Lectures on diseases of the lungs / by James Alexander Lindsay.

CLINICAL LECTURES ON DISEASES OF THE LUNGS

J. A. LINDSAY

SECOND EDITION

LECTURES ON DISEASES OF THE LUNGS

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ON

DISEASES OF THE LUNGS

BY

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THIS VOLUME IS INSCRIBED

TO

SIR R. DOUGLAS POWELL, BART., K.C.V.O.

PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON

AS A TOKEN OF FRIENDSHIP AND ESTEEM

BY

THE AUTHOR

PREFACE TO THE SECOND EDITION

The favourable reception of the first edition of these lectures at home, in America, and in the British Colonies having necessitated a second edition, it has been thought well to alter the form in which they were first given to the medical profession. The present volume is confined to the subject of pulmonary disease, and about half the lectures which it contains are new. The lectures on heart disease contained in the first edition are for the present omitted, as it seemed to the author better to attempt to cover the field of pulmonary disease from the special point of view contemplated by him rather than to embrace a wider field less completely. The reader is requested to observe that these lectures are intended to be supplementary to, rather than a substitute for, the ordinary text-books. They are mainly concerned with questions of practical diagnosis and treatment. Pathological questions are only alluded to incidentally, and with no attempt at complete exposition. The clinical point of view is kept constantly in mind, and the problems discussed are considered in the manner and order, as far as possible, in which they arise in actual practice. Differential diagnosis has been dealt with at length, and it is hoped that no important problem arising under this head has been overlooked. Treatment has also been fully dealt with in the light of the most recent advances. Advantage has been taken of the comparatively flexible lecture form to discuss most of the out-standing problems in connection with pulmonary disease. Numerous correspondents have testified

to the writer that this method of discussion and suggestion, as compared with that of dogmatic exposition necessarily adopted by the systematic manuals, has a considerable degree of practical utility. The reader will find most of his difficulties in this field of medicine considered, but theoretic completeness of description has not been contemplated.

The author desires to return his sincere thanks to numerous correspondents at home and abroad who have favoured him with criticisms and suggestions, of many of which he has gladly availed himself.

He is much indebted to Dr. David Lawson for contributing a section on Radioscopy and Radiography. Dr. Lawson's work in this department is well known to all engaged in this field of research.

J. A. L.

15, College Square East,
Belfast,
August, 1906.

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POSTURE OF THE PATIENT.

In chronic cases, where the patient's strength permits, he should be seated upon a common chair, with the chest fully exposed. A satisfactory examination can also be made with the patient standing, unclothed to the waist. He should be directed to let the arms hang by his sides, straighten the spine, slightly elevate the chin, and open the mouth. He should then be instructed how to breathe, an art which few patients perform correctly without some preliminary drilling. Many patients breathe too shallow; some—especially males—breathe almost entirely with the diaphragm; others, from nervousness, breathe in a jerky and unequal fashion. Many patients make noises with the lips and mouth, which cause difficulties in auscultation. These errors can be rectified by a little patience on the part of the physician, who will find the time thus spent by no means wasted. He may sometimes with advantage give a brief demonstration of the correct mode of breathing. In acute cases, or in presence of much prostration, it will be necessary to examine the patient in bed. The shoulders should be supported by an extra pillow, the head also supported, and the chest fully exposed as before. It cannot be too strongly insisted that no examination of the chest is adequate which does not embrace an

examination of all its parts. We must on no account give an opinion in a pulmonary case until we have fully explored the chest in every part. Female patients, who may at the outset object to this procedure on the score of delicacy, can usually by the exercise of a little tact and firmness be brought to recognise its necessity.

The examination of a young child sometimes involves special difficulties. The child is possibly alarmed by a novel situation, a strange face, or the production of a stethoscope. In such cases the child can be best examined while it sits upon the lap of mother or nurse; its attention may be distracted, and the use of a stethoscope may be avoided by the direct application of the ear to the chest—at all events, as regards the examination of the posterior parts.

