from exudation or occupied by it. It has been already shown, that exudation seldom or never occupies the whole cavity of the cells; nevertheless it does so, in reference to individual cells, in every case of cellulitis. The explanation, however, of this occurrence is, that such individual and complete cell-distension is not the result of exudation from any one particular cell, but that it rather results from the accumulated contents, in one cell, of several cells opening the one into the other, in connection with terminal bronchi, and that the distension occurs in cells which open directly into the bronchi; because, during the establishment of pulmonary collapse, the contractility of the cell-walls produces an outward tendency of the contained fluids, which are thus urged forward to the immediate seat of obstruction in the bronchi, into which they cannot of course pass. In the course of a terminal bronchus several such distensions of immediately communicating cells may exist; and, as the pulmonary tissue in connection with them is now in a state of collapse, and as the exudation, from its inherent vitality,

passes more or less into cell development, such occurrences obtain the features of bronchial abscess, to which further consideration will hereafter be given.

In the asthenic form of cellulitis, the total exhaustion of the vital tonicity of the affected capillaries allows fluid in such abundance to escape, that the sputa are much less viscid and tenacious than in sthenic cellulitis; whilst the comparatively deficient vitality of the exudation limits, as it were, cell-growth to a development of less diameter, and of a less spheroidal shape, than that of healthy or sthenic exudation. Under a persistence of capillary engorgement, after exudation has occurred in either sthenic or asthenic cellulitis, there is, for the reasons before stated, a strong disposition in the exudation to pass from its primitive form of cell to that of the plastic and pus type; but the disposition to assume the last-mentioned character is here, as in all other parts of the bronchio-pulmonary membrane, more manifested by asthenic than by sthenic exudation.

When, however, in both sthenic and asthenic cellulitis, the sinking stage of the disease has arrived, the condition of the system is one and the same in both instances; therefore its vital manifestations, as evidenced in the nature of the pulmonary discharge, are also the same in degree and speciality of character. At this stage of the disease we find that, so long as the power of expectorating continues, the sputa are increased in quantity, and altered more or less in consistence and colour. From a thick yellowish-white, the fluid now discharged has passed to a dirty brown, or reddish-brown, colour; and, although moderately tenacious, it is less viscid and consistent than at an earlier stage. Its microscopic characters are well delineated in the following figure, which represents the appearance of the sputum, in a case of extensive cellulitis, forty-eight hours before death.

Fig. 12.

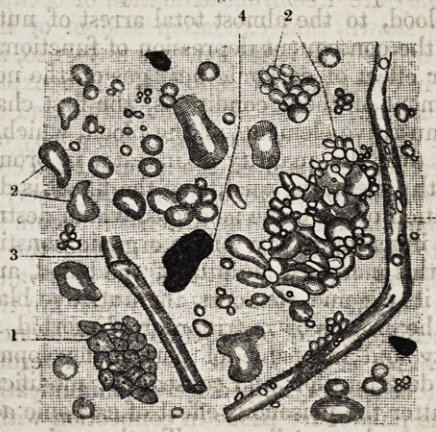

Fig. 12.—1. Epithelial patch.
2. Exudation masses and cells, showing their deficient vitality by their irregular shape.
3. Bronchial casts.
4. Mass of simply coagulated exudation.

At a more advanced period of the sinking stage, the sputa are yet more copious, of a thinly viscid character, and of a prune-juice colour, or of various modifications of this colour. Under the microscope, such sputa are observed to have manifested but a mere attempt at cell-growth, and to present shrivelled and abortive cells, cell-walls, minute basement patches, and occasionally bronchial casts, as in the subjoined figure, which represents the microscopic appearance of sputa discharged, during the last hour of life, in a case of cellulitis supervening on bronchitis of ten days' duration.

Fig. 13.

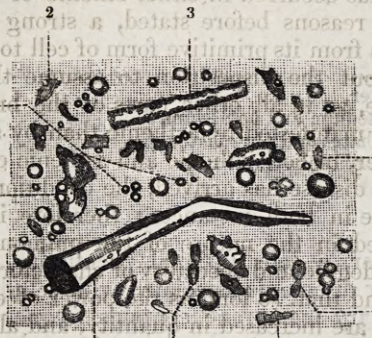

Fig. 13.—1. Minute and abortive cells and cell-walls.
2. Minute basement patches of exudation.
3. Bronchial casts.

