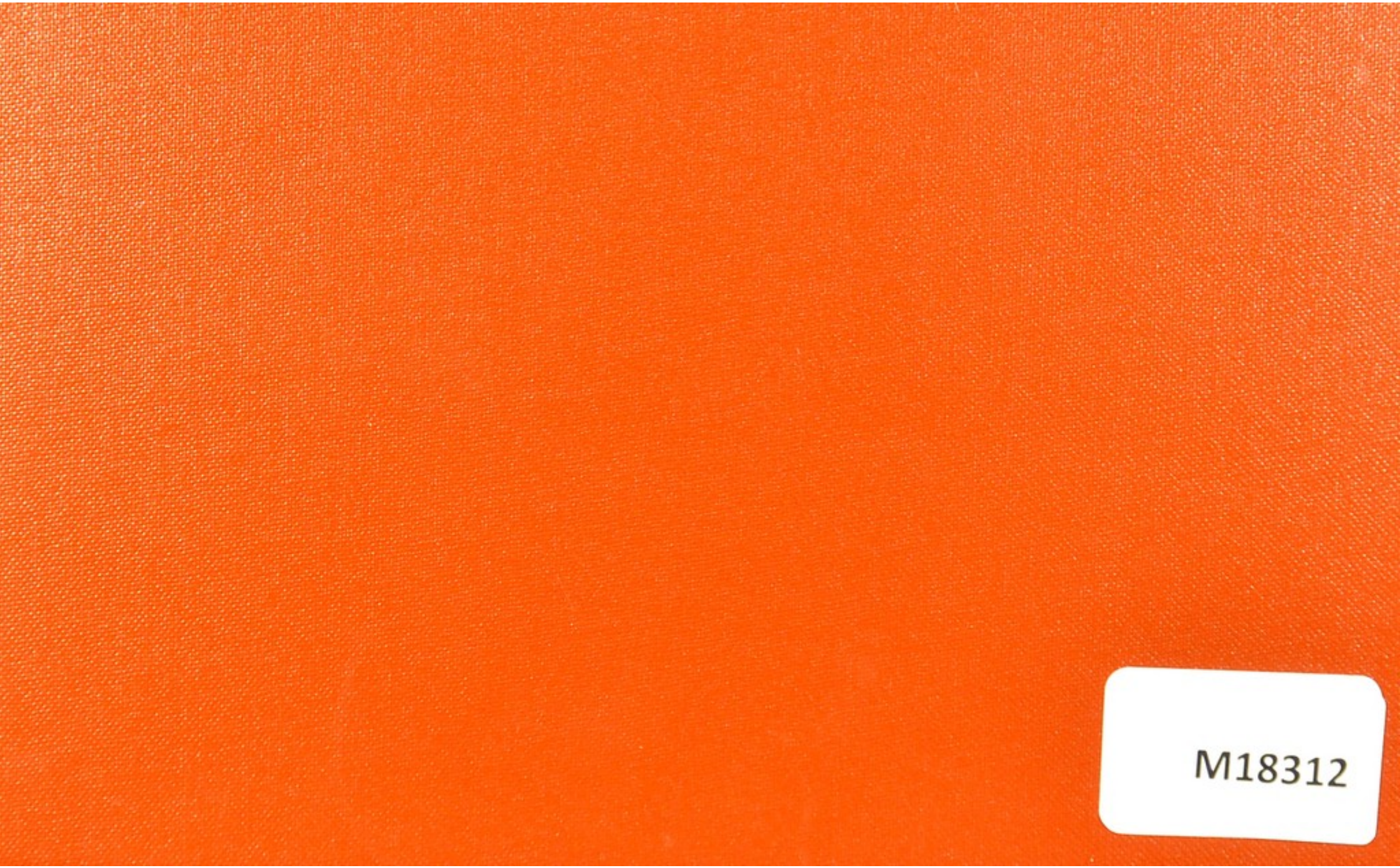
On the principal varieties of pulmonary consumption with practical comments on diagnosis, prognosis and treatment / by R. Douglas Powell.


M18312

ON THE PRINCIPAL VARIETIES

OF

PULMONARY CONSUMPTION.

ON

THE PRINCIPAL VARIETIES

OF

PULMONARY CONSUMPTION

WITH

PRACTICAL COMMENTS

ON

DIAGNOSIS, PROGNOSIS AND TREATMENT

BY

R. DOUGLAS POWELL, M.D. LOND.

MEMBER OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON;
SENIOR ASSISTANT PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST AT BROMPTON;
LECTURER ON MATERIA MEDICA AT THE CHARING CROSS SCHOOL OF MEDICINE AND ASSISTANT PHYSICIAN TO THE HOSPITAL.

LONDON:
H. K. LEWIS, 136 GOWER STREET,
1872.

LONDON:
H. K. LEWIS, PRINTER, GOWER STREET, W.C.

PREFACE.

The following sketch of the chief varieties of Phthisis pulmonalis, in which the connection between the morbid anatomy and the clinical symptoms is attempted to be delineated, was originally published in the form of a series of papers in the Medical Times and Gazette with the hope that it might prove useful to the student by helping him to recognise in the sick room the results of those lesions with which he is becoming daily more familiar in the deadhouse, and to the much engaged practitioner by serving to furnish some headings under which to arrange the details of his increasing experience.

R. DOUGLAS POWELL.

15 Henrietta Street,
Cavendish Square:
April, 1872.

CONTENTS.

ON THE

VARIETIES OF PULMONARY PHTHISIS.

CHAPTER I.

INTRODUCTORY REMARKS.

Much as has been written upon the subject of Pulmonary Phthisis in all its aspects, it cannot be denied that there are still many questions concerning it upon which professional opinion remains undecided, and many of which are of the greatest importance. Let me mention some of them. How many kinds of Phthisis are there? Is there sufficient difference in prognosis between the several kinds to make it worth while to distinguish them? Are not all merely different degrees of the same disease or the same disease localised in different parts of the lung by different local causes? Does an attack of blood-spitting mean that the patient is already phthisical, or only that he is liable to become so as a result of the hæmoptysis? Is diarrhœa or laryngitis complicating a case of chronic lung disease a sure sign of the supervention of tuberculosis or is it not? Finally, and this question underlies all the others—it has excited more disputes than any of them—what constitutes Pulmonary Phthisis? Is it a perishable blood exudation into the lung, a blood disease, with, like many supposed blood diseases,

a favourite seat for its anatomical manifestations? Or, is it the offspring of a mere catarrh affecting an organ so complexly loculated as to entangle and retain its own catarrhal products, so delicately sensitive as to become irritated to the point of ulceration by their presence, and, still further, so richly endowed with absorbents as to readily take up and disseminate throughout its tissues, even to distant parts, the poisonous products of their decay?

Again, what part has the *constitution* to play in all this? We see a man stricken down with acute pneumonia, yet, let the disease be ever so severe, the lung ever so completely stuffed with exudation, we know that he will most probably, with no other treatment than judicious nursing, make a complete and perfect recovery. Another patient will be so trivially ill that he will scarce keep his room, and only on careful examination is it discovered that the lower part of one lung is solidified, yet this consolidation is far less likely ever to completely clear up, his lung may never be again sound. It may be that *constitution* is at the bottom of this difference, but it is I think more than doubtful whether it is the sole modifying cause. On the other hand, the difference of anatomical structure in the two cases is alone sufficiently apparent to account for the different results. In one, the first, we have a mere croupous exudation of fibrin and leucocytes, which can liquefy, resolve, and be absorbed with great facility as soon as the acute disease has passed. In the other, we have the air cells blocked with dense

walled catarrhal cells, liquefaction and absorption of which is comparatively difficult, with but little accompanying fibrinous exudation to melt away into a molecular liquid and leave them free to escape. Let us glance at the catarrhal products distending any mucous follicle, *e.g.*, in the tonsils, or at the catarrhal collections occasionally found in the cœcal appendix, or that sometimes remain stuffing a bronchus for years; they are rarely absorbed, but if not expelled inspissate and shrink, remaining as sequestered masses, perhaps at some later period to set up irritation, and escape by ulceration. But granted that the difference between the two kinds of pneumonia above referred to is a structural one, is this structural difference but the anatomical expression of diverse constitutional states subjected to the same inflammatory test, or are the diseases in the two cases distinct and interchangeable? We practically recognise the one as an acute sthenic, the other as a cachectic, pneumonia.

It is perhaps because consumption is still too much shunned, or watched with too little patience, at our large clinical hospitals, as being a disease of long duration and of ultimately fatal issue, in which the diagnosis once made, the prognosis is a matter of common inference, that the student in his after career, finds himself gradually arriving at a state of bewilderment, increasing with his growing experience, concerning the future of those who present themselves before him suffering from some one of its many forms. For one case comes before him with an

amount of disease so slight that he cannot feel certain that any at all exists; the chest is only delicate, but the patient dies in a few months. He is shocked in another case to find a large cavity at one apex and his prognosis is very grave, yet the patient lives on in the enjoyment of fair health for years.

If, therefore, the few cases hereafter to be narrated illustrating some of the principal forms of Pulmonary Phthisis now recognised, with such practical comments on diagnosis, prognosis, and treatment as may be suggested by them, should tend in any degree, by bringing forward in an applied form the doctrines of the present day, to increase the precision and definiteness of the general knowledge of this always-prevalent disease, the object of this little work will have been entirely achieved.

CHAPTER II.

PATHOLOGY.

Two kinds of morbid processes—Inflammation: affecting the parenchyma, Catarrhal Pneumonia in three degrees of intensity; affecting the fibrous stroma, Pulmonary Fibrosis—Tubercle, nature and seat of, development—Hereditary predisposition to Tuberculosis and Consumption—Mechanical effects of the respiratory movements upon the Lungs and Pleura: pleural adhesions, mode of production, significance of friction—Thickening of pleura, how produced—Bronchiectasis.

It is not proposed to enter with any historical or descriptive minuteness upon so wide a subject as the morbid anatomy of the forms of lung disease included under the common term Phthisis; but it would be scarcely possible to carry out the objects in view without first giving a brief sketch of those pathological changes upon which the clinical features of the different varieties of phthisis are based. It must here, too, be observed that, though for the moment the morbid processes going on in the lungs will be alone considered, yet this is in no disregard to the importance of constitutional changes of which we can take no anatomical note, nor of degenerations, or active complications of other organs, which are comparatively simple anatomically, and about which there has consequently been less confusion of terminology and vagueness of thought. To all these conditions reference can best be made incidentally in the dis-

cussion of the points specially illustrative of individual cases.

On inspecting the lungs of one who has died of phthisis, we meet with very great variety of appearances, which may, nevertheless, be recognised as the results of a comparatively few morbid processes; we see consolidation of the lung in every stage of formation, decay, and removal; and, glancing at the emaciated form before us, we have a very practical definition of Phthisis Pulmonalis—progressive consolidation and decay of the lung with progressive wasting of the body.

As to the exact nature of the morbid processes which lead to this destruction of lung and waste of body, there are numerous and diverse opinions. These processes may, however, be said to be of two kinds. 1. Inflammation affecting with different degrees of intensity the different tissues of the lung, and running an acute, chronic, or chequered course. 2. A new growth—Tubercle—with its characteristic granulations disseminated through the lungs, or collected into nodular groups, or mingled with inflammatory changes, developing into fibroid tissue, or abruptly undergoing fatty change. We may meet with either of these processes in the acute or chronic form without any admixture of the other, but it is comparatively rare to meet with chronic tubercle unmixed with inflammatory changes.

It may be here recollected that fatty degeneration is the necessary consequence of inflammation, and is the means by which the products of inflammation

become removable by absorption or expectoration, or sequestered for a time by caseation. The tendency of all new growth is also sooner or later to fatty decay, and in Tubercle we find no exception to this rule.

There is yet a third set of processes of a physical kind, but having pathological results, which must be taken into account as leading to very important modifications in the signs of disease during life, and the appearances *post-mortem*—viz., 3. The respiratory movements of the chest walls, with the mechanical effects which they produce upon the lungs and pleuræ when modified in their physical conditions by disease.

The inflammatory process takes the largest and most important share in the production of the various appearances met with in phthisical lungs: it is the destroying element in this disease, as long ago pointed out by Addison.[1] It may therefore be appropriately spoken of first.

With that form of inflammation of the lung—acute sthenic pneumonia—agreeing in many of its characters with an acute specific disease (*e.g.*, idiopathic erysipelas), we have but little to do in dealing with cases of phthisis; we only meet with it as an exceptional complication. Doubtless, the subject of acute basic pneumonia already cachectic, or rendered so by neglect during the disease, may become phthisical; some cases of basic phthisis have this origin. But the pneumonia

[1] Collected writings published by Sydenham Society, p. 56.

which is the most constant element of true phthisis, and which will indeed as already hinted be found the main factor in the more chronic and cachectic forms of basic lung disease, is of a very different kind; its onset is usually insidious, and its origin appears to be generally by extension of a catarrhal process from the finer bronchial tubes to the interior of the alveoli: hence its name—*catarrhal* or *broncho*-pneumonia. It is identical pathologically with the lobular pneumonia with which we are familiar in hooping-cough.[2] This form of pneumonia is essentially lobular, though the coalescence of many adjacent lobules may cause the consolidation of a whole lobe. The alveoli are affected by this inflammation with all degrees of intensity, from mere superficial catarrh causing slight epithelial desquamation to the most deeply destructive involvement of their walls.

[2] Professor Niemeyer states, on the authority of Bartels and Ziemssen, that pulmonary collapse always precedes catarrhal or broncho-pneumonia. This is a question of little importance to the present subject; but I cannot think that such is the case with the most typical forms of broncho-pneumonia. It certainly very often occurs in hooping-cough that inflammation succeeds to collapse, less frequently so in measles; but induration and agglutination of the air cells is the result—not their occupation by the large catarrhal cells characteristic of true broncho-pneumonia. Professor Niemeyer's description of broncho-pneumona, as presenting, on cutting through the consolidation, a smooth surface, makes it appear that he must have drawn his description from collapsed lobules, which have become subsequently inflamed. In my experience, certainly, lobules of catarrhal pneumonia are strikingly granular on section.

In the simplest alveolar catarrh the cellular products may escape with the expectoration, leaving the alveolar wall undamaged. In the next degree of intensity, the alveoli and minute bronchi become blocked with the large granular cells, which are produced in great abundance. These cells, thus stuffing the alveoli, almost immediately begin to undergo fatty degeneration—the process by which resolution is naturally effected. They may liquefy, and be partially absorbed, partially expectorated; but the alveolar walls have been damaged, and permanent local collapse remains behind from their agglutination. This is the natural cure of the disease in this degree.

In some cases the cellular products, after having undergone complete fatty degeneration, become inspissated by absorption of fluid matter, and remain for a long time—perhaps for the lifetime of the patient—in the cheesy condition, or subsequently become cretaceous. This may be called natural arrest by obsolescence, and these cheesy masses are commonly looked upon as "old tubercle."

In the still more intense degree of the process—catarrhal pneumonia—now under consideration, the alveolar wall is deeply involved in the inflammation, so that it subsequently undergoes fatty degeneration together with its cellular contents to a depth which varies with the intensity of the process, and breaks down in the subsequent liquefaction, gradually or rapidly, according to circumstances.

The elastic tissue of the lung takes no active part in any of its inflammatory processes; it escapes but

little altered when the alveoli break down, and thus, on being recognised in the sputa, affords certain evidence of pulmonary destruction. It will, of course, be understood that there is no real line of demarcation between the degrees of severity above described separately. The intensity of the first attack may at once determine the depth of injury, or the lighter may gradually pass into the graver degree.

But, in addition to the parenchyma[3] proper of the lung, which, with its epithelium, is the special seat of catarrhal pneumonia, there is the fibrous stroma, if one may so style it, formed by the interlobular areolar tissue, supplying sheaths to the vessels and bronchi, contributing also to the formation of the alveoli, and intimately connected at the surface of the lung with the investing pleura. It could not be expected that an inflammation of the lung of any great severity would leave this widely spread tissue untouched; and it might also be anticipated, on reflection, that a tissue thus, comparatively speaking, deeply placed would, as a very general rule, only be affected secondarily to disease of the parenchyma or pleura. From this interstitial tissue are derived the tough, fibrous, pus-secreting walls of cavities, and the trabeculæ which for a long time resist the most severely destructive processes. The inflammatory process in this tissue is, as a rule, a much more deliberate one; even when in a state of active ulcera-

3 The term "parenchyma" is conveniently, and perhaps correctly, restricted to the minutest bronchi and the alveoli into which they are expanded.

tion, as in the walls of some cavities, the destruction is molecular, sphacelus is rare. Inflammatory reaction in this tissue, indeed, more generally partakes of the character of growth under irritation, producing a more or less general condition of *fibrosis* of the lung. Decay, however, the inevitable result of inflammation, finally sets in; the fibrous tissue, at first merely hypertrophous, loses its characters as such; its nuclei, at first very abundant, gradually fade; its fibres fuse into tough homogeneous bands, and in their turn become granular and fatty and finally crumble away.