The methods of the physical examination of the lungs and pleura are as follows:

A. Inspection.
B. Palpation.
C. Percussion.
D. Auscultation.
E. Mensuration.
F. Radioscopy and radiography.

It will be convenient to take these methods in the above order. Succussion hardly deserves to be ranked as an independent method. It will be considered under Auscultation. The data afforded by one method should be carefully noted before we proceed to the next method.

A. Inspection.

The importance of inspection of the chest is, perhaps, hardly adequately recognised. It is a method of the highest value, and should never be omitted. It often puts us at once on the track of a correct diagnosis.

The best point of view for inspecting the chest is a point on the level of the patient's thorax on the opposite side from

that from which the light falls. The next best point of view is from behind, the observer looking over the patient's shoulders while the latter remains seated. The least satisfactory point of view is from the front. It is impossible to inspect the chest adequately from this point.

THE NORMAL THORAX.—The normal thorax is symmetrical. The arching of the ribs is uniform. The intercostal spaces are visible only towards the lower parts. The sternum is almost straight. The angulus Ludovici is not prominent. The ridge between the second intercostal cartilages may be distinct or indistinct. The antero-posterior diameter of the thorax is somewhat less than the transverse. The scapula lies flat upon the back as the patient stands upright, with the arms hanging down. The clavicles are usually well developed, but owing to muscular development may not be prominent. The supraclavicular and infraclavicular fossæ vary much in depth, but are not usually deep.

While the above rules may be accepted, the student must be prepared for rather wide departures from the anatomical norm as regards the shape of the chest. Many irregularities in the shape of the bony framework of the chest are of no importance, and throw no light upon the state of the lungs. How far these abnormalities are physiological—*i.e.*, part of a development not obviously morbid; how far they are pathological—*i.e.*, the result of disease of bone or other morbid process in early life—it is, I think, often impossible to determine, nor is the point one of much practical importance. The important point for the observer is to recognise that these prominences, callosities, and irregularities may have no relation to the disease for which the patient seeks his advice.

By INSPECTION we seek information regarding—

(*a*) Abnormalities in the condition of the integuments.

(*b*) Abnormalities in the shape of the chest.

(*c*) Abnormalities in the movements of the chest.

(*a*) *Abnormalities in the Condition of the Integuments.*—Among the conditions which may attract our attention in connection with the superficial parts may be mentioned: Œdema, abnormal pulsations, venous turgescence, cyanosis, venous stigmata, malar flush, suffusion of the eyes, sudamina, jaundice, erythema, purpura, clubbing of the fingers, incurvation of the nails, herpes. Many of these conditions have much significance in connection with pulmonary disease. We shall consider them fully in a future lecture.

(*b*) *Abnormalities in the Shape of the Chest.*—Pulmonary disease may give rise to alterations in the shape of the chest in various ways. The most usual cause is some alteration in the conditions of pressure in the thorax, contributory causes being frequently disease of the bony framework of the chest, or, more rarely, some involvement of the nervous system. Frequently, several causes co-exist. The rickety child, with its soft bones, feeble nervous energy, and proneness to bronchitis, is a case in point.

Certain types of chest often associated with definite pathological conditions are well known, and are described in all text-books. It is unnecessary to discuss them minutely in this place. Of these types I may mention the following:*

1. The alar or winged chest, often tubercular.
2. The pigeon-breasted chest, usually rickety.
3. The barrel-shaped chest, usually emphysematous.
4. The boat-shaped chest (*i.e.*, a chest with a hollowing of the upper part in the middle line), usually due to syringomyelia.
5. The funnel-shaped or cobbler's chest (*i.e.*, a chest in which the lower end of the sternum is hollowed like a funnel). Common in cobblers, but sometimes congenital.
6. The kyphotic and scoliotic chest (*i.e.*, the chest modified

* See Sahli, 'Lehrbuch der Klinischen Untersuchungs-Methoden,' Dritte Auflage, p. 15 *et seq.*

by kyphosis or scoliosis), found in connection with curvature of the spine.

Abnormalities in the shape of the chest may take the form either of:

(1) An expansion of the chest, or

(2) A retraction of the chest.