The pathology of the above change, as contrasted with the appearance of healthy exudation, is rendered intelligible by reference to the failing powers of respiration, to the consequent deterioration in the quality of the blood, to the almost total arrest of nutrition thereby occasioned, and the consequent depression of functional energy, and to the particular effect of carbonic acid gas on the nervous system. In the establishment of these conditions, the first change is in the quantity of the nutritive elements of the blood, which, owing to the excess of egesta over the quantity of ingesta, is brought below the standard of health; next, the quality of the blood is deteriorated by the partial retention of carbonic acid gas, which destroys, to a certain extent, the inherent vitality of its organic constituents. As a consequence of this increased fluidity of the blood, and of the deficient vitality of its organic elements, the nutritive blastema poured forth, either in the process of nutrition or of morbid action, is deficient in quantity and incapable of perfect development. Hence arise structural deterioration and consequent modification of function, to which latter the poisonous effect of carbonic acid gas on the nervous system greatly contributes. To the action of this gas on the colouring matter, and also on the inherent vitality of the cell-

walls, of the red globules of the blood, to the mutual interchange of fluid between the serum and the fluid contents of the red globules, in the maintenance of their respective specific gravities, to the complete exhaustion of the vital tonicity of the pulmonary capillaries during the sinking stage, to their now passive engorgement, and to the ready escape of fluid through their inter-molecular spaces, must be ascribed the frequent presence of the colouring matter of the blood in the sputa discharged during the last hours of life.

As to the chemical composition of the sputa in cellulitis, after the full establishment of expectoration, the mean of eighteen analyses, uninfluenced by hereditary taint or constitutional peculiarity, gave,—

	In 100 parts.
Water, ...	94·80
Organic matter, ...	3·68
Chlorides of sodium and potassium, Alkaline sulphates and phosphates, ...	1·47
Sulphate and phosphate of lime, ...	·05
	100·00

When, however, a constitutional peculiarity, a particular diathesis, or another disease, is associated with cellulitis, the exudation from the pulmonary membrane contains frequent evidence of the presence of certain products, dependent on such peculiarity of constitution or other disease in the system.

I have, consequently, found the uric acid diathesis furnish urate of ammonia to the sputa of cellulitis,—the oxalic acid diathesis, distinct crystals of the oxalate of lime, together with the occasional presence of cystine,—and jaundice, cholesterine and the colouring matter of the bile.

On these points the following cases are interesting:—

CASE I.—*Acute Articular Rheumatism, associated with Pulmonary Cellulitis, with the Occasional presence of Urate of Ammonia in the Sputa.*

L. P., ætat 55, of a nervo-sanguineous temperament and intemperate habits, was seized with articular rheumatism on September 4th, 1852. The attack commenced in the ankle-joints, and affected the larger joints in succession. Present also epithelial bronchitis. On the second day the quantity of urine discharged in twenty-four hours was eighteen ounces. Colour, deep amber; sp. grav. 1·024; temperature, 94° F.; deposits on cooling a thick yellowish-red sediment of urate of ammonia. Expectoration of a thinly viscid, frothy sputa, consisting of epithelial patches and fully developed mucus-corpuscles. During the following night breathing became oppressed, accompanied by a sense of weight and burning in the posterior and lower half of the right side of the chest, by cough and partial suppres-

sion of expectoration. The physical signs indicate the existence of cellulitis in this portion of the lung. On the morning of the 3d day urine of a reddish-brown colour; sp. grav. 1·022; temperature 94° F.; a sediment of urate of ammonia on cooling. Expectoration re-established, and hourly becoming more copious. Under the microscope, it is seen to consist of a few epithelial patches, mucus-corpuscles containing distinct globules of urate of ammonia, free urate of ammonia, exudation masses and cells, and irregularly shaped flakes and masses of fibrine, as represented in the adjoining figure.

Fig. 14.