The various primary diseases—broncho-pneumonia, croupous pneumonia, chronic tubercle, pleurisy, etc.—upon which pulmonary fibrosis supervenes are thereby marked by clinical features of great interest and of significance for prognosis. But sometimes the fibrosis is so extensive as to become, whatever its origin may have been, the essential disease. Such cases have been very conveniently classified separately by Dr. Andrew Clark under the term "fibroid phthisis." He regards the disease as sometimes of idiopathic origin, or—what amounts to nearly the same thing—as a disease which progressively invades and destroys the lung from some one point of origin, as a local pleurisy or bronchitis. I must confess that in my much smaller experience I have not yet met with a case in which the fibrosis has appeared to me to have been either idiopathic or to have thus extended widely beyond the primary disease without the supervention of another disease, such as tubercle, or a repetition of

the primary malady. I will refer to this again, however, later.

It would be exceedingly profitless to go historically into the question as to the essential nature and mode of origin of tubercle.

Sufficient evidence has now accumulated to render it tolerably certain that tubercle is a morbid growth of the lymphatic gland class, and that it may be a mere hyperplasia of the gland tissue normally minutely disseminated through the organs of the body (Sanderson). The characteristic form of tubercle is the grey granulation which has its seat in the alveolar wall, or in the connective tissue sheathing vessels, or under the mucous membrane of the bronchial tubes. It is vascular, and under favourable circumstances undergoes development into a peculiar form of fibroid tissue, at first very recognisable from ordinary hyperplastic fibrous tissue, but which subsequently becomes converted into bands or tracts of uniform homogeneous texture, and finally fattily degenerates and is removed.

This development of tubercle, before its final decay, has hardly been sufficiently insisted upon as an essential character always observable if circumstances permit the attainment of the necessary stage. It is, however, in strict accordance with the lymphatic gland type of this morbid growth, and it is of some importance as affecting the clinical characters of chronic tuberculosis. In acute tuberculosis, the patient does not often live long enough for any process of the kind to take place. In chronic pulmonary tu-

berculisation, however, and when tubercle attacks a lung rendered quiescent by previous disease, the stages of development of tubercle into fibroid tissue may be seen.

Having said this, however, it is necessary to remark that many distinguished pathologists in this country and abroad refuse to admit the lymph-gland-like structure of tubercle, affirming that the true fibrillated stroma, with stellate cells in the angles of its network, always seen in the follicles of a normal lymphatic gland, is not to be recognised in tubercle. The stroma of tubercle differs from that of a gland follicle, according to these observers, in that it is made up of homogeneous fibrils shewing no trace of cellular development, and, it is hinted, that this stroma in tubercle is altogether a product of the hardening process to which the specimen has been submitted and is not to be recognised at all in fresh sections. It must of course, however, be remembered that the structure of tubercle is regarded as analogous to that of lymph-gland tissue indurated and enlarged by irritation—under which circumstances the cells become densely packed, and the gland stroma loses its cellular structure, and, finally, assumes the tougher homogeneous form undistinguishable from that of tubercle. I cannot help myself being firmly persuaded that this view of the lymphatic nature of tubercle is far more in accord with the results of careful microscopic observation than that which would class it amongst the ordinary products of inflammation.

As regards the origin of tubercle, opinions are extremely various and, indeed, irreconcilable; but the tendency of modern research—the experiments on inoculation in animals, and the very powerful advocacy of the late Professor Niemeyer—is certainly to show that tubercle is much more commonly a secondary disease than has until lately been suspected—that people are, in fact, only very exceptionally, if ever, born to die of tuberculosis. A due appreciation of this doctrine, so different from that even now accepted by many, is of almost national importance in giving encouragement to those hygienic and other measures of prevention, the neglect of which has too often been sanctioned by a foregone conclusion. It would, I think, however, be extremely injudicious to deny hereditary predisposition to tubercle altogether. Moreover, when we come to the question of hereditary predisposition to those forms of consumption which originate in catarrhal pneumonia, it is freely admitted that the offspring of consumptive parents have a tendency to this form of pulmonary phthisis, that the scrofulous have a like tendency [Niemeyer], and that scrofulosis is sometimes hereditary.[4] Again, from the tendency to the occurrence of chronic inflammation especially in glands leaving behind cheesy deposits, by which the scrofulous diathesis is characterised, it is regarded as the constitutional state in which true tuberculosis is most likely to

4 Waldenburg, *Die Tuberculose*, etc., p. 524.

occur. My own observation would not enable me to agree in this latter view.

These statements necessitate a considerable addition to the list of those who are hereditarily liable to tubercle in the old-fashioned sense of the term. But it must be remarked that catarrhal pneumonia and scrofulosis can be more efficiently guarded against, by attention to climate, soil, etc., and more successfully treated, than truly tubercular disease. My colleague, Dr. C. Theodore Williams, the latest authority on the question of hereditary predisposition to consumption (in its broad sense), in a paper read before the Medical and Chirurgical Society in January, 1871, gave 48 per cent. as the proportion in which, out of 1000 carefully noted cases, family predisposition could be traced, using the term "family" to include brothers and sisters and first cousins. It thus appears that, even making every allowance for alterations in terms and views, fewer people die of hereditary consumption now than formerly. It may be, of course, that traditional opinion has simply been erroneous in regarding consumption as so strongly hereditary, but it is perhaps nearer the truth to say that under the influence of superior hygienic circumstances, since Sanitary Science has been so much popularised, hereditary predisposition, as strong and real as ever, gets fewer opportunities of being nursed by neglectful hygiene into confirmed disease.

To return from this digression—perhaps a pardon-

able one, considering the importance of the subject —there are yet a few words to be said respecting the mechanical effects of the respiratory movements of the chest walls upon the lungs and their investing pleura.

In health, ordinary inspiration is a muscular act, by which the elasticity of the lungs is overcome, and their expansion is effected to a certain degree. Ordinary expiration, on the other hand, is the elastic recoil of the lungs bearing with it the chest parietes to a point beyond that to which their natural resilience would bring them. There is, consequently, a resilient force in reserve, which goes to help muscular action in the first part of inspiration. It is easy to see how greatly this arrangement adds to the smoothness of the mechanism of respiration, the commencement of each act of which is normally almost wholly accomplished by elastic power. A glance at one diseased condition—emphysema—in which the elasticity of the lungs is impaired so that they do not contract to the normal extent in expiration, awakens our attention to the importance of what may at first sight appear a trivial matter: the thoracic parietes in emphysema are not drawn in beyond their position of rest—*i.e.*, that position which they would naturally attain on a free opening being made into the pleural cavity. The abrupt and jerking manner in which inspiration commences in cases of emphysema, being effected by a conscious, albeit an habitual, effort on the part of the sufferer, cannot be regarded without offence to one's instinctive sense of

mechanical perfection. But in cases of Phthisis we have to do with local, rather than general, alterations in the pulmonary texture; diminution, rather than enlargement, of the space occupied by the lungs; increased local resistance to expansion, rather than diminished tendency to recoil; and the parts within the chest which are most affected by these causes are the pleura and the bronchial tubes.

Why is it that pleural adhesions are so common in chronic lung diseases, and especially in phthisis? The answer usually given is—Firstly, that inflammation of the lung is very apt to extend to the pleura; secondly, that tubercle is very prone to attack serous membranes, and tubercular pleurisy is conspicuously a dry adhesive pleurisy. Both these statements are doubtless true, so far as they go; but they are by no means sufficient to explain the frequent and inevitable pleurisies and local adhesions of Phthisis, and notably of those varieties of phthisis about which there is most dispute as to their having anything to do with tubercle.

Confining our attention now to cases of Phthisis, though these observations are really applicable to other chronic pulmonary diseases, there are a few points worthy of remark concerning pleural adhesions.

1. They are pretty accurately limited to those portions of pleura corresponding with diseased lung beneath.

2. The more contractile the lung disease, and the

tougher its texture, the thicker the pleura covering it.

3. *Post-mortem* there is frequently to be found no tubercle at all in the adhesions, and still more frequently no tubercular granulations in the pleura immediately in the neighbourhood of the adhesions.

4. In cases of very chronic phthisis (not secondary to pleurisy), with contraction and induration of part of one lung, we find *post-mortem* opposite the oldest part—*e.g.*, the apex—the two pleural layers perhaps intimately fused together, forming a white fibrous covering half or three-quarters of an inch in thickness. Lower down, however, we find the layers, each somewhat thickened, separated by a striated jelly-like material—œdematous connective tissue—the fine striæ of which pass vertically from one pleural surface to the other.

The real explanation of the recurring pleuritic pains and adhesions in cases of phthisis is, that when a portion of lung becomes damaged in texture by disease it ceases to follow accurately the expansile movements of the chest-wall; a certain gliding or rubbing motion takes place between the two normally corresponding pleural layers at this point; friction, local pleuritis, and adhesion result. We can readily understand, therefore, how it is that a friction sound is often the first evidence we get of local pulmonary disease, and that a new friction sound means most generally more than mere dry pleurisy; it means, in fact, an accession of lung disease.

When the lung disease is of a very chronic indurative, contractile character, as in the cases referred to above in section 4, the effect of the continued inspiratory efforts to expand the toughened lung is to stretch out the adhesions and to separate the pleural layers to a certain extent; the further contraction of the lung continues the process, so that the parietal and visceral pleuræ become separated by a considerable interval of half or three-quarters of an inch. This space is at first filled by serous fluid effused into the meshes of the areolar tissue of the stretched adhesions. We thus get the œdematous pleura. At a subsequent stage, however, of the disease, by the continued growth of the areolar tissue, the whole space becomes occupied by tough fibrous tissue, and the two layers become completely welded together into one uniform fibrous thickness. That this is the real history of the enormous thickening of the pleura in many cases of chronic phthisis I have satisfied myself by repeated observation.[5]

It has seemed to me that thickening of the pleura has been regarded too much in the light of a very dangerous pathological process, liable to extend into and by its contractile power to squeeze out of existence, so to speak, the proper lung tissue, whereas it will be found on careful examination to be most generally a condition secondary and quite subsidiary to the lung disease.

In primary pleuritis the thickened pleura is pro-

5 Vide *Path. Trans.*, vol. xx., pp. 59-61.

duced in a different way. After absorption of the fluid a certain thickness of lymph often remains between the two layers of the pleura, into which the granulations from each surface penetrate, and finally unite, completing the adhesion. There are many cases of phthisis of the pneumonic kind of tolerably acute progress, and attended with little contraction, in which, though the pleural surfaces are inflamed and covered with finely granular lymph, they do not become united. It is in these cases that pneumothorax is especially likely to occur.

Bronchial dilatation (bronchiectasis) is another important morbid condition with which the chest movements have something to do. The main causes of bronchiectasis may be summed up as follows:— 1. Damage to the texture of the bronchial tube— atrophy of mucous membrane and thickening of fibrous coat (Biermer).[6] 2. Increased air-pressure during cough, acting principally at those portions of the lungs where there is least support, notably the apices. 3. The expansion of the chest wall, or rather the struggle to expand it, failing to affect the air cells which are obliterated by disease, acting indirectly upon the bronchial tubes. 4. The diseased lung in its contractile forms, contracting in various directions tending to widen the imbedded bronchial tubes.

The constant movement of the lungs no doubt goes far to modify and hasten the progress of morbid pro-

6 *Krankheiten der Bronchien und des Lungen-Parenchyms: Handbuch der speciallen Pathologie.* Virchow.

cesses going on within them. This must have occurred to the minds of many Physicians, and even more forcibly to Surgeons, who have to deal with wounds of the lung. We certainly often see in the lung compressed by fluid or air, a complete arrest of the phthisical process which had been previously active there. At the same time it must be remembered there is also in such cases complete and often permanent arrest of the function of that lung. Had we complete command over the duration of any effusion into the pleura we might artificially induce, the possibility of employing such a line of treatment might be entertained. At present the question is worthy of being raised whether in the acute stage of one-sided apex disease any method for restraining the movements of that apex, might be adopted, as *e.g.*, by binding the arm to the side. I have myself no experience to guide me to an opinion. The constant admission of air to the softening tissues of the lung is also a condition impossible to remove, but which must be borne in mind in regarding the peculiarly destructive character of the inflammatory processes affecting it.

I have now passed in brief, and I fear very imperfect review some of the main points in the pathology of phthisis which have a direct clinical bearing, and a due appreciation of which will, I think, materially assist in the correct reading of the cases as they appear before us in the subsequent chapters.

CHAPTER III.

ALVEOLAR CATARRH AND CATARRHAL PNEUMONIC PHTHISIS.

Alveolar catarrh may advance to Catarrhal-Pneumonic, or Tuberculo-Pneumonic, or Tuberculo-Fibroid Phthisis: the first stage of phthisis most amenable to treatment—Diagnosis—Illustrative case of Catarrhal-pneumonic Phthisis in an early stage—Comments on etiology, prognosis, and treatment.

It would hardly be instructive to give an illustrative case of Alveolar Catarrh in the first and slightest degree, that stage which forms the connecting link between the prodromal catarrh of Niemeyer and catarrhal pneumonia. This condition is, however, an extremely common one, and may be very readily overlooked, for the signs by which it is recognised are only faintly marked. It must be considered as really the first stage of phthisis—through which, at least, all cases of catarrhal-pneumonic phthisis, and therefore the majority of cases of pulmonary consumption, pass.

The pathology of this disease consists, as has been before intimated, in the proliferation of the epithelium of the air cells by a catarrhal process of the most superficial kind affecting them. Tubercle has nothing whatever to do with this process, which may, indeed, pass on to catarrhal pneumonia and destruction of the lung without tubercle taking any conspicuous part in it. But an irritative overgrowth of the minutely dispersed adenoid or gland

tissue of the lung, which has been before referred to, may be set up secondarily by the catarrh, and then the disease in its further stages assumes the characters of that described by Addison as tuberculo-pneumonic phthisis. Further, the adenoid growth, accompanied, as it always is, by more or less increase of fibrous tissue, may, having once been set going by the irritation of a simple catarrh, take so prominent a part in the future progress of the disease as to eclipse altogether the catarrhal process, and we get a somewhat rare, insidiously progressive and very destructive disease, which, I think, has not been fully described in its entirety, but which closely corresponds anatomically with the iron-grey induration of Addison, and invades the lung by substitution of a fibroid tissue, inch by inch, from apex to base.

These may be said to be the three directions in which alveolar catarrh when it runs an unfavourable course may lead, and its tendency to develope into such formidable morbid processes is a sufficient reason for its being naturally regarded as the first stage of phthisis. Hereditary predisposition, existing cachexia, and the special nature of the influences which have produced the disease play an important part in determining its future characters—*i.e.*, whether it shall be pneumonic, tuberculo-pneumonic, or tuberculo-fibroid; also, whether its course shall be rapid, or insidious, or intermittent. But I must hasten to observe that if alveolar catarrh be early recognised and rationally treated its progress may be

in a large number of cases entirely checked, and the more adverse the circumstances which have led to the development of the disease, the more hope is there, on their removal, of convalescence.

How, then, can this condition be recognised in its earliest stage. Professor Niemeyer, who has more than any other author urged the importance of its early detection and treatment, regards the presence of pyrexia as the one symptom above all others indicating the extension of the catarrh to the alveoli, and he attaches some weight to streaky hæmoptysis as a sign of the alveoli having become involved in the catarrhal process. The significance of these symptoms when present is beyond question; indeed, I think, when there is elevation of temperature we always have some pneumonia present, and have therefore arrived at a stage beyond mere catarrh.

The patient who is the subject of alveolar catarrh has always been depressed in health, through tardy convalescence from some other disease, or bad living, or mental anxiety; he has had a persistent, though it may be, a slight cough, for a longer or shorter time, and has during that time been getting thinner. At this stage the physical signs are very slight, but sufficient for diagnosis in conjunction with the symptoms and history. There is no dulness or impaired movement, but the respiration is weaker at one apex, the inspiration being wavy, or even jerking. There are usually a few sonorous râles present, which, if limited to that apex, are very significant; and, in addition, one hears at the extreme summit of the lung (supra-clavicular or

supra-spinous region) a peculiar crumpling sound at the moment of cough, which differs both in time and degree from the crepitant sound audible at a somewhat later stage with the first inspiration following a cough.

Not to refine too much, though we have to deal with very slight physical signs—which, the slighter they are the more important are they to be recognised—we may say that physical signs of bronchial catarrh limited to one apex, and associated with a decided imperfection of the respiratory murmur at that apex, afford, when taken in conjunction with the symptoms —more particularly emaciation—unmistakable evidence of incipient phthisical disease, upon which we must advise most decidedly if we do not wish to see the patient pass beyond our control so far as positive cure is concerned.

There is no clinical line of demarcation to be drawn between this condition and the prodromal catarrh which precedes it, or the catarrhal pneumonia into which it is apt to pass; they shade imperceptibly into one another. I only specially refer to this stage as the earliest recognisable stage of phthisis. I now pass on to give a case illustrative of catarrhal pneumonia in an early stage, which also bears out some of the above remarks respecting the most common etiology of this form of consumption.

