The most careful examination has failed altogether to detect any separate active principle, though it has been shown that the air of marshes contains a variety of products that are absent from the normal atmosphere, e.g., excess of carbonic acid (6 to 8 per 1,000 volumes), sulphuretted hydrogen, phosphuretted hydrogen, watery vapour, ammonia, free hydrogen, organic matter, and the microscopical *débris* of vegetable and animal matter. One or other or several of these may be present, but they are not malaria. That some subtle agency, in addition to climatic influences, is at work, seems probable; but whether it be one or more, we know not. The theory of the material nature of malaria is, at any rate, a good working hypothesis; as such only would I receive it, pending more definite information.

A glance at the distribution of fevers in India will show that they occur in districts that present opposite characters, and it would be easy to show that they are sometimes absent altogether where marshes and other conditions presumably favourable to their existence abound; and that in arid regions in Africa, Spain, and on the sandy soil of Walcheren, in Greece, the Island of Ascension, etc., the fevers* are often severe.

MacCulloch believed in malaria from a variety of sources: the surface of damp ground; stagnant waters, from the lake to the smallest pool; the dung-heaps and pools at the doors of farmhouses and cottages; from sewers and ditches; from the mud left by the recess of the tide, and at the mouth of rivers; marshy and swampy spots in low situations near woods and on roadsides, or small spots of coppice and brushwood in England. He mentions a case in the West Indies where a number of men were seized with fever from exposure to ground which, by the removal of boxes, was exposed to light and heat. From the mere *malaise* of disordered health which many people feel in certain localities that are damp and ill-ventilated, to the severest form of remittent, he ascribes them all to malaria. We are not wont to call it malaria unless it produce periodic fever, neuralgia, or cachexia; but we do find people recover from a depressed state of health on removal from a damp locality, and in this sense we may call the cause malaria; and the ill effects of such localities on persons who have previously, it may be long before, suffered from tropical fever, are common. I have seen cases, to whom each autumn brought back vague indefinite symptoms of malarial fever, or neuralgia, and rheumatism, restored to health by quitting the locality at that season. Lately, we read of malaria from flower-pots. I have heard of it from watering a flower-bed in India, and of an insidious disease arising from the presence of living plants in the hot damp air of rooms.

Malarial fever is the origin of most of the diseased conditions I have to deal with in connection with Indians who appear before me at the India Medical Board; but I occasionally see cases that present the same appearances, and, indeed, who suffer from periodic fever, who have never been in the tropics, and for whom it is not possible to find a malarial origin for the disease.

The existence of malaria as a particulate thing has not yet been demonstrated, and it is still asked if such a thing exist. It may be so; there are circumstances connected with its action which are difficult to reconcile with a parasitic origin; and, for the present, our attitude with respect to that view, much as we may wish that it should prove true, must be one of reserve; but who that has followed the progress of pathological investigation during the last quarter of a century would venture to assert that, in such researches as those of Pasteur, Burdon Sanderson, Lister, Greenfield, Koch, Klebs, Tommasi-Crudeli, and others, we may not find a complete solution of the problem?

[To be continued.]

DEATH AND DUTY.—We regret to have to record the recent death of Dr. J. T. Gadsby of Mansfield, who died from illness contracted during the performance of his professional duties. It appears, according to the *Nottinghamshire Guardian*, that some weeks ago a serious outbreak of diphtheria occurred at the Mansfield Workhouse, and Dr. Gadsby, as one of the medical officers, devoted much time and labour to prevent the spread of the infection, and to aid the distressed children. When the contagion was at its height, seventeen children and the schoolmistress of the house having caught the disease, Dr. Gadsby contracted the malady, and was obliged to relinquish active duties. He was promptly attended by Dr. Godfrey and Dr. Sparke, and diphtheria presented itself in a virulent form. Medical skill, however, proved unavailing, and he succumbed a fortnight from the date when he received the infection. Dr. Gadsby, who was an M.D. of the University of London, and formerly house-surgeon at Richmond (Surrey) Hospital, was only about thirty years of age, and his loss is keenly felt by a wide circle of friends.