Among the causes which produce an expansion of the chest may be mentioned:—pleural effusion, pneumothorax, enlargement of the liver, enlargements of the spleen, intrathoracic tumour, aneurysm, enlargement of the heart.

In large pleural effusions the affected side is increased in circumference, the intercostal spaces are obliterated, the nipple is distant more than normal from the middle line, the scapula is displaced outwards, the shoulder is raised, the spine is convex towards the affected side.

Among the causes which produce a retraction of the affected side, the chief are:—chronic pleurisy, chronic pneumonia, and chronic tuberculosis. If the retraction reaches a certain degree, the shoulder and clavicle on the affected side are lower than normal, the nipple approaches the middle line, the spine is concave towards the affected side.

Sahli is of opinion that for the detection of these abnormalities in the shape and dimensions of the chest the eye often gives more trustworthy results than the use of the tape or cyrtometer.* This is also my experience, and this conclusion, if sound, still further emphasizes the importance of inspection of the chest in all cases of pulmonary disease.

(*c*) *Abnormalities in the Movements of the Chest.* — The careful investigation of the movements of the chest is of the first importance in diagnosis. Most forms of pulmonary disease involve some interference with the normal expansibility of the chest, or in the frequency, rhythm, and ease of the respiratory movements. If we can satisfy our-

* See Sahli, 'Lehrbuch der Klinischen Untersuchungs-Methoden,' Dritte Auflage, p. 15 *et seq.*

selves that the chest is expanding normally in all its dimensions, that its movements are neither hurried nor slowed, that they are rhythmical and free from pain or discomfort, we have gone far to exclude the presence of any serious form of pulmonary disorder. Further, the degree of interference with the pulmonary movements often gives us valuable information both as to the nature and the extent or gravity of the involvement of the lungs.

The movements of the chest may be altered as regards—

1. Frequency.
2. Extent.
3. Rhythm.

1. An increased frequency of the respiratory movements is characteristic of all acute, and of many chronic, pulmonary diseases. It is also found in most febrile affections, in diseases of the heart, in many nervous diseases, and in other affections.

A diminished frequency of the respiratory movements is found in cerebral hæmorrhage, brain tumour, meningitis, uræmia, occasionally in diabetic coma, in many infections, and in the death-agony.

2. Alterations in the extent of the respiratory movements are frequent, and may give information of great importance.

Immobility of the chest as a whole does not exist, such a condition being incompatible with life. Immobility of one entire side occurs in pneumothorax and in large pleural effusions. Immobility of one or both bases may be due to pleural effusion, pneumonia, pulmonary collapse, or to causes in the abdomen interfering with the descent of the diaphragm. Immobility, partial or complete, of one apical region may be due to apical pneumonia or encysted pleural effusion, but in the great majority of cases it is a sure sign of phthisis.

Bilateral paralysis of the diaphragm has some striking effects. The respirations become hurried, shallow, and

costal in type. The abdominal movements are inverted—*i.e.*, the epigastrium and abdomen generally are retracted on inspiration and protruded on expiration, the costal arch is widened, and on inspiration the lower ribs move too freely.*

LITTEN'S SIGN.†—Litten has drawn attention to the following phenomenon: In normal individuals a furrow can be seen on both sides of the body, chiefly in the axillary line, which becomes distinct a little after the beginning of a forced inspiration between the fifth to the eighth intercostal spaces, and moves downward a distance of from five to six centimetres during inspiration. This furrow is the visible sign of the descent of the diaphragm. It is wanting in well-marked cases of emphysema, and in pleural effusion.

The diaphragmatic movements are also impeded in diaphragmatic pleurisy, and in certain abdominal conditions, such as peritonitis and ruptured gastric ulcer. Both in connection with thoracic and abdominal disease, the beginner would do well to note carefully the character of the diaphragmatic excursion. If it is notably diminished we should inquire for the following conditions:—diaphragmatic pleurisy, peritonitis, paralysis of the diaphragm, hysterical or organic, strychnine poisoning, hydrophobia, tetanus, diaphragmatic hernia.