S. S., aged 29, a married woman engaged in domestic duties and suckling a child aged 7 weeks, came under my notice as an out-patient in April 1871. Her mother had died of consumption within two years

of the patient's birth, and an elder sister had been affected with the disease in an early stage. She had enjoyed fair health until her first confinement, when she was with difficulty delivered of twins, only one of whom survived the birth, which was stated to have been a "cross one." This child she suckled for eleven months, when she again became pregnant with her present child. Ever since her last confinement she had suffered with increasing debility, emaciation, and cough, and shortly before that time she had had slight hæmoptysis.

She was a tall, thin, anæmic woman, with the worn look so characteristic of over-lactation or rapid child-bearing; her large, heavy, pendulous breasts, marbled with large veins, increased by contrast the apparent general flatness and narrowed antero-posterior diameter of the chest. There was no local flattening however at either apex, and the respiratory movements though generally deficient were not more so at one apex than at the other. On percussion over the summit of the left lung the resonance was somewhat less than on the opposite side; the respiratory sounds there were harsh, and accompanied by some moist crepitation, which extended to the second rib. The respiratory murmur elsewhere was of fairly good quality, but somewhat feeble. The main symptoms complained of were troublesome cough and yellow expectoration, shortness of breath, general weakness, and giddiness in the head. The pulse was quick and weak, the appetite indifferent, but digestion fairly good.

The case was regarded as one of Catarrhal-pneumonic Phthisis in an early stage, the disease being limited to the left apex, and supervening upon the exhausting effects of more than thirty months' continuance alternately of gestation and lactation. She was directed immediately and completely to wean the child, to take abundance of appropriate food, with a moderate amount of beer. Some counter-irritation was applied at the left apex, and cod-liver oil and steel wine administered, with some morphia and ipecacuanha lozenges for the cough.

On again examining the chest a month later the moist sounds were no longer audible with ordinary respiration, but a few crackles were heard after cough. There was slight flattening at the left apex, which became more obvious on deep inspiration; the respiratory sounds were feeble there, while on the opposite side they were more developed, and on percussion the line of resonance of the right lung extended a little to the left of the mid-sternal line. There was no evidence of a cavity at the left apex, and no extension of the disease below. The health of the woman was, though improved, by no means restored; she was still anæmic and thin. Her cough was troublesome, especially in the morning, and expectoration difficult, the effects of coughing often causing vomiting at that time. She had neglected to completely wean her child, and continued to "give it the breast now and then." The pulse was quiet but weak; the appetite improved. She had been unable to take a stronger preparation of iron ordered

a fortnight previously, and was obliged to fall back upon the steel wine. Three weeks later she had very greatly improved in health and strength; some colour had returned to the cheeks, and she was gaining flesh rapidly.

This case, though not complete, illustrates so many points in the clinical history of catarrhal-pneumonic phthisis[7] in its earlier stages that I have related it in preference to others.

I employ the term "catarrhal-pneumonic phthisis" because the term pneumonic phthisis is often used to signify cases which have commenced as basic pneumonia in which the consolidation has not undergone complete resolution. It may be true that we sometimes meet with genuine croupous pneumonia at one apex. I believe such cases are, however, exceedingly rare. The case now under consideration could not be confounded with such; the dulness was never absolute, the respiration was not decidedly bronchial,[8] nor was the crepitation that of typical pneumonia.

7 This case would be grouped under one of the following headings by the authors named:—Pneumonic phthisis, Addison; catarrhal pneumonia, Niemeyer, Herard and Cornil; epithelial pneumonia, Andrew Clark.

8 The breath-sound which is audible over a portion of lung when the consolidation is not uniform, but in scattered nodules, is variable within certain limits; sometimes it is simply harsh, sometimes bronchial, but with some vesicular murmur superadded. I find the term "broncho-vesicular" a convenient one briefly to describe this sound. It also very well describes the sound frequently heard over a small cicatrising cavity with compensatory vesicular enlargement around it. When the nodules

But in other cases, which do not essentially differ from this save in degree, the dulness may be complete and the crepitation undistinguishable from that of true exudative or croupous pneumonia, the respiration being also tubular, but, I think, never so intensely so as that of basic pneumonia. This of course might be readily accounted for by the seat of the disease; but the subsequent course of such cases is very rarely indeed that of simple pneumonia. They do not undergo complete resolution. The most common course is for the consolidation subsequently to soften and break up into cavities; and on post-mortem inspection of such apex consolidations, we find the alveoli stuffed with large catarrhal cells, instead of being occupied by the fibrinous exudation with entangled corpuscles characteristic of true exudative pneumonia.

We must not, however, draw the line too absolutely between these two forms of pneumonia, for they are certainly not unfrequently mingled together; but it is the catarrhal pneumonia which is the important disease, the natural tendency of which is to break down into cavities, while any croupous pneumonia with which it may be complicated readily undergoes resolution. We thus, from clinical and post-mortem experience, know that an acute apex pneumonia is of a much more serious kind than a basic one—it should be always regarded as probably a phthisical pneumonia, and although we cannot strictly speaking agree

are large enough to be mapped out by percussion, of course the bronchial breath-sound will be correspondingly isolated.

with those observers who say that the inflammation has been determined to that part by the presence of tubercles, yet, clinically and in their acceptation of the term tubercle—as including both the miliary granulation and nodules of catarrhal pneumonia—this view will I think generally hold good.

The main points of the case before us may be summed up as follows:—A woman with a tolerably strong hereditary tendency to consumption (her mother was probably phthisical when she was conceived), enjoys fair health until she is 27, but in the course of the succeeding two years and a half she gives birth to three children in two confinements, two of whom live and are suckled by her. Shortly before the birth of her last child her health breaks down altogether, and she becomes the subject of phthisis. It is to be remarked that there is no history in this case of any exposure to cold.

Now, I think it is fair to assume that, had this patient been in happier circumstances, had her health not been depressed by the development at her expense of three infants and the maintenance of two of them, while she herself was doubtless not in the enjoyment of nutritious food in any great abundance, had she sought advice earlier (and taken it), she would never have become phthisical—her hereditary proneness to consumption might have remained a mere latent tendency. Had there been any family tendency to insanity, it is quite possible that the same evil conditions which have now led her to become phthisical might have caused her to be afflicted with some form

of puerperal mania. "It is true that privation, excess, errors in habits of life, the sedentary occupations, the pernicious influence of certain trades, grief, anxiety, and other wasters of vital power, will not suffice to induce consumption in all, or even in the greater proportion of, individuals; for these agents, so universally prevalent, are part of the daily lot or of the daily errors of many more than fall victims to consumption. But it is also true that if to any or all of these conditions that of inherited tendency to phthisis be superadded, very few indeed escape the disease."[9] The truth of this remark is well borne out by the above, amid numberless other cases which must be familiar to physicians.

The points about the case which rendered the prognosis a favourable one, with certain reservations, were:—1. The obvious and very sufficient determining cause. 2. The limitation of the disease to one apex. 3. The presence of considerable crepitation and some dulness, without any local flattening or marked difference in expansion. 4. The absence of fever at the time of coming under observation.

1. If the circumstances of the patient admitted of complete rest from the cares and anxieties of her position of life, and a change to a purer air, there would scarcely be a doubt as to the prognosis; and although she has had no advantages of this kind, and with the common dread of her class of becoming again pregnant, she could not be persuaded completely to

[9] Dr. Pollock, *Elements of Prognosis in Consumption*, p. 340.

wean her child, she has yet greatly improved; the disease has not extended, the physical signs show drying up of secretion sounds and pulmonary collapse.[10] The encroachment of the margin of the opposite lung, and the gradual appearance subsequently of some flattening, show that the lung beneath is cicatrising. This encroachment of the margin of the opposite lung towards the diseased side should always be anxiously looked for; it can be readily made out by percussion, and when the disease is one-sided, it precedes, often by a long interval, any decided apex flattening.

3. The late appearance of flattening—coincidently, that is to say, with the lessening and disappearance of moist sounds which have been considerable—is an important sign of arrest of the disease, in contradistinction to flattening which comes on coincidentally with an advance in the other physical signs, and which may be due, therefore, to sheer loss of lung substance, or to the presence of the indurative form of disease, chronic pulmonary tuberculisation, which, though of chronic course is yet one of the most intractable of lung affections. Flattening must then only be considered in conjunction with other signs, and with especial regard to the period of its appearance. In the case before us, it signifies together with the other signs pulmonary collapse, with perhaps a few shrunken nodules, adhesion and some thickening of the pleura.

10 Since the above was written this patient has had a second distinct attack which has resulted in further damage to the left lung but from which she has again rallied.

With regard to the management of such cases as this, there is little comment needed. It is unnecessary to point out the great importance of complete removal for a time, at least, from the adverse circumstances which in the first place induced the disease. In the case of women who are suckling, a partial weaning of the child is of very little use; the irritation and vascularity of the breasts is kept up by the occasional sucking of the child, and the nutrition is diverted from its natural objects almost as completely as before. Iron and cod-liver oil are the necessary remedies. One of the best forms of iron to begin with is the ammonio-citrate, to which a little aromatic ammonia is added. The sesquichloride will often not agree at first.

There is one symptom in the above-related case which deserves a special comment: the cough remained troublesome while the pulmonary signs were greatly improving and all secretion sounds rapidly drying up. Dr. Thorowgood[11] has drawn attention to the irritable dry cough which is so frequently attendant upon the subsidence of pulmonary disease, and truly observes that the patients should be encouraged to check the cough themselves as much as possible. This they can do to a great extent, and may be assisted by some sedative cough mixture, if necessary, to secure rest at night. The morning cough in these cases—and, indeed, in many others—is the most troublesome. It is, however, the natural

[11] *The Climatic Treatment of Consumption.*

consequence of a good night's rest, and should never be checked by a sedative, since the retained matters suitable only for expectoration considerably impede respiration, become highly irritating, and much increase the future trouble from cough. A cup of warm cocoa, or tea, or milk, taken before rising, will greatly facilitate expectoration. If this does not suffice, a simple steam inhalation is useful.

CHAPTER IV.

Case of Catarrhal Pneumonia, non-hereditary—Significance of Fever and of the signs of softening; their relation to one another—Gradual change in Physical Signs; Supervention of Pulmonary Fibrosis—A second case briefly referred to—Further progress of such cases; a hint as to their management.

It will, I think, be most convenient to follow up the remarks upon the case described in the last chapter by detailing one or two other cases which will serve to illustrate the transition, which not very unfrequently occurs, of pneumonic into fibroid phthisis. Bearing in mind the relation of the tissues to one another, which are affected in these two forms of phthisical disease, as pointed out in the second chapter, we perceive that the transition of the one form into the other is pathologically very easy; it is also, I am persuaded, clinically often to be observed.

I do not of course mean to contend that all cases of fibroid phthisis commence as catarrhal pneumonia; the question as to the etiology of fibroid phthisis is not for the moment before us. I only wish to refer to catarrhal pneumonia as one of the several processes upon which fibrosis of the lung may supervene.

John B., aged 29, a butcher's assistant, came under my notice in March, 1871. He was a broad-chested, powerfully-made man, of medium height and florid complexion. He had led a rough but sober life,

having followed his present business, which included the slaughtering of animals, for some years in Australia, and had enjoyed excellent health until shortly before Christmas, when, after getting wet, he caught a severe cold, which was followed by a cough, which had since increased, unaffected by treatment. Up to and at the time of his attendance, however, he was still following his employment, but he now did so with difficulty, complaining of his cough and of increasing weakness with decided emaciation. His father had died of consumption, brought on, the patient stated, by intemperance; there was no other hereditary tendency to the disease.

The chest, as before said, was broad and well-formed, without flattening or obvious impairment of expansion. The heart's apex beat in the natural situation. At the left clavicular and subclavicular region the percussion note was dull, the dulness extending to the fourth rib; posteriorly, the resonance was defective at the left supra-spinous fossa. Scattered over the dull regions there was coarse crepitation, mingled with a still larger humid crepitus. These moist sounds were abundant, and masked to a great extent the respiratory murmur, which was decidedly harsh, but not distinctly bronchial. Its vesicular quality became gradually restored as the stethoscope was passed downwards. At the posterior base there were some scattered sibilant râles. On the right side the percussion note was good, and the breath sounds were natural.

The disease in this case began, then with a cold

on the chest—*i.e.*, a more or less general bronchial catarrh—which became localised[12] at the left apex, and extended there into the alveoli, producing catarrhal pneumonia; yet he continued his daily work, though constantly losing strength, for three or four months, during which time the catarrhal process ran on insidiously to a more deeply inflammatory degeneration of the alveolar walls. The disease had been acquired by exposure, the family tendency being very slight.[13] The whole build of the chest was not that of a man who inherited any tendency to phthisis. The physical signs at the present stage showed consolidated lobules of blocked alveoli, which were softening with varying degrees of rapidity; the coarse crepitation answering to the redux crepitation of pneumonia, the larger click being due to more profound destruction of tissue (softening).

There were two data defective in this history:—1. Fever. 2. Rusty sputa, or hæmoptysis. Although, however, the patient could give no definite and trustworthy information as to fever, we know for certain that he must have had intermittent attacks of fever; for it is a matter of clinical experience that we never get the signs of pulmonary consolidation and resolu-

[12] This bare statement is a clinical one only. We find in almost all cases of phthisis one apex affected first. Why cachectic bronchial catarrh should so strongly differ in this respect from ordinary bronchitis remains a mystery, but the fact is not the less clinically important.

[13] The father of this patient acquired the disease from his intemperate habits since the patient's birth.

tion or softening, dulness and crepitation, without there having been with each increment of pneumonia a period of elevation of temperature. A remarkable case well illustrating this occurred some two years ago in the Brompton Hospital under Dr. Sanderson's care, in which a woman had for weeks together daily attacks of fever of an intermittent character, and post-mortem one of the lungs was completely, and the other partially, consolidated, and as it were mapped out on section by lobular pneumonia of different dates. The patient whose case we are now considering had, on the occasion of his first visit, a quick pulse and a somewhat red tongue, and though there was no elevation of temperature at the time, it is very likely that at night it was a little raised.

It is here, however, perhaps, well to remember that we may have dulness and large or small liquid rhonchus without there being any fever present *at the time*; the fever may have passed away, but the consolidation which accompanied the fever cannot disappear so rapidly, but must run through a series of pathological and chemical changes essential for its removal by absorption or elimination—changes which are not accompanied by fever, and which may proceed *pari passu* with the rebuilding of the frame exhausted by the previous fever.

This reflection is of very great importance in a therapeutic point of view; for if, for instance, we were to treat ordinary basic pneumonia with antipyretics so long as the stethoscope revealed tubular breathing and moist sounds to be present, we should

get results deplorable in direct proportion to our auscultatory skill; and this remark applies with equal force to the lobular consolidations and softenings of phthisis. We should be acting as foolishly, if we regarded them as indications for treatment of an antiphlogistic kind, as if we continued to wrap up our damaged water-pipes after the thaw had set in instead of hastening to adopt measures to repair the breach as soon as the frost had gone.

Rusty expectoration is by no means a constant symptom of catarrhal pneumonia—not so constant, indeed, as it is of the croupous variety. It appears to depend partly on the degree of intensity of the pneumonia and the congestion with which it is attended, but also very largely upon the constitutional peculiarities of the patient.

To pursue the case, however, one step further. The patient was treated with an alkaline mixture containing small doses of iodide of potassium, and with cod-liver oil. The next note of importance was taken on April 27, when the expansion of the left side of the chest was noted to be decidedly impaired, the dulness had increased in hardness but not in extent, and was very marked, especially between the left margin of the sternum and the mid-clavicular line.

In the space marked out by these two vertical lines, (left sternal and mid-clavicular), the respiration was extremely feeble, and not attended with any rhonchus; the heart's impulse was diffused to the second interspace, though the apex was only half an inch higher than natural. To the left, again, of the mid-clavicular

line the respiration was still feeble, and the rhonchus much diminished, the dulness being somewhat increased. At the apex posteriorly there was bronchial respiration and imperfect pectoriloquy; the bronchial râles at the base had cleared up. The resonance of the right lung extended to the left margin of the sternum.

These signs showed—1. That the disease had not extended; on the contrary, the signs of bronchial irritation at the base had cleared up.

2. A wasting of the parenchymatous texture of the lung had taken place; degeneration, absorption, and expectoration had removed the morbid contents of the alveoli, and some of the lung tissue itself, leaving, perhaps, at the apex a small cavity; the general result being collapse and agglutination of air cells. Hence a considerable reduction in the bulk of the lung and the retraction of its anterior margin *away from the median line;* so that between the left sternal line and a line drawn from the point of junction of the inner and middle third of the clavicle to the apex of the heart there was probably at this date no lung at all.