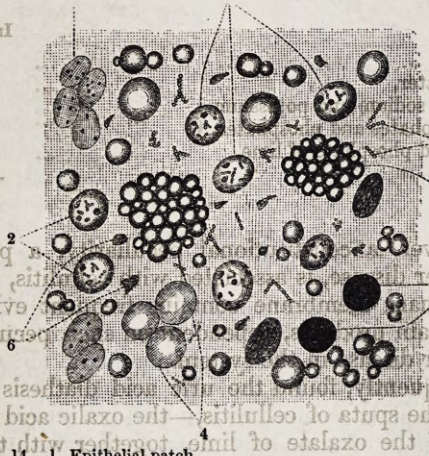

Fig. 14.—1. Epithelial patch.
2. Mucus-cells containing urate of ammonia.
3 Free urate of ammonia.
4. Exudation masses and cells.
5 Casts of cells.
6. Nodules of simply coagulated exudation.

During the third and following day, the urate increased in the urine and decreased in the sputa. On the fifth day, although the sputa were very carefully examined, no trace of it could be discovered. On the ninth day it re-appeared in the sputa, at which time the urine was quite free from it; but on the morning of the eleventh, and during the remainder of that day, a copious excretion of the urate took place from the kidneys; and on the following day there was no appearance of it in the sputa. From this time the urate was confined to the urine, in which, on the fourteenth day of the disease, a very copious sediment occurred. The cellulitis henceforth rapidly recovered; and, on the twenty-second day, convalescence from rheumatism was established.

To prove chemically the existence of the above salt, an ounce of the sputa was evaporated to dryness, at a gentle heat, in a porcelain capsule, over a sand bath; the dry residue was triturated in a mortar, and afterwards digested in warm distilled water. The whole

was next thrown on a filter, and to the filtered fluid animal charcoal was added, and digestion, at a gentle heat, again employed for an hour. After procuring, by repeated filtrations, a clear fluid, the latter was concentrated. A few drops of the concentrated solution were allowed to crystallise on a slip of glass, on which the following crystals were seen under the microscope. (Fig. 15.)

Fig. 15. Fig. 16.

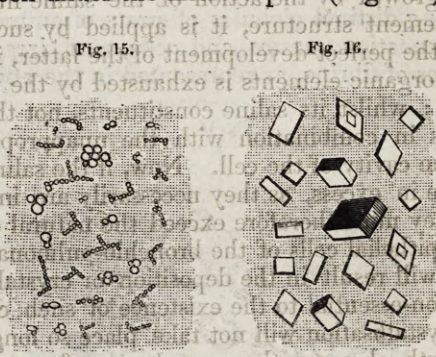

Fig. 15.—Urate of ammonia.
Fig. 16.—Crystals of uric acid.

To the remaining portion of the concentrated solution a few drops of hydrochloric acid were added; and in a short time crystals of uric acid, as above represented, were procured. (Fig. 16.) These were soluble in liquor potassæ and nitric acid, which latter, by evaporation, gave a pink residue, which became purple on being exposed to the vapour of ammonia, thus demonstrating the presence of the purpurate of ammonia of Prout, and the murexide of Liebig.

It is clear that, in the above case, which is but one of a number which has occurred to me within the last three years, the presence of rheumatism produced an occasional modification of the pulmonary discharge which existed as a consequence of cellulitis, and that this modification was manifested by the presence of urate of ammonia in the sputa, whenever it had, in consequence of a temporary suspension of the eliminating action of the kidneys, accumulated in undue proportion in the blood. So soon as the excretory power of these organs was again excited, an abundant deposit of the urate appeared in the urine, and all trace of it quickly disappeared from the sputa. When present in the latter, it existed both in a free state and as a deposit from the contents of the mucus-corpuscles, whilst the exudation masses and individual exudation-cells were invariably free. This fact seems to depend on the difference in the conditions of growth between the mucus-corpuscle and the exudation-cell, of which the following explanation may be given:—

The germinal point or centre of the future epithelial or mucus-corpuscle is surrounded, in its nutritive matrix, by the necessary