THE GULSTONIAN LECTURES ON PULMONARY CAVITIES: THEIR ORIGIN, GROWTH, AND REPAIR.

By WILLIAM EWART, M.D. Cantab., F.R.C.P.,
Assistant-Physician and Pathologist to the Brompton Hospital for Consumption;
Physician to the Belgrave Hospital for Children; Demonstrator of
Physiological Chemistry at St. George's Hospital.

LECTURE II.—PART I.

AMONG the varieties of pulmonary consumption, enumerated in my last lecture, there is one which, in its history, differs so widely from the rest, that I have ventured to isolate its description from that of other forms. Whereas, in the ordinary cases, the earliest symptoms are trivial, and it is almost impossible to trace with accuracy the beginning of phthisis, in this variety the transition from health to disease is indicated by a striking landmark, never overlooked by the patient nor by the physician. The causal relation implied in the phrase "phthisis ab hæmoptoe" had been accepted from the days of Hippocrates down to modern times—as a self-evident truth, even more than as a dogma. It may be taken as no small proof of the scepticism of the nineteenth century, that Niemeyer's reassertion of so venerable a statement should have called forth so much astonishment, and so strong an opposition. To what extent Laënnec's teachings may have assisted towards this effect of novelty, I will not attempt to discuss. Laënnec's assumption of a tuberculosis preceding and causing hæmoptysis (see *Traité de l'Auscultation Médiate*, vol. i, page 646), reasserted with less reserve by some of his followers, remains to this day unproved, although the *onus probandi* rests with his, rather than with the more ancient, doctrine.

Daily experience teaches us that hæmorrhage forms no essential part of consumption; neither does phthisis invariably follow upon hæmoptysis. Of this, we are reminded by Niemeyer's first proposition: "Abundant bronchial hæmorrhage occurs very frequently, it is admitted, in such persons who are neither consumptive at the time of hæmorrhage, nor become so afterwards." (*Clinical Lectures on Pulmonary Consumption*, Sydenham Society's translation, page 38.) Could there be any clearer proof that, in a large number of cases, tuberculosis is not the prime factor in pulmonary consumption? On the other hand, the time-honoured tenet, "phthisis ab hæmoptoe", was not adopted by Niemeyer without an important modification. According to him, catarrhal pneumonia forms a necessary link between the hæmorrhage and the phthisis.

To Dr. Reginald E. Thompson belongs the credit of having taken up the subject at this point, and vigorously pushed forward its analytical study. Some of his conclusions so immediately concern the etiology of cavities, that they claim our attention; but, for a full account of his interesting views on the subject of hæmoptysis, I must refer you to his original paper in volume lxi of the *Royal Medical and Chirurgical Society's Transactions*, "On the Pathological Traces of Pulmonary Hæmorrhage," and to his work on *The Causes and Results of Pulmonary Hæmorrhage* (London, 1879). The term "hæmorrhagic phthisis", frequently used by clinical observers, is not free from the reproach of ambiguity. Hæmoptysis, even of recurrent type, occurs from three very different causes: 1. As the result of confirmed phthisis; 2. As an early complication of the congestive forms of the disease; 3. As an event not dependent upon pre-existing phthisis, but capable of producing the latter. The results in the three cases are equally divergent. The first variety of hæmoptysis, commonly due to pulmonary aneurysm, usually terminates in fatal hæmorrhage. The second, supervening in the midst of a pneumonic phthisis, almost invariably leads to serious exacerbations, and to a rapid breaking down. There can be little doubt that the third variety comprises the strictly hæmoptotic forms of consumption. According to Dr. R. E. Thompson, hæmophilia bears the responsibility of the hæmorrhage in a majority of these cases; and, if I understand him rightly, phthisis and excavation are events subsequent to the local disablement of the lung by the products of hæmorrhage. The stagnation of the residues of hæmoptysis had been mentioned by Morton and by Hoffmann; and Niemeyer had described their infiltration in the alveoli as a factor in catarrhal pneumonia. But Dr. Reginald E. Thompson was the first to emphasise that the worst results

are apt to ensue, not so much in the vicinity of, as at a distance from, the seat of the hæmorrhage, and in localities which are almost constant. These localities are roughly depicted in the annexed diagram, according to Dr. Thompson's description.