3. An encroachment of the enlarging right lung, a slight shifting of the heart to the left, and a flattening of the chest wall to make up for the lost space. The chest wall flattening was, however, very slight, and not yet noticeable until the patient drew a breath. The man had powerful parietes, and in such cases the displacement of heart and encroachment of the opposite lung precede, often for a long time, any obvious flattening.

It was remarkable with what rapidity these changes were taking place, and there can be no doubt that the connective tissue of the bronchial and perivascular and pleural sheaths were undergoing rapid development, and that the case was at this date not merely one of catarrhal pneumonia which had subsided after having caused a certain loss of lung substance, but that an interstitial pneumonia was proceeding ; the case had changed its type to one of peribronchial, or rather pulmonary, fibrosis ; it had lapsed into one variety of fibroid phthisis. That the disease was not yet arrested seemed probable from the patient still losing slightly in weight and becoming more anæmic ; but it had clearly become limited.

During the next month he lost two pounds. He was during this time taking an acid preparation of iron, with a little quinine and the oil. Notwithstanding this slight loss of weight, he had improved generally ; cough and expectoration had diminished, and he felt stronger. On June 8 he was still better, and had gained one pound since last report. He had very little cough ; all moist sounds had disappeared except a slight friction (?) on cough at the outer side and a little above the left nipple. He has since steadily improved. (August 1871).

Dr. Andrew Clark related at the Medical Society a few months ago (vide *Lancet*, May 6, 1871) a case very similar to the above. The subject of his paper, a boy of 14, had been a patient of mine at Brompton some five or six months before he came under the notice of

Dr. Clark, and I took the same view as to the condition present, which Dr. Clark described by the name "peribronchial fibrosis." The case appeared to me, however, in its earlier stages, to be one of catarrhal pneumonia, undergoing the process of softening, upon which pulmonary induration supervened, in consequence partly of ordinary cicatricial collapse, partly from the vascular bronchial and pleural sheaths taking on an active growth. I should prefer to regard it therefore with that above described, as belonging to a section of the large class of pulmonary fibrosis, or fibroid phthisis, since, though in both cases there was undoubtedly at first more or less general bronchial catarrh, still it was the apex pneumonia, of an intensity sufficient to deeply affect the fibrous framework of the lung at that part, but not sufficient to lead to its destruction, which set going, as a subsequent result, that active hyper-growth which produced such important modifications in the morbid structure and physical signs. As I look upon this new disease, or phase of the disease, as affecting the whole framework of the portion of lung involved, I think the more general term pulmonary fibrosis a better one under which to include it.

This disease—fibrosis—has, I think, little or no tendency to spread beyond the limits of the original disease which gave rise to it, and, provided the patients are careful to keep up their general health, and to avoid fresh catarrhs, they do well. The indurated portions of lung usually enclose some nodules of dry cheesy matter, and one or two small excavations,

which are often the seat of a necrotic crumbling process, which continues for a long time without affecting the general health, merely causing a slight irritative morning cough.

This crumbling process is, however, very favourable to the uncovering of vessels of considerable size, without their becoming obliterated by the coagulation of their contents. Small ectasias of these pulmonary branches, or even considerable aneurisms, are thus more apt to arise in these indurated lungs than in others, and the possibility of severe or fatal hæmoptysis must not be absent from our minds in framing a prognosis.[14] Also, hæmoptysis occurring in cases of this kind must for the same reason be regarded with greater anxiety than in ordinary cases of phthisis.

There is only one special remark concerning the treatment of these chronic indurative cases of phthisis during the often extended period of quiescence, which seems called for, and it is this—that though such cases require careful *surveillance*, and for several years, where practicable, carefully selected climates to suit the different seasons of the year, they do not require the persistent administration of tonic medicines and cod-liver oil. They improve immensely under such remedies up to a certain point, which may be readily recognised by the medical attendant, and cannot be better described than by saying that it amounts to the most perfect health attainable by a

[14] I have treated this subject somewhat fully in the last volume of the Pathological *Transactions*, pp. 41-65.

patient who has had a certain area of respiratory surface cut off. If beyond this point we persevere with iron and oil and too nourishing or stimulating a diet, we may still further increase weight and heighten colour, but the pulse quickens, the patient gets more short of breath; he becomes, in a word, plethoric, and liable to pulmonary congestion and hæmoptysis, or to dyspepsia and diarrhœa; and a rapid neutralisation of all the good results obtained, with great danger of fresh and perhaps fatal renewal of the old disease, is the consequence of too great anxiety, both on the part of the patient and the doctor, to again arrive at a degree of health and bodily vigour which is impossible with a permanently damaged lung. I have repeatedly noticed the arrival of patients at this stage of constitutional balance, and have witnessed the quickened pulse and returning fever consequent upon the indiscreet endeavour yet further to urge on the health with tonics.

Having thus touched upon the subject of Fibroid Phthisis and shown how that contractile variety of the disease may arise secondarily by gradual transition from the ordinary catarrhal pneumonic variety, I will complete what I have to say about this form of phthisis in the next chapter, in the course of some comments upon a typical example illustrating its main features.

CHAPTER V.

Fibroid Phthisis, its characteristic signs and pathology summarised; reasons for selection of term as most applicable—A disease secondary to some perenchymatous affection of the lung, doubtful if pleurisy or bronchitis alone will give rise to it —Relations to other forms of phthisis—Example related—Summary—Remarks on diagnosis, prognosis, and treatment.

CERTAIN forms of pulmonary phthisis of various origins are attended by such an amount of interstitial fibrous growth as to give them clinical features of a very peculiar type.

The prominent symptoms and signs by which such cases are distinguished,—increasing contraction and immobility of the side, dragging pains, traction of organs to that side, deadened percussion-note, and weakened respiration of more or less bronchial quality, intensely so, or cavernous, at parts; breathlessness, paroxysmal cough, occasional hectic, but general absence of fever, very chronic progress, long-continued one-sidedness of the disease, and correspondingly slow failure of nutrition—show them to come within the definition of phthisis, but phthisis of a special kind.

The conditions presented to us, post-mortem, of a contracted, toughened, indurated, and usually pigmented lung, surrounded by a greatly thickened adherent pleura, containing one or more rigid, dense-walled cavities, dilated bronchi, and cheesy encapsuled nodules, are confirmatory of this view.

On minute examination, we further discover this condition of lung to have been produced by a growth of two kinds pervading it. 1. Connective tissue proliferation, resulting in the formation of bands and processes of *fibrous* tissue, derived from the sheaths of vessels and bronchi, and the sub-pleural and inter-lobular tissue of the lung. 2. A more important nuclear growth leading to the formation of broad tracts of *fibroid* tissue, thickening the walls of the alveoli, compressing, and finally effacing them, unless they shall have been previously stuffed with their own catarrhal products; this fibroid structure being very possibly derived from the lymphatic elements normally pervading the lung.

The products of these two processes become intimately mingled, but it is the latter which is the phthisical element in the disease, for mere connective tissue growth does not lead to organic destruction of the lung. This is also the element which specially gives to the disease its peculiar clinical features, and renders the name "fibroid phthisis" applicable to it.

The term "fibroid phthisis" has been productive of much discussion. Originally introduced by Dr. Andrew Clark as "embracing all those cases, whether local or constitutional, which are anatomically characterised by the presence, in a contracted and indurated lung traversed by more or less dilated bronchi, of fibroid tissue, and of tough fibrogenous substance, together with cheesy deposits or consolidations, and usually small cavities, commonly found about the middle and lower parts of the affected organ"[15]—it

[15] Clinical *Transactions*, vol. i. p. 188.

has been objected to as signifying too definitely the existence of the disease as a substantive or idiopathic one. Dr. Clark is, however, convinced that it may, and often does, appear as a primary affection, in which view he differs from most contemporary authorities. Still, the term is such a neat, concise, and clinically useful one, that it has been very generally accepted with the above reservation.

Dr. Clark argues, with much force, that these cases are indisputably cases of phthisis, while all their special characters are due to the pervasion of the affected lung by a contractile fibroid and *fibrogenous* tissue. These two facts cannot, I think, be controverted; and when it is further urged, as I think it may truthfully be, that this fibroid tissue is indistinguishable in situation and development from chronic tubercle, a very fair case may be made out for the preservation of the term "fibroid phthisis," though its distinguished author would, I fear, not admit this latter argument.

I ventured, in a paper read before the Clinical Society in October, 1868 (*vide* Clinical *Transactions*, vol. ii., p. 193), to question whether this term was advisable, since the pure constitutional disease was of such rare and doubtful occurrence. Later reflection and the observation of a good many cases have, however, led me to think that this objection, though by no means weakened,[16] is not sufficient to render

[16] See article by Dr. Wilson Fox on Chronic Pneumonia in Reynolds' *System of Medicine*, vol. iii. p. 772.

the term clinically inapplicable, since "fibroid" only corresponds with other adjective terms—"catarrhal," "tubercular," etc.—in describing the *predominant* character of the variety of phthisis thus distinguished; for in all cases of chronic phthisis the morbid processes are of more or less mixed character.

Dr. Wilson Fox in the exhaustive article above referred to, includes under the term "chronic pneumonia" all cases of pulmonary fibrosis uncomplicated by tubercle: his chronic pneumonia would therefore agree with Dr. Clark's fibroid phthisis, except that he does not admit the disease as a primary or constitutional one. On the contrary, he contends that it is always associated with, and dependent upon, catarrhal pneumonia; hence his employment of the term "chronic pneumonia." Dr. Fox, though thus characterising the typical disease as essentially one of pneumonia affecting the lobules of the lung, not the connective-tissue which binds them together, acknowledges that freedom from tubercle is very exceptional.

Dr. Bastian, on the other hand, would include all these diseases under the old term "cirrhosis of the lung." Cases to which the term "cirrhosis" is strictly applicable, in which there is no catarrhal pneumonia or tubercle, nor any true organic destruction of the lung are, however, so rare as to render this latter name a misleading one.

This disease, then—fibroid phthisis— is in the great majority of instances, so far as my own experience informs me, of a truly secondary nature, supervening

upon some more or less acute inflammatory affection of the lung, whether simple basic-, or broncho-, or catarrhal, or tubercular pneumonia. It is very doubtful whether pleurisy or bronchitis alone can give rise to it without the intervention of lobular pneumonia or tubercle. Local injury or pulmonary abscess may form the starting-point of the disease, but as before observed are not alone sufficient to cause any extensive fibroid invasion of the lung beyond their own immediate limits.

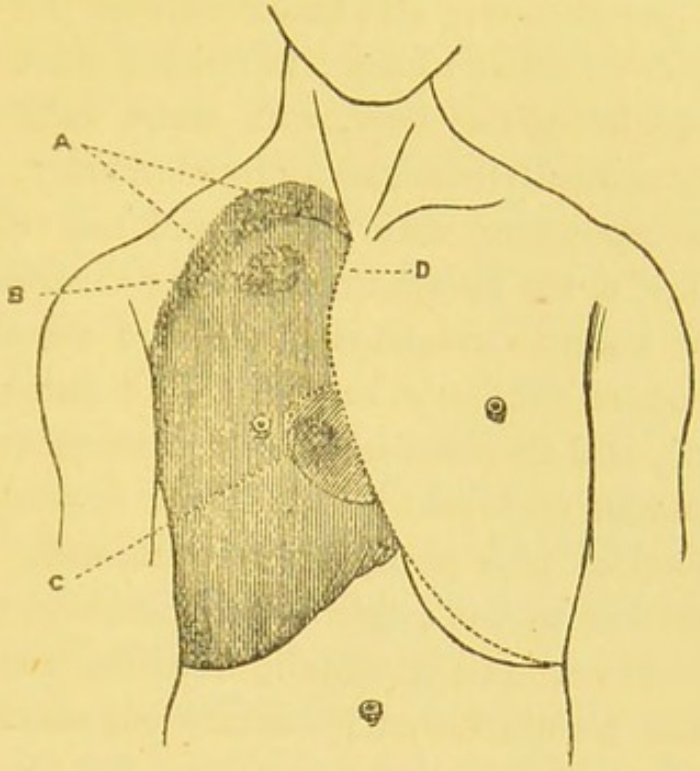
Numerous examples may be found of this somewhat inclusive disease, ranging from the most typical cases to those which are indistinguishable from ordinary chronic phthisis. In the last chapter I describe a case of catarrhal-pneumonic phthisis, in which the transition into one of fibroid phthisis or pulmonary fibrosis was traced. It would not be difficult to find examples in which the reverse takes place, the clinical characters of fibroid phthisis being gradually changed by subsequent pneumonic processes, and all the features of the special variety becoming merged in the diffuse pulmonary destruction.

Cases of fibroid phthisis may be roughly divided for convenience of clinical exposition into three varieties :—1. Those in which the disease has its starting-point, at the apex of the lung, and is proceeding downwards. 2. Those in which it commences at the base, and advances upwards. 3. Those in which the most marked signs are discoverable about the middle of the lung. Of the first variety the following case is a fair example :—

George P., a sawyer, aged 43, came first under my notice as an out-patient at the Brompton Hospital in August, 1868. He was a thin man, with dark hair, having no hereditary predisposition to lung disease, except that his father had suffered from "asthma." In the preceding January, having suffered from slight cough for years, he was laid up for six weeks with "inflammation of the right lung." Since that time the cough had been continuous, and three months ago had been attended with slight hæmoptysis. The cough was now paroxysmal, causing retching and often rejection of food; expectoration difficult, abundant, and of a pink tinge. He had got thinner lately. The digestive functions were fairly good; the pulse a little hurried; there was no fever present.

On inspecting the chest, cardiac pulsation was visible at the fourth right interspace, c, to left of right nipple. This side was diminished in size and much restricted in movement, the intercostal spaces deepening with inspiration; while the left side expanded freely, with an uplifting movement of the shoulder. On careful examination, the apex of the heart was found a little to the left of the ensiform cartilage.

On percussion, the left side in nipple-line anteriorly was dull to the second rib, comparatively resonant to the fourth, and below this point it was again toneless. To the left of the line of sterno-clavicular articulation, at the level of the second and third cartilages, there was good resonance continuous across median line with that of the opposite side.



The line of this resonance, D, sloped upwards to the episternal notch, in a downward direction being displaced by cardiac dulness at the fourth cartilage. Hepatic dulness barely reached the costal margin. There was dulness throughout the *axillary* region and *posteriorly* from apex to mid-scapula, the note having a tubular quality in this latter region. Below mid-scapula there was fair resonance to the ninth rib, though less and harder than on opposite side; the lower two or three inches on the right side gave a flat note on percussion.

The percussion-note over the whole left side, including the region of normal cardiac dulness and extending across the median line, as above indicated, was full and good in front and behind.

The auscultatory signs were in agreement with those of percussion. Above the clavicle the respiration was amphoric and dry; below the clavicle weak and bronchial to the base, with some rather large, moist rhonchus, friction, and bronchophony. At one spot corresponding with the second and third ribs nipple-line, B, the breath-sound was of tracheal quality, with scanty cavernous clicks and pectoriloquy. In the upper axillary region the respiration was amphoric, and the voice-sound pectoriloquous; in the supra-spinous fossa and interscapular regions, cavernous blowing, with pectoriloquy. Blowing respiration extended to the angle of the scapula, where it became weaker and gradually annulled at the base. The vocal fremitus was generally increased on the right side. Respiration throughout left lung exaggerated vesicular.

February 27th, 1869.—Patient improved in flesh and appearance; stronger than before, but complains much of cough, and expectorates much pink phlegm. Breath short on exertion; cough causes retching, but does not bring up food now; appetite fair; digestion not very strong; bowels regular. Continues medicine (oil with nitro-muriatic acid and cinchona). Fingers noted (February 13th) to be clubbed.

The additional physical signs noted at this date were, a distinct short systolic bruit at the point of maximum cardiac impulse, not appreciably increased by pressure nor confined to that spot, being also audible at the apex. Measurements of chest:—

From mid-sternum to nipple, right side, 4 inches; semi-circumference, 15¼ inches; expansion, ¼ inch.

Left sterno-nipple measurement, 4¼ inches; semi-circumference, 16 inches; expansion, ½ inch.

We may, by way of summary, aided by a glance at the figure (reduced as accurately as possible from a sketch taken at the time upon an outline diagram), interpret the above detailed physical signs as indicating at this period a general induration of the right lung, with much contraction, its anterior margin having receded considerably from the median line, exposing the pericardium, and having also shrunk away from the upper surface of the liver. Its upper and a portion of its lower lobes were extensively excavated, the cavities being old, tolerably dry, and shrunken with the general contraction of the lung. The pleura (from the hardness of percussion, feebleness of breath-sound, and great fixity of walls) was probably greatly thickened. The liver was drawn up within the costal margin, and the heart considerably displaced to the right, its axis being, however, but little altered. A short systolic murmur is heard over the heart.

Subsequently to the last note I repeatedly examined his chest during the many months of his attendance at the Hospital, but beyond some variation in the dryness of the sounds there was no important change in the physical signs. I fancy I examined the urine more than once, but the only note I have of it was taken in April, 1869, when it was acid, became slightly turbid on boiling, but cleared on adding a drop of nitric acid.