organic elements for its perfect development, which elements require the incessant yet gradual action of the saline constituents of the blood to render them subservient in the process of growth. As soon, therefore, as the smallest particle of the superficial layer of the basement membrane around a germinal centre has been rendered subservient to growth by the action of the saline fluid which permeates the basement structure, it is applied by such germinating centre to effect the perfect development of the latter, in doing which a portion of its organic elements is exhausted by the vital extension of the cell-wall; whilst its saline constituents, not thus necessarily localisable, pass in combination with the unappropriated organic elements into the cavity of the cell. Now, if the saline constituents of the blood are in excess, as they necessarily are in similar cases to the above, they must therefore exceed the natural demand in the growth of the epithelial cells of the bronchio-pulmonary membrane, and this excess will result in the deposition or crystallisation of the salt, which is non-essential to the existence of such cell. But this deposition or crystallisation will not take place so long as the cell is in absolute growth; because the same degree of temperature and of moisture, which maintained the solubility of the salt during its passage into the cell, is still present to maintain its solution within the cell. When, however, the cell has acquired a perfect development, and when, in consequence, it becomes a detached and isolated body, its temperature immediately falls, the fluidity of its contents diminishes by evaporation, which may cause a portion of its contained organic constituents to assume the semi-solid condition (hence the formation of granules and nuclei), whilst any abnormal salt is at once thrown down by amorphous deposition or distinct crystallisation.

In the germination and growth of the exudation-cell, however, the conditions are different; for here the germinating point is not situated within the substance of the basement membrane, but upon its free surface left bare by the shedding of its epithelium; and its connection with the basement structure is not a normal, but simply an abnormal, accidental, or adventitious connection, which results from the particular pathological condition of the part, and which resembles, in a great degree, the connection which exists between a plant and its parasite.

Growth, therefore, takes place by imbibition of the organic constituents, which, with the germinating point, or points, form the coagulated portion of the exudation, and which are rendered subservient to the vital activity of such germinating points or centres, by the resolvent action of the fluid portion of the exudation poured out upon the denuded basement membrane.

As, however, this fluid portion of the exudation is immediately exposed to the direct contact of the air, by which its temperature is reduced and a part of it evaporated, it is evident that any abnormal salt of the blood, present in the fluid exudation, will undergo imme-

diate deposition or crystallisation, and will not, therefore, enter the exudation-cell. Hence the reason why, in the above case, urate of ammonia was present in the epithelial or mucus, and not in the exudation-cell: and hence also, in explanation of the immunity of the latter cell, the reason why this salt existed as a free constituent of the sputa.

CASE II.—*Oxaluria—Pulmonary Cellulitis—Occasional Presence of Oxalate of Lime in the Sputa.*

W. S. B., a medical student, ætat 20, of a tuberculous constitution, and nervo-sanguineous temperament, consulted me on October 6th, 1852, for an occasional sharp, cutting pain in the loins, followed by a discharge of very bright, amber-coloured urine, which became extremely turbid on cooling. He stated that he had thus suffered for three months, that the pain generally affected him very suddenly, and that it was so lancinating and acute as to cause him involuntarily to sink beneath it. He had lost flesh, and of late his general health had rapidly been getting worse. His countenance is anxious and haggard; skin pale, harsh, and shrivelled; tongue large, flabby, red at the tip and edges, but covered with a white creamy fur in the centre; appetite bad; bowels irregular, but the evacuations generally of natural colour and consistence; pulse 89, small and irritable; disposition fretful; sleep disturbed by dreams; frequent desire to pass urine, which is of a clear, bright amber colour when first voided, but deposits on cooling a heavy white sediment; sp. grav. 1·026; temperature, 93° F. On heating the urine gently, the greater part of the sediment is dissolved, whilst almost the whole of the remainder is soluble on the addition of nitric acid; but a small proportion resists the combined action of both heat and nitric acid. A drop of the sediment, placed under the microscope, exhibits great numbers of the usual crystalline forms of urate of ammonia and beautiful octahedral crystals of oxalate of lime, crossed diagonally by light-coloured bands. In the supernatant fluid, the ovoid and dumb-bell forms of oxalate of lime abundantly exist. There are also present in the urine epithelial scales both from the lining membrane of the bladder and uriniferous tubes, mucus-corpuscles, and a few exudation-cells and uriniferous casts. After being under treatment for a fortnight, he took cold from incautious exposure, and pulmonary cellulitis supervened. On the establishment of the expectorant stage the sputa were daily examined, the result of which proved the occasional presence of the above salts, particularly at the time when they were being eliminated by the kidneys in small proportion, and when, in consequence, their accumulation was taking place in the blood. Crystals of the oxalate were present in the mucus-corpuscles to a limited extent, being for the most part free in the sputa; but the urate of ammonia was not infrequent as a constituent of the contents of the mucus-cells.

Fig. 17.