Fig. 1.—Usual situations of "Hæmorrhagic Deposits" (according to Dr. Reginald E. Thompson).

In profuse hæmoptysis, a single cough is insufficient to empty the air-passages; and the deep inspiration which follows draws into the bronchi a quantity of blood, which fills some of the lobules. Once in possession of the alveoli, the blood escapes the *vis a tergo* of expiration, and every inspiratory effort results in a deeper impaction of the clot. This blood, according to Dr. Thompson, never leaves the lung unless it be coughed up once more in the process of excavation.

It would carry me too far to attempt any detailed description of the resulting *hæmorrhagic nodules*, of their aspect, of their constitution, and of the puckering and pigmentation which they occasion. My present inquiry more specially concerns the subsequent fate of the nodules. The excavation to which they frequently give rise is slow to be established, but is rapidly carried through. The tolerance of the lung for solid residues of fibrin is a proof, according to Dr. R. E. Thompson, of the non-irritating quality of the blood; and the absence within the deposits of degeneration is due to the plug of fibrin which extends for some distance up the bronchus, effectually protecting the bulk of the deposit from contact with the air. Spontaneous breaking down occurs in the nodule whenever septic matter was originally mixed with the impacted blood; hence the disastrous effects of hæmoptysis in an incipient catarrhal pneumonia, especially if already softening. The blood inhaled under these circumstances leads to more of pneumonia, and to less of the consolidating fibrosis, than would have resulted in healthy tissue, and the masses are from the first marked out for disintegration. More usually the softening is secondary to the dislodgment or to the absorption of the protecting plug, or else to the progress of ulceration from a neighbouring cavity. In the more fortunate cases, the nodule, as elsewhere described, softens within its membranes; its contents may be coughed up, and the resulting vomica may undergo contraction. The stiffness, pigmentation, and the puckering of the limiting layer, which often sends out fine radiating striæ into the surrounding spongy tissue, for a long time, indicate the mode of origin of these cavities. At the time when excavation overtakes the deposit, the process of fibrosis is usually an accomplished event. As long as it remains confined within the limits of the capsule, the destructive process excites in the lung neither irritation nor reaction; but should the membrane, as too often happens, give way, the surrounding tissue, taken as it were by surprise, can offer no obstacle to a spread of the irritative pneumonia far beyond the limits of the original hæmorrhagic infiltration.

Should the nodule be seated at the periphery, the same insidious ulceration may attack the pleura. I believe this event to be a common cause of pneumothorax; and it should be noted that the posterior axillary region, where perforation of the lung frequently takes place, is also one of the situations pointed out by Dr. R. E. Thompson as most obnoxious to the inhalation of blood in hæmoptysis. The sternal region, which is especially liable to blood-nodules, is rarely the seat of perforating ulceration. I have observed, however, an instance in which perforation resulted in this situation; and, in another case, softening of a blood-nodule at the base was followed by the same event.

Cases in which hæmoptysis is followed by extensive and rapid softening probably possess a constitutional bias which intensifies the mechanical results which I have attempted to describe. They supplied Laënnec and his followers with a plausible argument in favour of a tuberculo-

sis anterior to the hæmorrhage; but precisely in these cases I have observed relatively more of pneumonia than of tubercle; and their later history has closely resembled the march of ordinary pneumonic phthisis.

Tuberculosis, on the other hand, is the common mode of termination in those cases where hæmorrhage leads to chiefly local damage, just as it is the most frequent termination of other forms of chronic excavation. The constitutional element of phthisis is almost absent from their primary stages; the pulmonary changes which they suffer as the mechanical result of hæmoptysis are changes characteristic of healthy lung-tissue. With the recurrence of hæmorrhage, the lung becomes more and more disabled, and consumption may be finally established. But there is much in the earlier progress of the disease which is capable of a mechanical explanation; and, in a great measure, the phthisis may be viewed as incidental to the results of hæmoptysis.