The left lung remained healthy, and, though the patient continued thin and cachectic-looking, with a troublesome cough, he held his ground fairly well, and rather improved in general health. At times the expectoration would become very abundant, and occasionally of a pink colour, (I thought due to fresh irritation and slight sanguineous discharge from the walls of the old cavity). The most troublesome symptom throughout the case—and one which is common in greater or less degree to all those cases of phthisis in which indurated thick-walled cavities are present—was the paroxysmal cough terminating in vomiting, occurring especially after meals.

No doubt the mechanical conditions of such a cavity, rendering the removal of expectoration very difficult, have much to do with the production of vomiting, and render it a particularly common symptom in these cases: but the reception of food into the stomach has seemed to me to be in many cases so constantly followed by cough ending in vomiting, as to render this mechanical explanation insufficient, and, in 1869, I was led to attribute it to an undue reflex irritability of the pneumogastric nerve and proposed strichnia as the best remedy.[17] I have since, in many cases, found this remedy valuable, but by no means invariably so.

On leaving off attendance at the Hospital in May, 1869, the patient, though not free from cough, con-

17 *Practitioner*, vol. i., p. 312 :—Dr. Hughes Bennett (Reynold's "System," vol. iii., "Phthisis," 1871) regards this as a cause of vomiting in the later stages of phthisis.

tinued for a time to improve, but he soon afterwards began again to emaciate, and the vomiting with cough returned. He again attended in January, 1870, resumed the oil and used carbolic acid inhalations, and left March 30th, improved. Since this time I have heard nothing of him.

The above-related case represents very well the main features of fibroid disease of the lung. The indurative disease supervened presumably upon an acute apex (tubercular or catarrhal) pneumonia, and did so with tolerable rapidity, the characteristic symptoms and signs being fully developed within six months of the termination of the acute disease.

The question as to the rapidity with which this disease may advance is one of great interest, and requiring further observation. I cannot but think that, reasoning from the morbid appearances found *post-mortem*, we are apt to regard such diseases as older than the clinical history will warrant us in believing; on the other hand, though it is very probable that the fibroid induration of the lung may proceed with great rapidity to such a stage of shrinking as to produce very marked clinical signs, its subsequent progress is very slow and difficult to measure, consisting mainly in the further hardening of an already indurated lung, the gradual widening of the bronchial tubes, and filling up of the loose œdematous areolar tissue between the separated pleural layers by dense fibrous growth. We can readily perceive therefore how the earlier stages of the disease which are attended with striking alterations in

physical signs may, if the original disease is of considerable extent, be passed through with comparative rapidity, while the later progress is necessarily slow and difficult to estimate.

The resonance of the opposite lung extending across the median line would emphatically exclude cancer, which the history of the case, and many other signs, particularly the kind of cardiac displacement, would also negative. While in certain cases of mediastinal cancer the heart is fixed in about its normal position, it is, I believe, never displaced towards the side most affected. In regarding the signs of cardiac displacement, however, it is important to avoid taking the point of maximum impulse as necessarily the apex beat. In this case the real displacement of the heart is much less than a first glance would lead us to believe. The absence, while the patient was under observation, of any evidence of complication of other organs would be in favour of the disease being of local origin.

In calculating the *prognosis* in these cases, we have to bear several things in mind—the cachexia of the patient, the size and freedom of communication with external air of any cavities present, and the evidence of disease of other organs especially of the opposite lung.

The cachexia is occasionally very manifest; without very marked emaciation the anæmia is apt to become great, and the complexion of a straw-tint, reminding one of that seen in the later stages of certain cases of cancer or in women suffering from uterine disease.

None of these peculiarities were in the present case noticeable in any marked degree.

A cavity of considerable size, and freely communicating with the outer air, is a more hazardous condition for the patient than one which is small, or which we may presume to have become flattened and partially or completely closed. In the former condition the patient is constantly liable, on exposure to cold etc., to recurrence of irritation and fresh ulceration of the cavity-wall causing profuse secretion and hectic, as in the present case, or laying bare vessels which may at any time rupture and cause death from hæmoptysis. Out of eight well-marked cases of which I have made post-mortem examinations, this has been the cause of death in two.[18]

When the opposite lung is affected, it is most generally by grey tubercle, and it in most cases becomes so sooner or later, unless the patient be cut off too soon by some intercurrent affection. We must, however, not give a too hastily fatal prognosis from mere physical signs in such cases, for the course of the tubercle is disposed to be very chronic and indurative, and the signs may again subside and long remain in abeyance. But when attended with decided elevation of temperature, the prognosis is most grave, for pulmonary tuberculosis is perhaps the most common cause of death in these cases.

[18] For an account of one of these, Downer, vide *Clinical Transactions*, vol. ii., p. 181 ; the other is referred to in a Table on Hæmoptysis, *Pathological Transactions*, vol. xxii., p. 58, Case 1, F. W.

Dr. Clark regards albuminoid degeneration of other organs—the liver and kidneys—as commonly supervening in the later stages of this disease. Of the three purest cases of which I have made *post-mortem* examinations, in which there was no obvious grey tubercle in the other lung (though islets of peribronchial induration were present in all), in one there was extensive albuminoid degeneration of liver and spleen with granular kidneys; in another of the spleen only, with ulcerated intestines. Albumen in the urine, absent in the present case, affords us the earliest clinical evidence of this degeneration.

Although the prognosis is always, in cases of fibroid phthisis, a precarious one, from the circumstances above mentioned, yet the course of the disease may be very long, and may with due precautions in some instances be almost indefinitely extended. The condition of health and *physique* maintained by some patients is remarkably good. In this respect the example above related is an unfortunate one; I have notes, however, of a postman presenting exceedingly well-marked signs of this form of phthisis, who almost entirely lost his cough while attending the Hospital, and was able to resume his duties, walking fourteen or fifteen miles a day; and I might mention two or three other patients capable of considerable physical exertion on level ground.

The management of this variety of phthisis calls for no special remark; the prevention of fresh catarrhs by judicious clothing, the selection of climate when

practicable, the avoidance of night-air, and protection from irritating fogs or cold or damp winds by respirators, are, with a nutritious but not stimulating diet, the hygienic measures to be adopted. Iodine frictions, soothing or antiseptic inhalations (carbolic acid being particularly useful when there is any fœtor of expectoration), seem the best local remedies; while the general condition, including that of digestion, the nature of the cough, and profuseness of expectoration, supplies us with indications for the administration of appropriate drugs—iron, cod-oil, strychnia, alkalies, tonics, etc,—or the abstention from all medicines.

The very full consideration of the above example of fibroid phthisis, and of the general bearings of the disease, renders it perhaps unnecessary for me, to extend this already very long chapter by entering into those variations in the signs of this form of phthisis which depend upon its seat of origin.

There are a few points for consideration in the *diagnosis* of the above case.

That it is not simply a case of contracted cavity is evident from the contraction of the side being general, from the heart being displaced laterally, not specially drawn up towards the right apex, and from the weakness of breath-sound, with dulness at the base, and raising of the liver. The presence of considerable excavation at the apex would be in favour of the disease having commenced there as an apex (phthisical) pneumonia, and not being secondary to basic pleuro-pneumonia or empyema. It would also negative its being a case of simple cirrhosis of the lung.

CHAPTER VI.

On Hæmorrhagic Phthisis—Subject divided into Hæmorrhagic Phthisis proper and Recurrent Hæmoptysis: distinction between the two—True hæmorrhagic phthisis very rare; difficult to ascertain the true relation of the hæmoptysis to the primary disease: views of Niemeyer—Illustrative case of hæmorrhagic phthisis—Remarks: (1) Why considered a case of phthisis; (2) Why hæmorrhage not bronchial, and affecting lung secondarily; (3) Why probably coincident with and caused by active pulmonary congestion; general conclusion, with definition of Hæmorrhagic Phthisis; further points for and against hæmorrhage in such cases being bronchial—Disease has no relation to Hæmorrhagic Diathesis—Remarks on Treatment; value of thermometer as a guide.

The term "hæmorrhagic phthisis"[19] has crept into use to distinguish those cases of phthisis which have a distinctly hæmorrhagic origin, and also those in which, however they may have arisen, hæmoptysis is a

19 "Phthisis after Bronchial Hæmorrhage" (Niemeyer, Burger, 1864), "Phthisis Hemoptoique" (Herard and Cornil, 1867), "Hæmorrhagic Phthisis" (Dr. Williams, 1868). "Tuberculose nach Hæmoptoe" (Waldenburg, 1869), "Hæmoptysical Variety" (Dr. Peacock, 1870). These several authors, though doubtless they would agree in acknowledging certain typical cases as representative of this hæmorrhagic form of consumption, would differ widely in the liberality with which they would group more doubtful cases among such representative ones, nor would they more closely agree as to the pathology of the affection. The observation of this difference of opinion among enquirers so eminent, is alone, I hope, sufficient to make me fully aware of the difficulties of the subject.

marked and oft-recurring symptom. For clearness sake it will, I think, be better to restrict the term to the first set of cases to which it was originally applied—those of hæmorrhagic origin; and to refer to the second class later as cases of phthisis with recurrent hæmoptysis.

The question as to the *frequency* with which phthisis arises as the direct result of hæmoptysis is one of great difficulty to determine, and must indeed, I think, ever remain one of opinion. Of the possibility of blood having obtained entry to the air-cells and coagulating there giving rise to broncho-pneumonia, and subsequently to phthisis, the observations of Professor Niemeyer, and Drs. Hermann Weber, Baümler, and others have furnished us with clear evidence. I have myself seen on several occasions *post-mortem* examples of inhaled blood forming the nucleus of fresh lobular pneumonia in the grey stage in cases—it is true, of tolerably advanced phthisis—in which death ensued after recent hæmoptysis.

The difficulty, of course, lies in determining whether the hæmorrhage is the cause of the disease or is itself the result and evidence of pre-existing or coincident disease. It is an undisputed fact that in a certain number of cases, more or less copious hæmoptysis is the very first symptom of the pulmonary disease, preceding, even for a considerable time, all reliable physical signs.

Professor Niemeyer asserts that the hæmorrhage in these, as in the majority of cases of phthisis in all its stages proceeds from the bronchial mucous mem-

brane; that a portion of the blood becomes inhaled into the air-cells of the previously healthy lung, coagulates there giving rise to irritative lobular pneumonia, the consolidations of which may subsequently decay and soften leading to destruction of the lung, or may become cheesy, and give rise at some subsequent period to tuberculosis of the lung of a secondary or infective kind.[20] I must confess that I have never met with a case which I could distinctly refer to this category, nor are the cases which Niemeyer quotes in support of his view to my mind conclusive.[21]

I think the phenomena exemplified in the following case may be fairly regarded as illustrative of the hæmorrhagic variety of consumption, while they also point to the hæmorrhage being of pulmonary, not of bronchial origin.

J. P., aged 20, a servant residing at Faringdon, came to see me at the Brompton Hospital on September 21st, 1871. She was a fine, well developed woman, with a decided tendency to *embonpoint*, a clear complexion, a high colour, in which, however, a slight degree of lividity was noticeable. She was complaining of shortness of breath and bad cough with expectoration, and had the following history:—Of

20 Waldenburg, fully admitting this view, seems inclined further to think that the inhaled blood, by the re-entry of its shrivelled elements into the circulation, may give rise directly to true tuberculosis. *Die Tuberculose*, p. 496.

21 This point is very fully discussed by my colleague, Dr. C. T. Williams, in his chapter on Hæmoptysis in the recent work on *Pulmonary Consumption*, by Dr. C. J. B. Williams and himself.

healthy parentage, and with no hereditary tendency to consumption,[22] she had enjoyed good health—with the exception of the formation of an abscess at the top of the sternum, which discharged eight years ago, leaving a depressed scar—until twelve months ago, when she began to suffer from palpitation on exertion; she did not consider that she had been ill, however, for more than six months, with increasing breathlessness and palpitation and slight dry cough.

Three months ago she had an attack of tolerably copious hæmoptysis which lasted nine days, and kept her in bed three weeks: five weeks ago she had a repetition of the hæmoptysis to a less degree. She had not menstruated for four months, having previously done so with regularity; but though the catamenia ceased about a month previous to the first attack of hæmoptysis, she did not herself connect the two facts together; they had again appeared a day or two ago. She had never suffered from epistaxis. She now complained of shortness of breath, troublesome cough with expectoration, which, however, was not tinged with blood. She had got thinner of late, and the pulse was rather accelerated and small.

There was at this date no alteration in the shape of the chest, which was remarkably good, nor any decided dulness; but on the right side there was diffused crepitation throughout, mingled with vesicular harsh breath-sound. On the left side the respiratory murmur was exaggerated. The case

[22] There is some doubt about one sister who died of bronchitis, after about six weeks' illness, at the age of 4 years.

was regarded as one of irritative catarrhal pneumonia of the right lung, secondary to copious hæmorrhage (probably vicarious) from the apex of that lung. At this time there was no evidence of positive destruction of lung, and I strongly advised her to seek the shelter of the Hospital as an in-patient with the hope that she might make a complete recovery. She was not able to do so, however, until November after having had a slight return of the hæmoptysis at the end of October.

November 17th.—Having been in the Hospital for a few days, under the care of Dr. C. T. Williams, I again examined her. There was slight but decided dulness on percussion at the right supra- and infra-clavicular region, fading both downwards and laterally towards the median line into good resonance; above the clavicle the resistance to percussion was greater than on the opposite side; at the extreme posterior base there was almost complete dulness for three fingers breadth, not so anteriorly nor in the axillary region. Heart's apex-beat in normal situation. The respiratory murmur at the apex was entirely masked by coarse moist crepitation accompanying both inspiration and expiration; vocal resonance increased, however, in supra-spinous fossa. Below the apex respiratory murmur feeble; crepitation to base, but less abundant than above; at posterior base absence of respiration over the dull portion, with some incomplete ægophony. On the right side respiration loudly puerile, well audible to mid-sternal line. The complexion was still fresh

and highly coloured, without, however, any lividity. Cough now only slight; expectoration a tenacious mucus, clear and slightly pigmented. Temperature normal: pulse 104 to 112; weight of patient, nine stone.

I quote this case because I think it is as good an example of hæmorrhagic phthisis, in the sense of phthisis secondary to hæmoptysis, as is ever met with. There are three queries which must, however, be answered respecting it: 1st, Is it a case of phthisis at all? 2nd, Is it a case of phthisis caused by the inhalation of blood effused from the bronchial mucous membrane—phthisis *ab hæmoptoe*, in the sense of Niemeyer and others? or, 3rd, Is it a case of pulmonary hæmorrhage coincident with the occurrence of that active pulmonary congestion which is the very first stage of a certain number of cases of phthisis?

1. Though at present (November 17th, 1871,) there is no evidence of *wasting* of the lung, yet there is sufficient evidence of irreparable damage to its texture—*i.e.*, there has been slowly diminishing breath-sound, with abundant crepitation, since she first came under observation in September, until now the signs at the right apex seem only compatible with the air-cells there having become completely blocked with their epithelial products, now in the process of degeneration (caseous pneumonia).

The significance of such gradually developing apex-signs after hæmoptysis cannot, I think, be mistaken; softening and removal of lung tissue, to a greater or less extent, with indurative shrinking of

the lung, are the almost necessary consequences. In all probability, the disease in the lower part of the lung is the direct result of the irritation of the inhaled blood, and much of it may no doubt yet clear up—indeed, had the patient obtained proper shelter and care from the first, it might have done so entirely; but, though all fever is now absent, the physical signs show that much of the lung yet remains clogged with inflammatory products.

2. I think the facts *(a)* of the opposite lung having wholly escaped, and *(b)* of the apex of the right lung being so decidedly affected, are very strong evidence against the probability of the hæmorrhage having been from the bronchial mucous membrane.

3. The same two facts, and more particularly the gradual increase in the apex-signs, and the concurrence of the *copious* hæmoptysis with the severe lung-symptoms, are equally strongly in favour of the hæmorrhage being truly pulmonary—*i.e.*, of an acute pulmonary congestion, the true first stage of this (pneumonic) form of consumption having been attended with hæmoptysis to an unusual extent, the blood having also gravitated to other parts of the lung, and set up irritative changes there.

No doubt, at first—*i.e.*, soon after the hæmoptysis—the basic- were in excess of the apex-signs; this is the case in many instances of copious hæmoptysis in the first stage of phthisis. The explanation which appears to me most plausible is, that the natural effect of gravitation, aided by the expansile movements of the lung, is to remove the blood from the air-cells at the

apex of the lung[23] which, therefore, often escape blocking, while they are the very physical conditions which most aid its entry into the lower portions. On the other hand, had the blood welled up from the base of the lung, it is unlikely, for the same reasons, that the opposite lung would have escaped.

It may be said—and here lies the uncertainty of all cases of this kind—that there was some disease existing at the right apex prior to the hæmoptysis. It is true that the patient had some cough, but it was only very slight, unattended with constitutional symptoms of any special kind, and not apparently differing from the short cough so commonly associated with palpitation, of which she also complained.