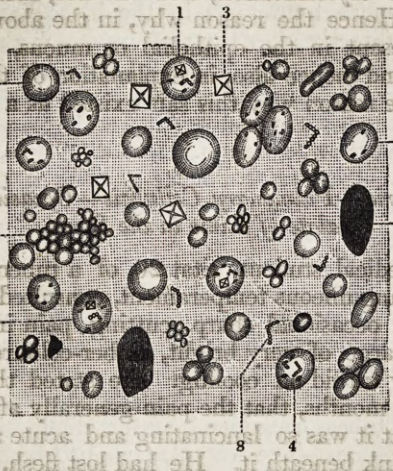

Fig. 17.—1. Mucus-cells containing urate of ammonia and octahedra of oxalate of lime.
2. Mass of exudation-cells.
3. Free crystals of oxalate of lime.
4. Mucus-cell containing urate of ammonia only.
5. Coagulated fibrine, showing the commencement of cell development.
6. Exudation-cell of full growth.
7. Mucus-cell of full growth.
8. Free urate of ammonia.

To prove chemically the existence of the oxalate of lime, two ounces of sputum were evaporated to dryness over a sand bath, and the dry residue was incinerated. To the ashes thus left, distilled water was added, which dissolved out the soluble salts. The insoluble salts were then collected, and thrown into nitric acid and water, when a distinct effervescence occurred, together with the escape of carbonic acid gas and the solution of the salts. To a few drops of this solution in a watch-glass, a drop or two of a solution of oxalate of ammonia were added, which produced an immediate white precipitate of oxalate of lime, which underwent no change either in boiling acetic acid or liquor potassæ.

To a drop of sputum between two slips of glass, a single half-drop of dilute nitric acid was added, and the solution of the oxalate was watched under the microscope.

The above case was clearly one of oxaluria, in which cellulitis supervened as a mere accidental occurrence from exposure to cold. It was, however, evident that the bronchio-pulmonary membrane became at times a partial outlet for the urate of ammonia and oxalate of lime, which action, although the result of disease in the membrane itself, nevertheless exerted a salutary influence in the purification of the blood. But the eliminative action of the membrane was here, as in the case first detailed, only manifested when

the excretory action of the kidneys was insufficient to prevent an accumulation of morbid products in the blood, and it invariably ceased for a time directly after the appearance of an unusual quantity of the above salts in the urine. The elimination of such products, therefore, by the bronchio-pulmonary membrane, can only be regarded as an extraordinary occurrence, and vicarious for the time being of a deficient renal excretion. It, nevertheless, shows the conservative tendency of the system at large, and the disposition which exists between structures of a similar nature to sympathise with each other, and to assist, by a compensatory action dissimilar to their own, any part or structure the functional activity of which is for the time unable to prevent the undue accumulation of injurious products in the blood. In the above case the presence of uriniferous casts in the urine would have afforded ground for serious apprehension as to the structural integrity of the kidneys, had not their existence been rightly referred to a degree of inflammation of the mucous lining of the uriniferous tubes, occasioned by the elimination and passage of the oxalate of lime. The vascular engorgement thereby produced, led to epithelial desquamation in the uriniferous tubes, and to exudation upon the denuded basement membrane, and sometimes into the uriniferous tubes themselves, producing casts of the latter in a manner similar in all respects to what has already been stated, in reference to inflammation of the bronchio-pulmonary membrane.

The presence of uriniferous casts was invariably associated with an abundant deposit of the oxalate; and at this time, too, the urine became slightly albuminous. When but a scanty deposit of the oxalate existed, the casts and albumen disappeared almost entirely from the urine, in which they re-appeared on the recurrence of the salt in unusual quantity. These results clearly showed the relation of cause and effect between the oxalate and the other morbid products observed; and the inference thence drawn, was fully justified by the complete and permanent disappearance of the latter, when the natural and healthy action of the kidneys had been fully restored.