With these cursory remarks I close the subject of the etiology of excavations; and I would invite you next to a study of the anatomical characters and of the functional peculiarities of pulmonary cavities.

The *shape* of vomica is generally given by the mode of their formation. They originate in deposits, which, being usually peripheral and situated around a bronchial termination as a centre, impart to the softening roughly a spherical outline; but their ultimate contour is governed by a variety of circumstances, especially by the extension of the inflammation in one or the other direction, and by the coalescence of outlying groups. This fortuitous element renders it impossible to point to any special variety in shape as being absolutely typical of cavities; and we cannot apply literally to them the statement of Rindfleisch, "that every space acted upon by uniform forces, either from within or from without, tends to a spherical shape." Even in their later stages, when the contraction which I shall have occasion to describe approximates their internal surfaces, the forces acting upon various points of their circumference are usually unequal. Pleural adhesions are a source of irregular traction. The persistence of bronchial connections favours shrinking in definite lines, and the influence of outlying consolidations further disturbs the even pressure of the spongy tissue by which they are surrounded.

A very different view of the shape of cavities is implied in Gerhardt's attempt to determine during life the longest diameter of a cavity by means of Helmholtz's resonators. This method might have led to better results, did cavities usually possess a simple outline and a smooth internal surface. Such is far from being the rule. Cavities are often traversed by trabeculae; and they are commonly provided with irregular prolongations, which explain the failure of Gerhardt's ingenious device. A safer guide is to be found in a combination of the ordinary methods of auscultation with a careful regard to the anatomical relations, and to the average shape of cavities as observed after death.

Whilst admitting the great uncertainty of the shape of cavities, I am led to believe that, in definite districts of the lung, excavation roughly tends to assume definite development. Nowhere is this more apparent than in the pectoral region. Superficial cavities in this situation, if of sufficient size, invariably acquire a flattened condition in harmony with the bronchial distribution. Cavities at the apex proper are ovoid or rounded; in the mid-dorsal region, they are habitually spherical; but in all situations, whenever excavation increases largely, it tends more and more to assume the ultimate dimensions of the lobe affected.

More important than the inquiry into the shape of cavities is a determination of their *size*. When cavities approach the surface, this determination can be effected without much error; but it is rendered uncertain by the presence of an intervening layer of lung-tissue, whether spongy or consolidated. In the first case, the cavernous sounds are imperfectly transmitted; in the second, they are apt to be conducted beyond the excavated region. Thus it frequently happens that the cavity diagnosed is larger than the cavity discovered after death. On the other hand, cavities may be, and often are, underrated by the ear; putting aside the comparatively rare cases in which their detection is rendered impossible, owing to the occlusion of the corresponding bronchus. Our estimate of the size of vomica is further unsettled by the presence of trabeculae and of secretion, which restrict the area of sound-production, or impair the sonorous vibrations.

It is no part of my present duty to point out the element of a correct clinical diagnosis. My object is simply to call attention to the great assistance which our practical conclusions would receive from a more accurate knowledge of the anatomical varieties of excavation.

It is customary, in describing cavities, to allude to the *trabeculae* which they often contain. The absence of trabeculae in cavities of recent date points to their having been produced from an extensive pneumonia, or from some equally destructive congestion affecting the whole area of excavation. In chronic cavities, the removal of tra-

beculæ is effected by a slow superficial necrosis, which I have described as never absent from the internal surface of cavities; it is hastened by all those circumstances which promote the necrotic process, and especially by maceration. As an instance of this last influence, I would point to the excavations situated at the posterior part of the upper lobe, and in the mid-dorsal region, where, owing to the supine posture, fluid secretions are apt to be retained for long periods. Anatomical peculiarities are in favour of a more extensive and of a more persistent trabeculation at the pectoral region than at other parts. In this situation, behind a tolerably shallow layer of spongy tissue, the main bronchial ramifications form a *pes anserinus* of considerable strength, and the larger divisions of the blood-vessels contribute greatly to the resisting power of the district. Thus the normal type of cavities in the pectoral region would be mainly trabecular; the normal type at the apex proper, and especially at the posterior apex, where the bronchial and vascular supply is terminal, mainly the non-trabecular form.