Recession of the chest, as seen in the supraclavicular fossa, the suprasternal notch, the intercostal spaces, and the epigastrium, is a physical sign of great importance. It suggests some obstacle to the free entry of air into the lungs, with or without pulmonary collapse. In the capillary bronchitis and broncho-pneumonia of children the amount of recession of the chest is one of the best gauges of the gravity of the attack. With much recession life is usually in danger. It shows that air is not entering the lungs adequately.

* West, 'Diseases of the Organs of Respiration,' vol. ii., p. 830.

† Litten, *Deutsche Med. Wochenschrift*, 1892. Hoffmann, 'Nothnagel's Encyclopædia of Practical Medicine,' English edition, article 'Emphysema.'

3. Alterations in the rhythm of the respiratory movements are seen in emphysema and asthma (expiration prolonged), in laryngeal obstruction (inspiration prolonged), and in Cheyne-Stokes breathing, where periods of apnœa alternate with periods of dyspnœa.

It will be convenient to postpone the consideration of these matters until we come to deal with the subject of dyspnœa.

B. Palpation.

Mode of Palpating.—One hand should, as a rule, be used in palpating, and it should be applied successively to different parts of the chest, while the patient pronounces the words 'ninety-nine.' The ulnar edge of the hand, being the most sensitive part, may often be employed with advantage in palpation. The two sides of the chest must in all cases be carefully compared, as there is no fixed standard of conduction of the vocal fremitus, and we cannot draw any safe conclusions until we have satisfied ourselves of its normal condition in the case before us. Vocal fremitus may be well appreciated by laying the ear direct upon the chest.

The vocal fremitus may be entirely absent in normal individuals, especially in women and children with thin, high-pitched voices; but it may also be absent in vigorous and muscular subjects, in whom the thickness of the parietes seems to prevent the conduction of the vibrations to the hand. As a rule, vocal fremitus is more marked the deeper the voice.

The fremitus is more marked upon the right side than upon the left, nc doubt owing to the greater width and straighter course of the right bronchus. The difference is usually quite distinct, especially in the infraclavicular regions, and must be allowed for before any conclusions are drawn. There is an old clinical rule to the effect that, 'If the fremitus be equal on both sides and well marked, suspect a lesion of the left apex. If the fremitus be equal on both

sides and ill marked, suspect a lesion of the right apex.' In the former case we have probably to deal with an increase upon the left side, in the latter case with a decrease upon the right.

Vocal fremitus depends on the conduction of the vibrations from the glottis through the bronchi to the parietes. Hence, it is naturally most marked where the bronchi are large and near the surface—*e.g.*, in the infraclavicular regions; while it is comparatively ill marked where the bronchi are small and deeply placed—*e.g.*, in the inferior axillary region. Further, it is evident that the fremitus will be intensified if good conducting material (*e.g.*, consolidated lung) intervenes between the bronchi and the parietes, while it will be weakened or annulled if bad conducting material (*e.g.*, fluid or gas) intervenes between the bronchi and the parietes. Intensification of vocal fremitus depends in the main upon the same physical conditions as those which produce bronchial breathing and bronchophony, but this rule is not without exceptions. Thus in some cases of pleural effusion we get bronchial breathing and bronchophony, depending on compression of the lung-substance by the effusion; while we get diminished vocal fremitus, depending on the intervention of a layer of fluid between the bronchi and the parietes. Diminution of vocal fremitus depends in the main upon the same causes as those which produce absence or feebleness of the breathing and diminished vocal resonance—viz., thick parietes, thickened pleura, solid tumours, pleural effusion, and blocking of the bronchi by pressure from without or the presence of secretion.

Let us now consider in more detail the cases in which the state of the vocal fremitus presents some departure from the normal.