We may say, at least, that the *onset* of the disease was *with copious hæmoptysis in a person previously with no apparent chest disease,* and, with the exception of some menstrual irregularity, and the palpitation so commonly associated with this condition, in fair health; we are further certain that *a considerable amount of the disease*

[23] The blood sometimes coagulates too quickly for this, and then the physical signs vary greatly from day to day, as in an interesting case of Dr. Gee's, of which he has kindly shown me the notes. In this case the signs were *nil* after the first hæmoptysis: immediately after a second attack, however, there were signs at the right apex, which were modified from day to day. The hæmorrhages were frequently repeated here, and the blood expectorated was commonly dark and clotted, having apparently been retained some little time. Some temperature observations are much wanted immediately after the first copious hæmoptysis in cases pursumed to be of hæmorrhagic character. Dr. Gee considers it to be elevated from the first.

present is the result of the hæmoptysis; and these two facts are sufficient to mark the case clinically as one of *Hæmorrhagic Phthisis.*

It is obvious that it is impossible to draw the line between those cases in which the acute congestion—whether of vicarious or mechanical[24] origin or arising from a chill or other cause—results in hæmorrhage in a lung previously sound, or determines what is often the first sign of consumption, hæmoptysis, from a lung whose vessels are frail from previous error of nutrition or disease at one portion. The distinction is mainly drawn from the clinical history of the case; and there is perhaps no true pathological difference, since we cannot on present evidence admit the hæmorrhage to be bronchial.

The chief fact upon which those who attribute early hæmoptysis to hæmorrhage from the bronchial tubes rely, is the extreme difficulty often experienced by the ablest auscultators in detecting any physical signs (but those, perhaps, of bronchial irritation) a short time after even very copious hæmoptysis. A man comes for examination a day or two after bringing up a large quantity of blood, and absolutely no signs which one could definitely pronounce as in-

24 The term mechanical source is meant to include those cases in which the excited action of the heart and pulmonary engorgement connected with violent muscular exertion—excessive dancing, rowing, etc.—lead to hæmoptysis. I do not know of any well-authenticated case of the kind. In Dr. Weber's first case (*Clin. Trans.*, vol. ii., p. 143), dancing evidently caused a recurrence of the hæmoptysis.

dicative of the origin of the hæmoptysis are discoverable. This very commonly happens. There may be the slightest comparative harshness and feebleness of respiration at the summit of one lung, from which long experience of the subsequent phenomena leads one to judge that the hæmorrhage has arisen there, but which without such experience would be considered wholly inadequate to account for the astonishing hæmorrhage.

I have already pointed out above that the readiness of escape permitted to the blood from the apex by the aid of gravitation and the expansile motion of the air-cells appears to me to be the explanation of this difficulty. It is the too common experience of the subsequent development of physical signs indicative of decided disease at the point which we could only guess to be the source of hæmorrhage before, which enables us in such cases to speak decidedly upon what would otherwise be insufficient evidence.

It will be observed that the *hæmorrhagic diathesis* has not been spoken of in connection with hæmorhagic phthisis. I believe, indeed, that the two diseases have no causative relation to one another. Cases are common enough in which there is a tendency to slight hæmorrhage from the gums, slight hæmoptysis apparently from the mucous membrane of the large bronchi or the throat, often associated with menorrhagia. I have watched many such cases for a long time, but none of them have ever become phthisical, or suffered from very copious hæmoptysis. Cases of true hæmorrhagic diathesis are of course

rare, and I am therefore glad to have the opinion of my friend Dr. Legg, who has paid much attention to *hæmophilia* and its literature, and who finds the subjects of this disease are rarely affected with copious hæmorrhage from the lungs while among them Phthisis is still more rare.

Perhaps the chief advantage in retaining the term "hæmorrhagic phthisis" lies in its directing attention forcibly to the fact that hæmoptysis must not be looked upon only as the symptom or sign of disease, but as being also the *potential cause* of fresh disease. This consideration has a very important bearing upon the treatment of hæmoptysis.

We know that this early hæmoptysis is rarely fatal, and therefore, after calming the patient and securing for him perfect repose, we may anticipate spontaneous arrest of the hæmorrhage, or may endeavour to stop it by appropriate drugs and other means. Our whole anxiety is, however, in the immediate future, to watch for, and if possible to avert, the secondary consequences of the bleeding. Any detailed physical examination of the chest is, while the hæmorrhage continues, to be carefully avoided.

The thermometer, happily without danger to the patient, gives us the information we most require, and, together with the pulse and general aspect of the patient, is the best guide in the management of the case. If the temperature is raised at the time, or within a few hours of the hæmoptysis (it is often depressed for a few hours by hæmorrhage from the lungs), we may conclude it to be of congestive or in-

flammatory origin, and we anxiously watch for a few days to see whether the fever subsides with the hæmoptysis, or whether a fresh accession takes place significant of those secondary inflammatory changes we have reason to dread.[25]

The patient is usually seen after the first burst of hæmorrhage; and if the bleeding continue after quiet is secured, astringents may be found useful. I believe gallic acid to be the best of them, but it must be given in large doses of gr. xx. or ʒss. Unless, however, active bleeding is going on, I am content in cases of hæmoptysis to give nitro-muriatic acid and ipecacuanha, the acid serving, I fancy, to give tone to the relaxed vessels which have yielded the blood.

The subsequent treatment of these cases requires the greatest care, and may be rewarded with brilliant results; for they are cases in which the disease is often in the smallest sense constitutional, and therefore in which recovery is always to be hoped for, while in no kind of condition is neglect attended with more unfortunate results than in hæmoptysis. I shall have a few more special remarks to make on the treatment of pulmonary hæmorrhage in connexion with the subject of Recurrent Hæmoptysis.

The prophylactic treatment is of much importance

[25] Drs. Baümler's and Weber's cases, recorded in the *Clinical Transactions*, vol. ii,, in which, however, they take a different view as to the source of the hæmorrhage, are most instructive in pointing out the value of the thermometer as a guide in the management of cases of hæmoptysis.

when we have any suspicion of a tendency to pulmonary hyperæmia, especially in young girls before menstruation is thoroughly established, or if it be irregular. Violent exercise of any kind should be strictly forbidden, the underclothing should be of flannel throughout, and the air of the bedroom should be warmed.

CHAPTER VII.

Recurrent Hæmoptysis: Illustrative Case—main features of the disease; repeated copious hæmoptysis; chronicity of pulmonary disease—Pathology: slowly forming or old excavation, not necessarily tubercular; frequent absence of secondary fever; modes of arrest of hæmorrhage—Treatment, Prophylaxis—Both the forms of hæmoptysis described rare; significance of true hæmoptysis but little weakened by modern research.

The following very typical example of recurrent hæmoptysis has now been under my observation for nearly five years and I will summarise my notes which have extended over the whole of this time, as briefly as possible:—

Thomas W., aged at the present date, 31, described as a fitter, first came under my notice at the Brompton Hospital, in May, 1867. He had been ailing for some years with occasional cough, and had been three years previously under the care of Dr. Stone, at the same Hospital. He complained of pain in the chest and bad cough, but with, he said, no expectoration; he had had streaky hæmoptysis several times. He was doubtful whether he had got thinner; the appetite and digestion were good, the bowels regular, and the pulse slow. The only history of hereditary predisposition consisted in his father, an intemperate man, having died of consumption at the age of 44. The patient himself has always been a tolerably steady man of very active habits. He is

very intelligent, of sanguine temperament, clear complexion, of medium height, and slight, though robust build. A striking feature about him, and worthy of note, is his extreme excitability—an almost superfluous energy with which he is gifted, which leads him to do everything with exaggerated effort.

At the date of his first attendance there was present at the left apex some dulness, with a few clicks.

On June 29th he reported having expectorated on the previous day a considerable quantity of blood, and, as he was still spitting some, he was ordered gallic acid powders.

On August 3rd he spat more blood, and *a note is entered of the existence of a small vomica at the left apex.*

8th.—"Hæmoptysis one pint this morning;" repeated powders, and ordered mist. acid. sulph. co.

On the 10th the hæmoptysis continued in a less degree and the breath was freer; he was ordered counter-irritation to the left apex and to continue the medicine.

On the 17th croton oil liniment was applied to the left apex. At this date, regarding the continuance of the hæmoptysis and its repeated occurrence at intervals together with the absence of any corresponding progress in the pulmonary physical signs, which were still limited to the summit of the left lung, I was first led to suspect the existence of a small aneurism of a pulmonary vessel there.

On the 24th, however, the hæmoptysis had almost ceased, and as the patient was emaciating, mineral acid and bark with small doses of oil were pre-

scribed, and a linctus for the cough which was troublesome. With the exception of a very trivial attack he had no return of hæmoptysis, and ceased attending the Hospital, greatly improved in health, at Christmas of the same year.

He returned again in October, 1868, having continued, as he expressed it, "well" and at work until a fortnight previously. He had now slight cough and had expectorated some blood but not so much as on previous occasions.

At this date there was "dulness on the left side anteriorly to the mamma, with high-pitched bronchial breath-sound, pectoriloquy, and cavernous cough; sounds very dry; some crepitus at angle of left scapula." The oil was repeated, and an alkaline bitter ordered with small doses of iodide of potassium. He again improved, having only one slight attack of hæmoptysis in November; and at Christmas, having an in-patient letter, was admitted into the Hospital under the care of Dr. Pollock, who confirmed the accuracy of the signs as above described. He only remained in a month, however, during which time I saw him on several occasions, and cautioned him against displaying so much energy in doing the most trivial thing and coughing with such unnecessary violence. He had no appreciable expectoration, and left the Hospital feeling well.

I did not see him again until August, 1870, when, having remained quite well and at work until the previous Wednesday, he expectorated half an ounce of blood. The physical signs were still limited to

the left apex, where there was dulness, bronchial respiration, crepitus, and friction (creaking pleura), and a whiffling murmur, systolic, audible in infra-clavicular region, continuous from subclavian not from pulmonary arterial region (and no doubt conducted subclavian murmur).

He ceased attendance in October, and continued pretty well until March 4th, 1871, when he again attended with hæmoptysis, and was seen by my colleague Dr. C. T. Williams in my absence, who ordered gallic acid immediately and directed him to send in three days' time. With his usual imprudence he attended personally on March 8th, having come from Battersea, though still spitting blood freely, and brought up a considerable quantity in the out-patient room. I prescribed gr. xx. of ergot every two hours for twelve doses, and ordered emp. lyttæ, four inches by four, to the left infra-clavicular region. This attack proved the most prolonged and desperate one he had yet had, and nearly terminated fatally.

March 11th.—Wife attended; hæmoptysis still continues; brought up half a pint of blood this morning at 2 a.m. Ordered six ℥ss. powders of gallic acid, one to be taken directly, and one-third (gr. x.) every two hours, with morphia linctus to allay the cough, and a brisk purge.

15th.—Hæmoptysis continues in a less degree. A mixture containing nitro-muriatic acid with glycerine and ipecacuanha (which I have found of great service at the close of an attack of hæmoptysis) ordered, and some more powders and purgative.

On the 22nd he had had three more attacks of copious hæmoptysis three days previously, and was extremely exhausted by the continual loss of blood. Sulphate of iron and alum were prescribed.

29th.—"Hæmoptysis half a pint yesterday; same amount to-day." 3ss. doses of gallic acid ordered every four hours for six doses.

On the 30th, mist. acid. sulph. co. 3tis. horis; pil. plumb. c. opio gr. v., nocte maneque; iodine paint under left clavicle. From this date the violence of the attacks much abated—I should imagine rather from lack of blood-supply than from the efficacy of the remedies used, which, however, were steadily continued until April 13, when he had had no hæmoptysis in quantity for a week.

On April 20th, some small doses of cod-liver oil were ordered and the acid ipecacuanha mixture with a little morphia. He had no more hæmoptysis after this and again attended personally, though with great difficulty from his extreme weakness, on May 4th. At this date there was noted at the left apex "retraction of lung, dulness, cavernous respiration, and rhonchus (slight)." Posteriorly there was "diffused crepitation, with some defective resonance." This was the first occasion on which the lung had appeared to suffer from the effects of the hæmoptysis.

The cough was troublesome, especially in the morning, and on the 11th he was ordered ether and ammonia expectorant in the morning, lest his violent and unaided efforts at expectorating should lead to a reopening of the broken arterial branch or possible

aneurism, which seemed to have been the only conceivable source of such profuse and repeated hæmorrhage. It was extraordinary to note the rapidity with which the patient regained flesh, strength, and colour, though butcher's meat was only allowed every other day, stimulants were cut off, and abundance of milk alone permitted. He continued to take mineral acids and oil 3j. a day. He did not at all approve of this diet, but from previous experience of his rapid blood-making qualities I was convinced that a more generous regimen would have led to a return of the hæmorrhage.

On June 29, having only (on the 8th) had one comparatively slight attack of hæmoptysis, the physical signs showed enlargement of the right lung the margin of which reached across the median line; still some irritative bronchitis at the left base indicated by diffused submucous râles. Frictions with compound iodine ointment ordered.

Beyond an occasional tinge of the morning expectoration he has had no more hæmoptysis up to the present time, (December 1871) and has returned almost to his usual health, though the breath is shorter. Since June he has taken no oil; some digitalis was added to his mixture for a few weeks; and the diet has continued restricted, though less so of late.

This case, the great length of which demands some apology to my readers, is one of extreme interest to me, as exemplifying well what I believe to be the main features of recurrent hæmoptysis, viz.:—1. Re-

peated copious hæmorrhages obviously arising from disease localised at one portion of the lung. 2. Pulmonary disease chronic in its course and but little influenced directly by the hæmorrhage. The hæmoptysis, though it may prove directly fatal, is accompanied by no severe fever or secondary pneumonia, and from it the patient frequently makes a speedy recovery.

The pathological condition common, I believe, to all these cases of recurrent hæmoptysis is that of a slowly forming cavity, or one formed by a very localised process of an active character, in the walls of which pulmonary vessels still patent are exposed. It will be observed that the case above described did not begin with hæmoptysis; the man had had some dry cough and occasional streaky hæmoptysis for some years previously, and a few days after the first considerable hæmoptysis a vomica was found at the left apex, where some two months previously there was consolidation and softening.

But the vomica, which yields the blood, need not be of "tubercular" origin—*e.g.*, a soldier has been under my care at Brompton for the last fourteen months, who, in March, 1869, while blowing the clarionet, in India, was seized with hæmoptysis to the amount of about a quarter of a pint, which did not quite cease for about a week. A month or six weeks later, after having suffered for four or five days from severe pain and oppression in the right infra-mammary region, he suddenly brought up about a pint of "corruption" and some more blood, and since that time he has had

hæmoptysis every few weeks. Since he has been under my notice, the attacks of hæmoptysis have usually been preceded by severe oppressive pain in right mammary region. The pulmonary disease is mainly at the base or rather the middle of the right lung, there being scattered moist crepitation over the lung, with dulness, most marked at the base. Within the angle of the scapula, and also at the corresponding point in front, opposite the fourth rib, tubular respiration with some large click is heard.

This case appears, then, to have begun with abscess in the lung—whether secondary to pulmonary apoplexy or not it would be difficult to say—which has probably left behind a chronic deep-seated cavity.[26]

The patient, Thomas W., whose case is above related, has never appeared to be febrile, and during the short time he was in the Hospital, on one occasion when he had hæmoptysis, though to a much less degree than usual—viz., one ounce—my friend, Mr. Bartlett, the Assistant Medical Officer, found his temperature to be normal.

In two other men now under my care as out-patients, who, while in the Hospital, suffered from severe hæmoptysis, this same gentleman found no elevation of temperature—*e.g.*, one case, James A., a

[26] He was discharged from the army, from Netley Hospital, with "abscess of the liver and phthisis."

Added note: April, 1872.—No copious hæmoptysis for three months but two or three ounces of current jelly-like expectoration daily. No evidence of malignant disease. Signs of cavity very obscure.

stonemason (who had previously been under my care for some time with a vomica at the base of the lung, and induration at the apex, and who had several times had copious hæmoptysis while in the Hospital under Dr. Quain) on November 14th brought up half a pint of blood at 10 a.m.

At 7.30 repeated half a pint, temperature 97·8°; 10 p.m. four ounces, temperature 99·2°.

15th.—10 a.m., temperature 98·8°.

5 p.m., temperature 98·2°, hæmoptysis three-quarters of a pint half an hour before.

7 p.m. and 9 p.m., temperature 98·2°. He had no more hæmoptysis, and the temperature, taken twice daily by Mr. Bartlett, up to the 21st, never rose above 98·4°. The physical signs were not altered, and the patient rapidly improved in general health.