In size and in aspect, trabeculæ differ widely. They may form bands of substantial thickness, or mere cylindrical cords; and they are liable to present every degree of ulceration, but they all agree in possessing a lining similar to that belonging to the cavity. Their destruction usually proceeds with the greatest activity at the point furthest removed from their attachments; and, when disruption has finally occurred, the divided stumps gradually waste and disappear.

The sudden rupture of a trabecula under the influence of strain is probably of less uncommon occurrence than is generally suspected. Dr. Douglas Powell narrates a case of this nature in which the physical signs enabled him to recognise, during life, this event, which was subsequently confirmed by the necropsy.

The exact nature of trabeculæ has been the subject of some divergence of opinion even among competent observers, a circumstance which I am at a loss to understand. By some, the vascular nature of trabeculæ is upheld; and it cannot be denied that blood-vessels often constitute the only bond of connection between the opposite parietes of a large cavity. This is, indeed, the usual appearance presented by completed excavations, due to caseous pneumonia. But the smooth surface, the tough yellowish aspect, closely resembling in colour and consistency the fibro-elastic coat of a large artery, and the mode of branching special to the blood-vessels, constitute as many unmistakable features, which render impossible a confusion between the latter and trabeculæ proper. Trabeculæ have too often been stated to consist of bronchial tubes. From a large number of observations, I am able to affirm that patent bronchi very rarely, if ever, enter into their composition. I cannot recall more than one instance of a bronchus traversing a cavity of some magnitude, and even in this case the tube was the seat of incipient ulceration. With Dr. D. J. Hamilton's opinion that trabeculæ are the remains of interlobular septa, I can partially agree. I am familiar with a form of pulmonary liquefaction in which the lobules are completely dissected; here, neither tubercular fibrosis nor inflammatory thickening of the tissue obscure the simplicity of the pathological process, which is one of moist necrosis. The remarkable honey-combed condition which results is, however, far from being typical of what is usually understood by a trabeculated excavation. In the formation of the ordinary trabecula, the fibrous tissue of the interlobular septum undoubtedly constitutes an element of resistance; but the position of trabeculæ is mainly determined by the blood-vessels, and especially by the bronchial tubes. As pointed out by Dr. D. J. Hamilton, trabeculæ commonly are inserted into the side of a bronchus. If the bronchial tube which appears to enter a trabecula be carefully probed, the instrument introduced at the bronchus cannot be made to pervade the trabecula, but issues immediately in a direction parallel to it, clearly showing that the tube has been gutted. In earlier stages, the bronchial tube presents a series of fenestrations in which we readily recognise the orifices of ulcerated bronchioles. The larger air-tubes are gradually overtaken by the same ulceration, but the peribronchial sheath, with its vessels, remains an important constituent of the trabecula, and assists, in no small degree, in its conversion into fibroid tissue.

I have placed before you drawings and diagrams of trabeculæ both in longitudinal and in transverse section, demonstrating the partly alveolar nature of their structure. The sections exhibit the following component parts: 1. Blood-vessels usually contracted and thickened, and occasionally obliterated; 2. A layer of spongy substance always much collapsed; 3. Fibrous elements such as normally belong to the peribronchial sheath; 4. A false membrane analogous to that which lines the cavities. If I understand rightly the mechanism and the formation of trabeculæ, the latter are but bridges of pulmonary tissue which originally separated distinct cavities, and had escaped the influence of the disease until deprived of their air-supply by the laying open of their bronchi. The collapse of the alveolar tissue, and its transformation into a compact layer, tightly fitted around a branch of the pul-

monary artery as an axis, are nothing more than late consequences of the bronchial ulceration.

New vessels, often of considerable size, are abundantly developed in connection with inflammatory changes in the trabecula; but, of the original structures, the only one preserving functional activity is the artery. In this fact consists one of the chief uses, as well as one of the main dangers, special to trabeculæ. The trabecula remains for a considerable time the chief source of nutrition for the segment of lung-tissue into which it is inserted. As long as blood flows to the periphery through the trabecular artery, some risk of hæmorrhage may be said to exist. But, practically speaking, bleeding very seldom occurs from these vessels, which remain well protected by a layer of collapsed alveoli, until finally overtaken by obliteration. Aneurysm is remarkably more frequent in arteries of the wall than in those crossing the cavities. Sooner or later, the lumen of the trabecular vessels lessens in proportion to the contraction and to the thickening of their walls, and finally it disappears.