CASE III.—*Jaundice—Pulmonary Cellulitis—Cholesterine and Biliphœin in the Sputa.*

S. S., ætat 36, sanguineous temperament, married, and mother of six children, was attacked with jaundice about three weeks before her last confinement, which happened on October 2d, 1852. Medical attendance was not requested until five days after labour, when, in consequence of having taken cold on the previous day, she complained of cough, straitened breathing, together with a sense of weight and uneasiness in the left scapular and subscapular regions. On visiting her, I found the following symptoms and physical signs:—Skin and conjunctiva of a deep yellow colour; tongue coated with a dirty, yellowish-white load; thirst, loss of appetite,

frequent vomiting, constipation, alvine evacuations knotty and clay-coloured; urine, deep greenish-yellow. Respirations twenty-four per minute, somewhat laboured; cough frequent, short, and dry; pulse 94, full and strong; skin hot and dry. In the regions above noted there is comparative dulness on percussion; the vesicular murmur is diminished in intensity, but is nowhere absent in the affected parts; bronchial respiration and bronchophony are heard in low intensity over the seat of disease, together with puerile respiration at the apex of the affected lung.

On the establishment of the expectorant stage, the sputum expelled was similar in consistence to that of ordinary cellulitis, but its colour was of a strongly marked greenish-yellow, from which it never varied during the whole course of the disease. Viewed under the microscope, the usual bodies of cellulitis were observed, in addition to which plates of cholesterine were present.

To prove chemically the existence of the latter, three ounces of sputum were evaporated to dryness over a water bath, and the dry residue was digested in ether for several hours. The solution thus obtained was next decanted and allowed to evaporate. The residue left by evaporation was washed with cold alcohol, which removed a small proportion of oily non-crystallisable fatty matter, leaving the cholesterine almost free from impurity. A small portion, placed under the microscope, exhibited the usual transparent crystalline plates of this substance.

To another portion of sputum a few drops of nitric acid were added, when, after a few minutes, the yellowish hue of the former gave place to a permanent green, thus indicating the presence of bile.

From the foregoing cases, which are but single examples of a number of such that has occurred to me, the following deductions seem to be justified:—

1. That the manifestation of a particular diathesis, or the presence of a particular disease in the system, is capable of influencing the character of the sputa in inflammation of the bronchio-pulmonary membrane.

2. That this influence is manifested by the presence, in the sputa, of the morbid products of the particular diathesis or disease with which inflammation of the bronchio-pulmonary membrane is associated.

3. That such morbid products are eliminated by the epithelial cells of the membrane; that they, therefore, appear as accidental constituents of the mucus-cells; that they likewise escape in the exudation from the denuded surfaces of the basement membrane; and that, for the reasons before-named, they are never present in the exudation-cells.

4. That the bronchio-pulmonary epithelium can thus act the part of an excretory organ; but that this peculiarity of action is

probably rather dependent on the particular character of the blood, than on any selective power inherent in the epithelial cells.

5. That, before such eliminative action takes place, an undue accumulation of morbid products occurs in the blood; that the action of the different organs, intended for the elimination of such morbid products, is for the time being deficient; and that, on its becoming more vigorous, the sputa quickly regain their simply morbid character.

The indications of treatment to be deduced from the foregoing observations, are the same in the second pathological stage of cellulitis as in that of bronchitis involving the submucous tissue, to which latter the reader is therefore referred.

(*To be continued.*)

ARTICLE II.—*On the Improvements which have been Introduced into the Practice of Surgery in Great Britain within the last Thirty Years.* By JAMES SYME, Esq., Professor of Clinical Surgery in the University of Edinburgh.

(*Communicated to the Edinburgh Medico-Chirurgical Society.*)

THE great improvements which in recent times have been so profusely contributed to the well-being of society by other departments of practical science, frequently suggest the question, Has the practice of surgery advanced in a proportionate degree? The older members of our profession, who have not quite kept pace with the march of progress, are apt to express doubts as to any real advantage having been gained of late; and our younger brethren, who are occupied more with learning present opinions than in tracing their origin, although inclined to believe that things are better than they used to be, feel rather at a loss, when called upon, to explain the grounds of their persuasion. In these circumstances, I have thought that it might not be improper for a member of this Society, who can speak from his own observation and recollection, to mention some of the improvements which have been introduced into the practice of surgery within the last thirty years.

In attempting to execute this task, I do not propose to record those particular achievements of operative surgery which merely reflect credit upon the skill and intrepidity of the gentlemen who performed them, or those new modes of treatment which are either decidedly objectionable or of questionable expediency. The excision of ovarian tumours; the aperture of the larger intestine to afford relief from obstructions of the bowels; the opening of the abdominal cavity in search of internal strangulations; the excision of the knee-joint instead of amputation; the remedy of anchylosis by forcible rupture of the adhesions between the articulating surfaces,—and other less pro-