The danger in these cases is from the abundance of the hæmorrhage, which in a great number is the cause of immediate death on the first occasion. It is surprising this should not be so in almost all,[27] and nothing is more striking than the recovery of some patients from what appears to be the most hopelessly profuse hæmoptysis—Nature apparently seizing the

[27] In cases of fatal hæmoptysis, with very few exceptions, aneurism or erosion of a branch of the pulmonary artery has been found post-mortem at the Brompton Hospital. In some of these cases there had been previous attacks of hæmoptysis of the same character as the fatal one, while in several of them other vessels were found broken across, and occluded only at the very points of the fragments. Other observers (notably Dr. Rasmussen) have insisted on the frequency of pulmonary aneurisms in fatal hæmoptysis.

moment when, from faintness, the blood is at a standstill to heal the breach by the formation of a coagulum. Hence the importance of withholding all stimulants till the latest moment.

Rokitansky refers to another mode of arrest of the hæmorrhage from a large vessel in a cavity—viz., by the cavity becoming blocked by coagulum, which thus compresses the vessel. I have seen an instance, *post-mortem*, in which the apex of the right lung was converted into a blood-cyst, quite closed, as large as a lemon, which had been produced by hæmorrhage into a cavity,

In the treatment of the form of hæmoptysis now under consideration, besides the general principles of absolute muscular rest, etc., before referred to, we must be more diligent with astringents and remedies which control the heart's action and allay cough: *ergot* acting upon the muscular walls of the arteries, *digitalis* diminishing the frequency of the heart's action, and *opium* lessening excitement and allaying cough, are of the greatest value.

Ipecacuanha emetics, admissible in certain cases of primary hæmoptysis, would be certainly harmful in these. Our object is to allow the blood to coagulate at the seat of rupture, and faintness short of actual syncope should be encouraged, rather than prevented by stimulants. Nauseant remedies, however, from their relaxing effects on the vessels, are inadmissible. Interrupted cold applications to the chest may be tried in these cases more usefully, I think, than in those in which the hæmorrhage is capillary.

With reference to prophylactic treatment, patients the subjects of phthisis, particularly with chronic cavities, should be cautioned against muscular efforts, such as running upstairs or walking fast. The experiments of Colin[28] show that on exertion the pressure of blood in the pulmonary artery increases in greater ratio than that in the aorta. In those patients, too, who are gifted with rapid blood-making powers, and who pick up flesh with great rapidity after hæmoptysis, a timely partial abstention from butchers' meat, and the complete withdrawal of stimulants, may ward off or postpone the next attack.

I am inclined to think from my observation of the above related, and of some other cases, that after a greater or less number of recurrences of the hæmoptysis the vessel yielding the blood may become obliterated. There is perhaps, however, a greater liability to the exposure and dilitation of a fresh vessel in such cases as have once borne the recurrent hæmoptysical character.

Having dwelt at considerable length upon two classes of cases in which copious hæmoptysis is a very prominent and important symptom—in the one class because it is the first symptom, which, though rarely directly fatal, is yet often attended with secondary results endangering the life of the patient; in the other, because the hæmorrhage is always extremely dangerous, and may at any time prove directly fatal, while its secondary results are, as a rule, trivial, and but slightly influence the progress

[28] *Compte-Rendus*, p. 759, 1864.

of the disease, which is usually one of the very chronic forms of phthisis—we must not omit to point out, for fear of misconception, that the cases which constitute these two classes are few and exceptional. Hæmoptysis, as a rule, whether very slight or moderately copious, is a merely casual, though very important symptom, in the course of phthisis.

I must, in conclusion, further state it as my firm conviction, that, take hæmoptysis from what point of view we may, its *genuine* occurrence in any degree beyond a mere streak in the expectoration is a symptom the gravity of which, in the enormous majority of cases, has not been in the least exaggerated by the much-abused Laennec and others of equal experience—*i.e.*, so far as it is significant of positive disease.[29] Putting the matter in the most practical form, I presume there are very few Physicians who would venture to consider a candidate for life assurance as a "good life" who had the history of a distinct attack of hæmoptysis. But, on the other hand, by fully recognising the gravity of this symptom, and by, at the same time, bearing in mind the often-proved results attained by due precautions and improved treatment, we may justly give that very decided and conditionally hopeful advice which is most likely to meet with obedience and to be followed by corresponding success.

[29] Having redistributed the many diseases collected together by Laennec under his comprehensive term "tubercle," we must beware lest, in our criticism of the symptoms attached by Laennec to the disease Tubercle, we do not make sufficient allowance for our very restricted use of that term.

CHAPTER VIII.

Alveolar Catarrh before spoken of may set up local tuberculisation; why disease tubercular, not simple chronic inflammation; seat; progress independent of pneumonia; How different from miliary tubercle; mode of extension by continuous growth; clinical differences—Chronic Tubercular Phthisis the best clinical name for this disease—Relationship of local tuberculisation to local, miliary, and general tuberculosis one of degree or intensity as regards infective origin or specific constitutional nature—Distinctive characters of Chronic Tubercular Phthisis; prognosis—Sketch of a case of acute Tuberculo-pneumonic Phthisis; distinguishing characters from Acute Tuberculosis and Acute Pneumonic Phthisis—General rule as to prognosis; necessity of watching the signs of fever as well as physical signs—Table representing chief varieties of phthisis, with their distinguishing characters.

In speaking of alveolar catarrh, it was remarked that, as in some cases it may proceed to catarrhal pneumonia of varying degrees of intensity, in which the pneumonic process with its intra-alveolar products gives the prevailing character to the disease, so in other cases the lymphatic or adenoid structures which so largely enter into the formation of the lung stroma, may, under the same primary catarrhal irritation, take on the more prominent growth: and great thickening of the alveoli, grey induration in which some individual granules of tubercle may or may not be distinguishable by the unaided eye, is the result—in fact, we have a local pulmonary *tuberculisation*, of slower and more insidiously destructive progress

than caseous pneumonia, so far as the lung is concerned, but more obstinately and continuously progressive, more prone to be succeeded by early implication of the other lung, supposing both are not from the first implicated, more quickly followed (sometimes even preceded) by disease in other organs particularly the larynx and intestines; and, in short, though a chronic or subacute disease, yet one of more early average termination than the corresponding pneumonic forms of phthisis.

It will no doubt be said by those who only admit the existence of tubercle in the discrete or disseminately grouped miliary forms that this *local tuberculisation* is no tuberculisation at all, that it is merely chronic inflammation. If so, it is an interstitial inflammation of a very special kind, that in typical cases spreads through the lung from apex to base, with a well-defined grey advancing margin, immediately beyond which the highly vascular but crepitant lung-tissue presents a striking contrast to it. On examining, however, more minutely with a lens, the alveolar walls are found considerably thickened to some little distance (perhaps half an inch) beyond the defined margin, though the alveolar spaces are not occupied with catarrhal cells—at least, not uniformly so, or to any material extent. I am not aware of any mere inflammation at all analogous to this in its invasive characters. It most resembles lupus of the cutaneous surface, which, I presume, no one would venture to describe or treat as merely inflammatory.

On the other hand, this form of tubercle (as I think

it must be considered) differs from miliary tuberculosis pathologically by its primarily attacking one portion of one or both lungs (almost always the apex), and spreading therefrom, not by the dissemination of miliary tubercles far beyond the margin of advance, but by a continuous growth involving the destruction and subsequent excavation of the affected tissue. Clinically, the peculiarly insidious origin and progress of the disease with the gradually increasing *malaise* and anæmia, nocturnal cough, irregular fever, and the physical signs at first very obscure at one apex gradually increasing and developing at the other, are in accordance with its pathology, and distinguish it also from the still more severe miliary form of tubercle. "Chronic tubercular phthisis" seems the best clinical name for this variety, of which the pathological process is, as above stated, best represented by the term pulmonary tuberculisation.

Miliary tuberculosis, though not differing essentially in anatomical characters from the infiltrated form of tubercle above spoken of under the terms pulmonary tuberculisation, grey induration, etc., appears to differ somewhat in its mode of origin, which has been proved by late experiments and is now generally admitted to be *infective*. It may be, however, that we should better appreciate the relationship, so striking anatomically, which exists between these two forms of tubercle if we were to bear in mind that as there are two degrees of miliary tuberculosis—viz., first, that in which the tubercles are widely dispersed

through many organs (infection through vessels?), second, that in which the granules are merely sprinkled around some old cheesy disease or induration (infection through lymphatics?)—so in a similar fashion a still less intense degree of infective power, amounting only to a specific irritation, may lead to the production of tubercle more limited still in extent, affecting only the parts contiguous to the infecting agent. Further, with a certain predisposition, it seems clear that both in animals and men miliary tuberculosis may arise from the mere irritation of a catarrh or local lesion, the specific quality being yielded by the constitutional peculiarity of the patient. And it may, correspondingly, be a question of degree of constitutional aptitude, whether the disease shall be as local as the irritation giving rise to it, or shall extend only by contiguity from the point of origin, or whether it shall at once take on the more virulent diffused miliary form. These observations are, I admit, speculative in some degree, but they surely find some support in clinical experience, and more from recent anatomical inquiries.

That some constitutional aptitude, hereditary or acquired, is necessary for the occurrence of tuberculosis is proved by its so often failing to arise in cases of chronic phthisis and scrofulosis, in which for months and years inflammatory products in every stage of degeneration have existed as apparently efficient sources of infection; while in other cases the pathologist must explore with great diligence the body of one dead of tuberculosis to discover the required *cheesy mass*.

Returning now to the consideration of chronic tubercular phthisis, it is very difficult to depict in writing a case with sufficient accuracy to bring out those somewhat minute differences, the accumulation of which build up the distinction between the cases of catarrhal pneumonic phthisis already described, and those of chronic pulmonary tuberculisation to the pathology of which we have referred. And indeed it has already been pointed out that alveolar catarrh is really the first stage of both these diseases—or, rather, it is the true first stage of the one (pneumonic phthisis), and the determining cause of the other (chronic tubercular phthisis).

It will, then, perhaps be most instructive to enumerate those symptoms and signs the presence of which will warrant us in regarding the case as one of tubercular phthisis—as one, at all events, in which tubercle is the prevailing lesion; for it has been before stated that chronic tubercle is rarely wholly unmixed with other inflammatory products.

So far as temperature is concerned, there is nothing at present known characteristic of chronic tubercle. It is at times elevated, during which periods there are fresh accessions of disease, and the non-febrile intervals are of varying duration. In this respect the disease presents no important difference, so far as I have been able to observe, from the chronic pneumonic forms of phthisis. The physical signs are more characteristic. The obscure signs of alveolar catarrh do not give place to the well-marked dulness and coarse crepitation or crackling of catarrhal pneumo-

nia, but to continued weakness of respiratory murmur, with impaired expansion or actual flattening, while moist sounds may be altogether absent, or one or two dry crackles may be elicited on cough. The percussion-note becomes hardened, and we may suddenly be surprised by the development (having omitted to examine the patient for a week or two) of some feeble, blowing respiration, of hollow quality, still very dry, which increases in the same obscure way until an unmistakeable cavity is present. This formation of a cavity by a process of dry crumbling is very characteristic of the typical form of pulmonary tuberculisation.

Huskiness of voice, or actual aphonia, is commonly one of the early symptoms in this variety of consumption, and is, I think, characteristic of tubercle. The huskiness may clear off, but the voice remains more or less altered permanently in quality. Too hasty a diagnosis must not, however, be made from this symptom, lest a grave prognosis be rested upon a simple laryngeal catarrh. The digestive organs are early affected; the tongue presents a scanty white fur on a very red ground, with prominent red papillæ, an appearance which is very significant of intestinal lesion, still more so if the fur clears off in patches leaving raw-looking glazed surfaces, and the symptoms characteristic of this lesion—alternating diarrhœa and constipation, with colicky pains, especially after food—soon appear.

Patients the subject of this form of phthisis are usually of slender figures and good features. Among

them are those more interesting examples of consumption or decline that novelists prefer to describe. This variety is, however, much more uncommon than the pneumonic forms of phthisis.

As to prognosis, these cases admit of considerable temporary relief, and may appear to do well for the first few months. The physical signs progress however, and I think the duration may be pretty safely reckoned as within two years of the first appearance of definite signs. The intestinal and laryngeal complications cause great distress towards the last, and hasten the fatal termination.

The following sketch illustrates the phenomena characteristic of *acute tuberculo-pneumonic phthisis—i.e.*, a case of pulmonary tuberculosis, in which the tubercular granulations and groups of granulations are attended with much pneumonia, which latter is the main element destructive to the lung; while the former appears to stamp the disease with its peculiar adynamic characters, its continued fever, and determined progress without check to a fatal termination.

A woman, aged 31, was admitted into Dr. Cotton's ward at the Brompton Hospital in October last, and seen by me in his temporary absence. She had had "inflammation of the lungs" (there was no sign of old pneumonia *post-mortem*) two years ago, but had suffered from more or less cough, with frothy expectoration, for three years. Four weeks ago, however, she expectorated a small quantity, two teaspoonfuls of blood, and the sputa continued to be tinged with blood for five days. She had since suffered from night-

sweats, emaciation, cough, and pain in the side and between the shoulders, of which symptoms she complained on admission. The pulse was 112; the tongue furred; catamenia regular. She knew of no family predisposition to phthisis. The physical signs on admission were—harshness at the right apex, with subcrepitant rhonchus; at the left, jerking respiration and prolonged expiration. She lost rapidly in weight however, losing $3\frac{1}{2}$ lbs. between October 26th and November 14th.

On November 8th the physical signs were noted as unchanged. On the 21st she was much worse, with a red tremulous tongue, a rapid pulse, great breathlessness, and much heat of skin. She was sitting up, but could with difficulty stand from the trembling of her limbs and weakness. Subcrepitant rhonchus was found diffused throughout the right side behind, with some defect in resonance not amounting to dulness. At my request Mr. Garton, clinical assistant, took the temperature night and morning from this date. During this time—twenty-nine days (five days the temperature was not taken)—the maximum morning temperature was 103°, average 101·6°; maximum evening temperature 104°, average 102·3°; difference between the average morning and evening temperature ·7°. On the frequent occasions when I saw the patient in the middle of the day the skin was uniformly hot, and the pulse very rapid—usually about 120. Meanwhile the pulmonary physical signs advanced, the crepitation became more abundant, and extended through both lungs. There were signs

of breaking down at the right apex, though the presence of a cavity could not be with certainty ascertained. On December 19th there was present "diffused blowing respiration, with sonorous rhonchus and scattered crepitation more abundant at the bases, with some dulness; high temperature, and much dyspnœa." On December 15th the patient began to be troubled with diarrhœa, which continued more or less to the last. The emaciation and loss of power rapidly increased, the smooth red tongue became white with aphthous patches, and she gradually sank, having never evinced, however, any delirium or other morbid brain symptoms.

The *continued* fever, the great and early prostration, the diffused crepitation heard over the lungs, without any defined dulness, rendered the diagnosis of Pulmonary Tuberculosis being the prevailing lesion a tolerably certain one, while the absence of that degree of utter prostration with occasional muttering delirium, and the early presence of decided pulmonary signs, prevented one from regarding the case as one of acute general tuberculosis. There was, however, but little satisfaction to be derived from this reflection, for the prognosis was, so far as present knowledge could decide, inevitably fatal.

Post-mortem the lungs were found studded with racemose groups of tubercle surrounded by ill-defined areas of soft catarrhal pneumonia in active process of formation and degenerative softening; the right apex was breaking up into small cavities. There was no miliary tubercle on the pleural surfaces.

It will be observed that in this case there was a history of "inflammation of the lungs" two years before her fatal attack, and of three years' slight occasional cough and frothy expectoration. More or less long-continued bronchial catarrh is the very common precursor of pulmonary tuberculosis. It has before been observed that catarrh long limited to the bronchial mucous membrane may extend to the alveoli, giving rise to simple alveolar catarrh, catarrhal pneumonia or tuberculosis, as something we do not yet understand shall determine.

There are, it is true, certain cases of acute pneumonic phthisis, "galloping consumption," in which the broncho-pneumonia is so diffused as to render the diagnosis from acute tubercle very difficult; this matters little, however, for the prognosis is identical in the two kinds of cases. But the acute pneumonic phthisis, as a rule, proceeds by consolidation rapidly extending from one apex, or more rarely one base with increasing dulness, and abundant metallic crepitant sounds soon changing to gurgling. In such cases at the moment when the condition of the patient seems almost immediately hopeless the extension of the disease may stop, the temperature (more intermittent than in tubercle) may fall, and the patient may again begin to pick up strength while yet auscultation informs us that breaking down of the products of inflammation is still proceeding. Such cases point out what will generally be found a true guide to prognosis—viz., that the more prominently the symptoms of acute phthisis are attended with *defined*

auscultatory and percussion signs, the more hope is there that the active disease may subside and the patient recover from the attack with so much damaged lung—in fact, the more certainly is the disease to be acute *pneumonic* and not acute *tuberculo-pneumonic* phthisis, or acute *tuberculosis*. Although however it is true that the disease which we have watched progressing with such fearful rapidity, as to lead us to anticipate death within a few days, may stop short, yet the amount of damage its active extension has occasioned may be too great and the attendant systemic shock too severe for the patient ever to rally; he succumbs at last to the exhausting influence of prolonged hectic after having lingered it may be for months.