In their ultimate shape, trabeculæ may be said to consist largely of elastic elements, namely, collapsed alveoli and vascular membranes. The importance of this fact will become apparent when I describe the mechanism of the contraction of cavities, and will afford some excuse for the development which I have given to this part of my subject.

Concerning the blood-vessels which traverse cavities, I need add but little to what I have incidentally stated. In the moister varieties of phthisis, the bronchial and pulmonary divisions are carried away with the general softening; but where fibroid change is active, they survive for a time, and partake in the fibrosis. Of this we have seen a striking instance in caseous pneumonia. In transverse sections of trabeculæ, it is often possible to recognise the blood-vessels belonging to various systems. In the drawings and in the diagrams, which I owe to the skill of Mr. Arthur Grayling, house-surgeon to the Belgrave Hospital for Children, you will notice, in addition to numerous thin-walled and distended vessels of new formation, a central cluster of large blood-vessels undergoing degenerative thickening. The pulmonary artery, the pulmonary veins, and a branch of the bronchial artery, all come into view; and in this instance, the trabeculæ contains in addition, by the side of a lymphatic duct, a smaller bronchial tube completely obliterated by cell-growth. Among these vessels, it will suffice for the present specially to consider the most important—the pulmonary artery.

When finally laid bare by gradual removal of the condensed spongy tissue, the surface of the artery becomes an excellent indicator of the slow destruction which incessantly proceeds within cavities. This secondary ulceration of the blood-vessels constitutes the real danger of hæmorrhage. It was pointed out by Dr. Douglas Powell in his book *On Consumption* (2nd edition, 1878, page 74) that the fibrotic cavities of old standing are most likely to develop aneurysm, and that aneurysm is more especially found on the exposed side of vessels which are partly buried in indurated tissues. I have often verified the correctness of this observation. For the fact itself, the following appears to me to be a satisfactory explanation. Owing to their persistent connection with living lung-substance, the flow of blood within the vessels in question suffers no diminution; but, inasmuch as, in the segment of their wall which is sunk in fibrous tissue, absolute rigidity has taken the place of the elasticity natural to the vascular membranes, the blood-pressure is entirely thrown upon the exposed segments, which are ill nourished and weak. The tendency to bulging once established, becomes progressive. If the cavity be small, its limits may be reached by the vascular membrane, and the danger of rupture averted. The cavity then becomes a blood-space, in which opportunities are afforded for coagulation. Obliteration of an aneurysm may also ensue, as originally pointed out by Rokitsky, even after rupture, from the lateral pressure of the blood effused into the cavity, provided the bronchial orifice be temporarily obstructed, or naturally so small as to be unable to allow the blood to escape before the occurrence of coagulation. Most usually the cavities are too large to render possible either of these events, and, after repeated premonitory hæmorrhages, the final burst occurs with irresistible violence.

A description of those vessels which do not traverse the cavity, but are distributed to its walls, must be deferred until my next lecture; and I will pass at once to the consideration of the relations of *bronchi* to *vomicæ*. As long as the excavation is lobular, the conditions are simple, the vomica is terminal to a single bronchiole. This lobular stage is extremely transient; in most cases, the destruction attacks from the first or in rapid succession several groups of lobules; and the good-sized trabeculated cavity which results intersects the path of numerous air-tubes.

It may be accepted as an invariable rule that all non-cartilaginous bronchi are destroyed at an early date of the excavation; but the same fate sooner or later awaits the larger branches. The difference in the

resisting power of the two sets of bronchi may be plainly seen in cavities which occur in the mammary region near the root of the lung. The larger bronchial divisions which commonly project into these cavities are cleared of all their smaller branches, and in this denuded state they closely resemble the heavier roots of a tree torn up from the soil.

The mechanism of the destruction of the bronchi throws much light on the pathology of the pulmonary changes. Let us follow the course of an ordinary bronchus of the third order passing through an early excavation. The bronchioles formerly distributed to the excavated region, are represented by simple perforations resembling the holes in a surgical drainage-tube. These perforations impair the air-conducting function from the first; but, gradually enlarging at the expense of the bronchial wall, they lead to its ultimate destruction. Inasmuch as the proximal perforation (that nearest the trachea) is swept by a more powerful draught, it is apt to suffer earlier and more severely than the more distal perforations, which are not reached by the air-currents until the latter are attenuated by the first leakage; it follows that a bronchus will give way at a point in the cavity nearest the root of the lung, and this in reality happens with great regularity. The ultimate removal of the remains of the bronchial tube is merely a matter of time. As the cavities acquire extension, the same process is repeated in the case of bronchi of a higher order, until the presence of strong cartilages delays for a while the advance of the ulceration. The stumps of the larger bronchi, when denuded, usually present a terminal perforation, which acts much in the capacity of the eye of a catheter; but here also the ulceration of the orifice of a bronchiole ultimately taps the tube at the level of the cavity-wall.

The foregoing remarks explain the oft-quoted experiment of M. de Castelnau, who, by injecting water through the trachea, demonstrated the multiplicity of the orifices leading into cavities. With the progress of excavation, this multiplicity tends to lessen, every successive ulceration cutting off a fresh bronchial set, until ultimately the cavity may empty itself into the lobar bronchus.

The completeness with which a cavity may be drained greatly depends, not only upon the position, but upon the size of the bronchial orifice. I have often found the aperture of outlet to be out of all proportion with the size of the vomica, fibrosis and contraction having taken place in the peribronchial sheath. Where two or more large bronchial divisions terminate in the same cavity, this contraction is invariably more advanced in the upper branches, which have comparatively little work to perform. In the contraction of the bronchial orifices, we find an explanation for the great difficulty attending the expectoration of the contents of very chronic vomicae. The patients are struggling with mechanical disadvantages which no medical treatment can wholly remove.

The changes which I have described as occurring at the proximal side of cavities proceed likewise, although less rapidly, at its distal aspect. As a late result, the fibrosis extending from the cavity-wall and the cell-proliferation special to the mucous membrane leads to an obliteration of all air-tubes not actively in use; but, during the interval which precedes this occlusion, the distal segments of numerous smaller bronchi take their origin, as it were, from the cavity; any air they may obtain is derived from the vomica, and contaminated with its impurities. This is a fertile source of secondary infection for the surrounding spongy tissue. More favourable in this respect are the cases where the interruption of the bronchial air-current has led to a collapse of the corresponding respiratory district. Excavation, in this instance, put a stop to its own progress by surrounding itself with a zone of compressed tissue more prone to fibrosis than to inflammation.

The contents of vomicae possess for the physician considerable practical interest. In the early stages of cavities, it is usual to find within them structural traces of the tissues whose removal has led to excavation, sodden epithelia, fragments of fibrous and elastic tissue, and the products of simple and of tubercular inflammation, mixed with a large proportion of mucus, all in various degrees of degeneration. If the softening be of the moist type, these *débris* will be shed into the vomica very gradually, and it will be difficult to find either in the sputum, or, after death, in the cavern, any recognisable masses of pulmonary texture. Louis, however, has recorded the discovery within a vomica of a detached piece of spongy tissue; and, more recently, in a case of pulmonary gummata, masses of pulmonary tissue invaded by syphilitic cell-growth were repeatedly recognised in the sputum by Cube. But in caseous pneumonia and in gangrene, the rule is for sphaceli to become detached and expectorated. The heavy blocks which occasionally are loosened in caseous pneumonia from the undermined cavity-wall, constitute a tolerably common *post mortem* object. During life, their size may prevent their removal through the bronchi, but they are placed

in a favourable position for rapid dismemberment, and their smaller fragments are readily expectorated.