It is imperative to watch closely, not the physical signs alone, for they will mislead us, but the signs of fever (temperature, tongue, and pulse), to learn the moment when the spread of the disease stops, and when tonic medicines, food, oil, etc., will have their due effect. Did we judge by the stethoscope alone, ws should find softening and excavation signs increasing long after this period of true pause, because the products of the past inflammation must run through their normal stages of caseation, or liquefaction and removal, and these mere physical processes largely contribute to the production of auscultatory signs. Nor is it easy by physical diagnosis alone to say when the limit of the extension of the disease has been reached.

I may, perhaps, usefully summarise in a tabular

form the views advocated in the preceding chapters as the distinguishing characters of the principal varieties of phthisis, which views have been acquired from the observation of a very large number of cases and the making and supervision of many post-mortem examinations at the Brompton Hospital.

Table representing the chief Varieties of Phthisis, with their distinguishing Anatomical Characters.

Primary Lesion.	Pathological Nature and Tendencies.	Anatomical Characters.	Variety of Phthisis.
Alveolar catarrh ..	Terminates in recovery, or proceeds to catarrhal pneumonia.	Large cells of epithelial type, more or less blocking alveoli; greater or less degree of implication of alveolar wall, and proliferation of its elements.	Catarrhal pneumonic ph. (*progress:* acute, chronic, or intermitting).
Alveolar catarrhal pneumonia.	1st degree terminates in resolution and recovery, or alveolar collapse. 2nd degree terminates in caseation, softening, excavation, or induration from thickening and agglutination of alveoli. 3rd degree terminates in simultaneous degeneration and softening of alveolor wall and contents; ulcerative destruction.		
Pulmonary capillary hæmorrhage	(*a*) Copious hæmoptysis may be coincident with the commencement of, and may perhaps determine, *pneumonic phthisis*, giving rise at least to secondary pneumonia.	Coagulated blood blocking alveoli, with surrounding inflammatory changes.	(*a*) Hæmorrhagic ph.
Pulmonary tuberculosis.	Of primary irritative origin, or part of general tuberculosis, or supervening upon caseous pneumonia or chronic tubercle 1. Pure, terminates in death without breaking down of lung. 2. When mixed with pneumonia, caseation softening and ulcerative destruction of tissue.	Nuclear growth of adenoid kind occurring as miliary grey granulations—(1) disseminated singly, or (2) in racemose groups, often associated with catarrhal pneumonia.	1. (*a*) 2. Acute tubercular or tuberculo - pneumonic ph. (*progress:* acute, fatal).
Pulmonary tuberculisation.	3 (*a*) Irritative local grey induration, secondary to caseous pneumonia. (*b*) May be the primary disease progressively invading lung.	Coalescing granulations, proceeding to grey induration from fibro-nuclear development; degeneration.	3. (*b*) Chronic tubercular ph. (*progress:* subacute or chronic, continuous.)
Pulmonary fibrosis	Present more or less in association with the chronic forms of the above varieties; never primary; when so marked in unilateral cases as to give a special clinical character to the disease is conveniently named separately.	Contractile fibroid cicatricial tissue, the results of proliferation of elements of fibrous stroma of lung, including vessel sheaths and lymphatic tissues, mingled with products of primary disease.	Fibroid ph. (*progress:* very chronic; with sometimes long quiescence).
Pulmonary arterial hæmorrhage.	(*β*) Copious hæmoptysis may be a marked and recurring symptom in the more chronic indurative forms of phthisis, attended with quiescent or slowly forming cavities.	Aneurismal dilatation, or erosion of vessels within cavities.	Ph. with recurrent hæmoptysis.

(a) Acute tuberculosis cannot be regarded as a variety of phthisis, though it frequently supervenes as a fatal complication of the disease.

NEW BOOKS

Published and Sold by

H. K. LEWIS, 136 GOWER STREET LONDON.

A Treatise on Hæmophilia sometimes called the Hereditary Hæmorrhagic Diathesis. By J. WICKHAM LEGG, M.D. Fcap. 4to, 7s 6d

A Text Book of Practical Medicine, with particular reference to Physiology and Pathological Anatomy. By FELIX von NIEMEYER. Translated from the Eighth German Edition, by special permission of the Author, by GEORGE H. HUMPHREYS, M.D., and CHARLES E. HACKLEY, M.D. 2 vols. large 8vo, 36s.

"The publication of these volumes brings within the reach of the busy practitioner, the student, and the teacher in our schools, one of the best text-books in any language. As the translators say, 'Professor Niemeyer's volumes present a concise and well-digested epitome of the results of ten years of carefully rendered clinical observation by the most illustrious medical authorities of Europe,' elaborated, we may add, by a most able teacher and an experienced physician. . . . . Not the least valuable part of the book will be found in the therapeutical sections; and the numerous formulæ, which are given most clearly, will prove a boon to many a hardworked practitioner."—*Brit. and For. Med.-Chir. Review.*

"The task of a reviewer is an easy and glorious one in noticing such a work as this. It is at once comprehensive and concise; it is characterised by clearness and originality and it differs from many German works on Medicine in the sagacious appreciation of the value of therapeutics which is manifested by the author....We can cordially recommend the work as one from the perusal of which every physician may derive profit, which will be found replete with information of direct practical value, and which is instinct with a spirit of candid and earnest inquiry, which is worthy of all praise. The translation on the whole, is very creditably performed, and the volumes are handsomely brought out."—*Dublin Quarterly Journal of Medical Science.*

A Guide to the Examination of the Urine; intended chiefly for Clinical Clerks and Students. By J. WICKHAM LEGG, M.D. Third Edit., fcap. 8vo, cloth, 2s 6d.

Notices of Second Edition.

"We are glad to welcome this little work....Just what was wanted."—*Brit. Med. Journal.*

"This little work forms a concise guide to the complete 'clinical' examination of the urine....This work is a very handy one, and we recommend the student to make it his companion in the wards of the hospital."—*Lancet.*

A Hand Book of Therapeutics. By

SYDNEY RINGER, M.D., Professor of Therapeutics in University College. New Edition, thoroughly revised. Small 8vo, 10s 6d.

"As we have said before, Dr. Ringer has the field all to himself, his book being the only one in the English language in which a serious attempt is made by the author to speak in his own name on the general subject of Therapeutics. The able and useful work of Dr. Waring is mainly a compilation, and scarcely comes into the same category with Dr. Ringer's book; and certainly, the latter author need fear no competition from the mere chipps and sawdust of therapeutics which are served up at the end of botanical and chemical descriptions of drugs in the ordinary treatises on Materia Medica....It contains a large amount of material, a good deal of it original, to which English students had no access, and there is this very great value in anything that Dr. Ringer reports on his own authority, that we instinctively feel sure that the observations have been made with the greatest care, and reported with anxious fidelity."—*Practitioner*, Feb. 1872.

"This treatise on Therapeutics at once assumed the first position among kindred works on the subject in the English language. . . . . In conclusion, we have great pleasure in recommending this treatise."—*Brit. and For. Med.-Chir. Review*, January, 1872.

"On the whole, we can speak very highly of the volume in its present form, as a most useful and handy book to the practitioner and student."—*Lancet*, Nov. 25th, 1871.

General Surgical Pathology and Therapeu-

tics, in fifty Lectures. A text-book for Students and Physicians. By THEODOR BILLROTH. Translated from the fourth German Edition, with special permission of the Author, by C. E. HACKLEY, M.A., M.D. 18s.

"Professor Billroth has long been recognised as one of the most scientific of living Surgeons, his lectures forming one of the most popular text-books of Surgery in the German language. They have already gone through four editions in German, and have been translated into all the chief European languages, English being the last. . . . . The want of a book in the English language presenting in a concise form the views of the German pathologists, has been long felt, and we venture to say that no book could more perfectly supply that want than the present volume."—*Lancet.*

"Eminently satisfactory as a treatise on the principles of Surgery. The historical and pathological groundwork of surgical science is laid down by Billroth with admirable clearness. He shows a perfect grasp of the value of the investigations whose results he sums up, and an excellent appreciation of their bearing on practice. His diagrams of specimens are numerous and valuable .... Destined for a wide popularity amongst surgeons."—*Brit. Med. Journal*, July 1, 1872.

"Very few medical works have had the good fortune to attain, in so short a time, the universal popularity enjoyed by Dr. Billroth's volume on Surgical Pathology; and Dr. Hackley, the able translator, has conferred a boon on the English-reading members of the profession in this country and Great Britain, by placing within their reach the latest views and opinions of one of the most progressive pathologists of the age. Indeed we cannot be too thankful for the accuracy and elegance of the translation, remembering, as we must, how grievously some writers have suffered at the hands of their translators.—*New York Medical Record*, June 1, 1871.

On the Principal Varieties of Pulmonary Consumption, with Practical Comments on Diagnosis, Prognosis and Treatment. By R. D. POWELL, M.D. Lond. Med. 8vo, cloth, 3s 6d

On Abscess and Tumours of the Orbit. By By SPENCER WATSON, Surgeon to the Great Northern and the Central London Ophthalmic Hospitals. 8vo, 6s.

Remarks on Diabetes, Especially in Refer- ence to Treatment. By WILLIAM RICHARDSON, M.A., M.D., M.R.C.P. Lond. Demy 8vo, cloth, 4s 6d.

"Finally, we again recommend this as a book well worthy the attention of the Profession. . . . . As the work of a diabetic, it is sure to contain everything of importance on the subject; as that of a man who has cured himself, it is certain to contain rules and axioms which others may follow."—*Medical Times and Gazette.*

What is Malaria? and why is it most in- tense in Hot Climates? An Explanation of the Nature and Cause of the so-called Marsh Poison, with the Principles to be observed for the preservation of Health in Tropical Climates and Malarious Districts. By C. F. OLDHAM, M.R.C.P.E., M.R.C.S.L., Assistant Surgeon H.M. Indian Forces, Fellow of the Royal Medical and Chirurgical Society, Late in Medical charge of Dalhousie Sanitarium. 8vo, 7s 6d.

"In the preceding abstract we have endeavoured to lay before our readers Dr. Oldham's views as to the cause of diseases which have been hitherto attributed to malaria. There is certainly much in them that is novel to those who are acquainted only with the works of the most popular writers on malarial disease and we certainly cannot refuse him the tribute due to one who has good opportunities of studying the subject, and has made good use of them. We find ourselves, however, not fully convinced by his arguments, although we confess he has done much by them to destroy the wavering faith we reposed in the more generally accepted theory."—*American Journal of Medical Science*, October, 1871.

The Diseases of the Ear: Their Diagnosis and Treatment. By J. TOYNBEE, F.R.S. With a Supplement by JAMES HINTON. Demy 8vo, 8s 6d.

"The work in its new form constitutes a most reliable guide to the treatment of aural diseases, and is without a rival in the English language."—*Lancet.*

*Digitalis: its Action and its Use. An In-*quiry illustrating the Effect of Remedial Agents over Diseased Conditions of the Heart. The Hastings Prize Essay of the British Medical Association for 1870. By J. M. FOTHERGILL, M.D. Small 8vo, cloth, 2s 6d.

"…. If the prize calls forth work so original and so thorough as the present it is a more valuable foundation than such prizes usually are. Dr. Fothergill's essay does not give us the notion of being written to order, but might well have been the spontaneous utterance of a man who had studied a certain question long and closely, and had something to say about it."—*Westminster Review*, Jan. 1872.

"This Essay is one that should be in the library of every Practitioner."—*Edinburgh Medical Journal*, Nov. 1871.

*Electricity in its relation to Practical Medi-*cine. By Dr. MORITZ MEYER. Translated from the third German Edition, with notes and additions by WILLIAM A. HAMMOND, M.D. With illustrations, large 8vo, 500 pages, cloth 18s.

"Meyer's book is well known. . . . . He has himself made no slight original contributions to electro-therapy. Dr. Hammond deserves our hearty thanks for his translation—a boon which is as much for us as for his own countrymen."—*Brit. and For. Med.-Chir. Review.*

A Practical Treatise on the Diseases of Children. By Dr. A. VOGEL. Translated and Edited by H. RAPHAEL, M.D. From the fourth German Edition, illustrated by six lithographic plates, part coloured, large 8vo, 600 pages, 18s.

"The merits of Dr. Vogel's work—which has been translated into three languages, and has attained its fourth edition in less than eight years—are of a high order; the pathology especially is most complete, the deviations peculiar to infantile life being fully explained and an amount of knowledge and anatomical research displayed, which could only have been obtained by opportunities afforded for the accurate study of a large number of cases; indeed the entire work bears evidence of intelligent observation and original thought."—*Dublin Journal of Medical Science*, Jan. 1872.

"Several excellent lithographic plates, and the admirable way in which the volume is got up, add to the attractiveness of Dr. Vogel's work. Dr. Vogel is more a physician than a surgeon; and he is clearly a physician of no mean order, judged either from a pathological or a clinical standpoint. When he treats a subject exhaustively he produces an essay, clear yet elaborate, which inspires the reader with confidence that the author believed himself to have the best possible reasons for all that he says. Of Dr. Vogel, as a medical practitioner, we would only say that he seems to belong to the comparatively small class of physicians who, when they give a medicine, have a clear notion of the effects which they desire it to produce."—*Brit. Med. Journal.*

The Practical Medicine of To-day: Two Addresses delivered before the British Medical Association, and the Epidemiological Society. By Sir W. JENNER, Bart., M.D. Small 8vo, cloth, 1s 6d.

Remarks on Army Surgeons and their Works. By Dr. C. A. GORDON. Demy 8vo, 5s.

On the Restoration of Health. By Dr. T. INMAN, Physician to the Royal Infirmary, Liverpool, &c. Demy 8vo, cloth, 7s 6d.

On the Preservation of Health. By Dr. T. INMAN: Second Edition, Demy 8vo, 5s.

On Diet and Regimen in Sickness and Health, and on the Interdependence and Prevention of Diseases and the Diminution of their Fatality. By Dr. HORACE DOBELL. Fifth Edition (*In the press*).

Cazeaux's Obstetrics: A Theoretical and Practical Treatise on Midwifery including the Diseases of Pregnancy and Parturition. Revised and Annotated by S. TARNIER. Translated from the Seventh French Edition by BULLOCK. Royal 8vo, over 1100 pages. 172 Illustrations. 30s.

"It is unquestionably a work of the highest excellence, rich in information, and perhaps fuller in details than any text-book with which we are acquainted. The author has not merely treated of every question which relates to the business of parturition but he has done so with judgment and ability."—*Brit. Med.-Chir. Review.*

"M. Cazeaux's book is the most complete we have ever seen upon the subject.—*N. A. Med.-Chir. Review.*

On Epilepsy: Anatomico-pathological and Clinical Notes, with original Coloured Plates and Engravings. By M. G. ECHEVERRIA, M.D., Professor of Mental and Nervous Diseases at the Medical College of New York, &c. Large 8vo, 22s.

Disinfectants and How to Use Them. By E. T. Wilson, M.B. Oxon., F.R.C.P. Lond., Assoc. Memb. Metrop. Assoc. Med. Offs. Health, Physsician to the Cheltenham General Hospital and Dispensary. Third Edition. in packets of one dozen, price one shilling.

The importance of Disinfection is now fully understood; there seems however, to be a danger lest an ignorant or an imperfect employment of disinfecting substances may lead to a misplaced feeling of security. The right choice of a disinfectant and the manner of its use are important elements in its successful application. In Dr. Wilson's little card full directions are given as to the strength of the various disinfecting solutions, also how and when to use them. To these are added rules, clear and concise, for the guidance of nurses and others in charge of the sick—with a list of the legal restrictions placed upon infected persons.

The card will be found peculiarly suitable for heads of families, clergymen, and nurses; or for distribution amongst the artisans and tradesmen of our larger towns.

A Practical Treatise on the Diseases of Children. By Drs. Meigs and Pepper. Fourth Edition, roy. 8vo, cloth, cut edges, pp. 920, 26s.

"We have no hesitation in saying that it is a treatise of extreme value and of great practical utility; the authors have indubitably had large experience, and are fully *en rapport* with the present state of knowledge. As a text book and general guide to practitioners as well as a standard work of reference, we know of no other volume which contains more useful or reliable information.—*Dublin Journal of Medical Science*, Jan. 1872.

A Practical Treatise on the Medical and Surgical Uses of Electricity, including Localised and general Electrisation. By Beard and Rockwell. With 102 illustrations. 8vo, cloth, 21s.

Vesico-vaginal Fistula, from Parturition and other Causes. With cases of Recto-vaginal Fistula. By Dr. T. A. Emmet. Large 8vo, 12s.

On Intermittent Malaise. By Dr. H. Adams. 8vo, 1s.

Morbid Conditions of the Throat, in their relation to Pulmonary Consumption. By Dr. S. Scott Alison. Post 8vo, 3s 6d.