More or less suppuration always accompanies the discharge of the products of disintegration, and persists for a time in the fully cleared vomica; but, sooner or later, the pyogenic energy of the internal surface becomes exhausted. The patches of bronchial membrane, by which mucus was secreted, gradually become absorbed, and secretions, if present, are more watery. Hence it has been correctly observed by Lebert that, the older a cavity, the more fluid are its contents.

In proportion as the walls of the cavity become rigid from fibrosis, and the bronchial orifice less patent, the secretion is with greater difficulty expelled; that the latter should become somewhat offensive, is nothing more than we would expect. More habitually, however, we are astonished to find that septic decomposition is almost entirely absent from the contents of vomicae in phthisis. Perhaps the free currents of air, set up by periodical expectoration, even in cavities which, from the rigidity of their walls, possess but a sluggish respiratory traffic, may have the effect of washing the effete products, and of supplying more oxygen than is favourable to the growth of micro-organisms. As a broad fact, it may be stated that the contents are preserved from putrefaction in proportion to their free communication with the outer air. This, however, should be understood to apply strictly to phthisical excavations. Decomposition within the cavities is a characteristic feature both of bronchiectasis and of gangrene; in both oxygen exerts its influence upon organic matter, of complex chemical constitution, in the presence of abundant moisture. In gangrene, the decomposition may receive a natural termination in the removal of the offending matter. In bronchiectasis, on the other hand, fresh pabulum is perpetually added to the decomposing mass, which largely consists of mucin—a substance, in its moist form, unusually prone to decay. Hence the mucous fermentation and the acid reaction often present in bronchiectatic sputa, which in this respect contrast with the ammoniacal decomposition special to gangrene. Phthisical cavities are almost as free from the one as from the other form of decomposition. Mucus is only secreted in minimal quantities by the surviving shreds of mucous membrane, and true gangrenous action is entirely absent. Chemical metamorphosis has taken place in the comparatively dry state, at the inner surface of the capsule; and its products, both morphologically and in respect of their chemical constitution, have reached so low a standard as to afford little room for further degeneration. However, the innocuous character of the contents of the cavity has its limits. The secretions, in chronic phthisis, are apt to become offensive in a measure as their expectoration is rendered more difficult; and, after death, the cavities may be found to contain horribly foetid fluid. This condition I have often noticed in the larger vomicae of fibro-tubercular lungs, where the walls become rigid, and the bronchial orifice is considerably reduced in size. Whilst fully admitting the influence of stagnation in promoting septic changes in the secretions of phthisis, we must recognise that septic action does not appear spontaneously to originate within the cavities, but is usually dependent upon some infection introduced from without.

ENDOWMENT OF RESEARCH.—It is announced that the Lords of the Treasury propose to allot a sum of £4,000 in continuation of the endowment of research. The £1,000 fund, heretofore known as the "Government Grant," will cease to exist in a separate form. Applications for grants are to be sent in by the last day of the present month.

THE POLLUTION OF THE THAMES.—A meeting of the committee for the Protection of the Lower Thames from Sewage was recently held in the Cannon Street Hotel. Professor Thorold Rogers, M.P., presided. The chairman mentioned, as an evidence of the growing magnitude of the evil, that the daily discharge of sewage from the Metropolitan area was 180,000,000 gallons.—Mr. Bennock, director of the London Steam-boat Company, said this meant the precipitation of about 300,000 tons of solid matter yearly in the bed and on the banks of the river. He moved:—"That in the opinion of this committee, a Royal Commission should be appointed, or a Parliamentary inquiry instituted, for investigation of the present state of pollution of the River Thames from sewage, and the means to be taken to remedy this alarming and growing evil." Mr. Hinton, Master of the Waterman's Company, who seconded the resolution, described the prejudicial effects which the state of the river had on the watermen; and Captain Gillet, of the *Warspite*, who supported the resolution, described the Thames as a common sewer. The resolution was unanimously passed. On the motion of Lord H. Lennox, it was resolved to appoint an executive committee to take the necessary steps to give effect to the first resolution, and to adopt such other measures as they might think desirable in furtherance of the object in view. The executive committee was then appointed including Lord Henry Lennox, M.P., and Viscount Lewisham, M.P.