Increase of the Vocal Fremitus.—The rule given above—viz., that the vocal fremitus is increased in all cases where good conducting material intervenes between the bronchi and

the parietes—holds good, unless some other contravening factor comes into play. Thus, vocal fremitus is increased with great uniformity in conditions (*e.g.*, pneumonia, tuberculosis), where the lung is infiltrated or consolidated. It is most important, however, to note that this increase is *conditional upon the bronchi remaining patent.* If, as often happens, the bronchi become blocked with secretion, the vocal fremitus will be temporarily weakened or annulled. On coughing and dislodgment of secretion in such cases, the vocal fremitus will reappear. If this simple rule is not borne in mind, the practitioner who finds bronchial breathing and bronchophony replaced in a case of obvious pneumonia by weak breathing or silence and diminished vocal resonance, may come to the erroneous conclusion that a pleural effusion has suddenly supervened. Vocal fremitus is commonly increased above the level of the fluid in pleural effusion, owing to compression of the lung. Vocal fremitus is also increased in cases where the lung next the chest-wall has lost its elastic tension—*e.g.*, under the clavicle on the affected side in pleural effusion—*i.e.*, where skodaic resonance is frequently present.

It might have been expected that solid tumours of the lungs would tend to increase the vocal fremitus, but the contrary seems to be the rule. In the seven cases of carcinoma of the lungs of which I possess notes,* vocal fremitus was usually diminished, though I am not sure that in every case this matter was fully considered. This fact is, however, in no way surprising. A solid tumour of the lungs might easily in the earlier stages of its growth have the same effect as infiltration and consolidation—viz., an intensification of the vocal fremitus—but it is certain before long to compress a bronchus, and hence to lead to diminution or loss of vocal fremitus.

Pleural adhesions lead to an increase of vocal fremitus, and in cases where a fresh pleural effusion has supervened

* See Lecture XXII.

upon an old case of pleurisy, the resulting physical signs may be for a time complicated and puzzling.

DIMINUTION OF THE VOCAL FREMITUS.—As already stated, vocal fremitus is diminished in all cases where bad conducting material intervenes between the bronchi and the parietes. Thus the fremitus is diminished with great uniformity in thickened pleura, pleural effusion, pneumothorax, hydrothorax, and in some cases of pulmonary tumour.

The state of the vocal fremitus in pleural effusion is worthy of special study, as it gives us one of our most trustworthy diagnostic signs. As a broad rule, the fremitus is diminished or lost over a considerable effusion, especially towards its lower parts, while above the level of the fluid the fremitus is increased. Exceptions to this rule are, however, not very rare. In cases of moderate effusion, where a thin layer of fluid intervenes between the lung and the parietes, and where, we may assume, the bronchi sometimes remain patent, the vocal fremitus may be fully preserved or even intensified, owing to increased conduction from the compressed lung. This phenomenon is commoner in the child than in the adult, and may give rise to much difficulty in diagnosis, especially as in the child pleurisy sometimes sets in very acutely, and may simulate pneumonia in its symptoms as well as in its physical signs. In such cases careful attention to the position of the apex beat, and, if necessary, the employment of puncture, will be our best resource. Thickened pleura causes an enfeeblement but not an obliteration of the vocal fremitus.

In pneumothorax the fremitus is diminished, but not usually obliterated, by the intervention of a layer of air between the bronchi and the parietes.

As already stated, vocal fremitus is diminished or lost in cases of consolidation of the lungs when the bronchi are blocked by secretion, and in cases of pulmonary tumour when the tumour causes compression of one of the main bronchi.

BACCELLI'S SIGN.—Baccelli holds that the vocal fremitus is better conveyed through serous than through purulent effusions, and that this difference possesses diagnostic importance. I believe this rule to be untrustworthy.

Tussive fremitus is sometimes helpful in the diagnosis of pulmonary disease in children.

Palpation assists us in detecting the following conditions in the chest:

(*a*) Painful areas—*e.g.*, depending on bone disease, nervous conditions, etc.

(*b*) Pleuritic friction.

(*c*) Rhonchal fremitus.

(*d*) Splashing—*e.g.*, in pyopneumothorax.

(*e*) Fluctuation.

(*f*) Pulsations—cardiac, arterial, venous, aneurysmal.

We shall briefly consider these various conditions:

(*a*) Painful areas in the chest: The presence of painful areas in the chest may have many explanations. The following conditions require to be borne in mind: Pleurodynia, pleurisy, empyema, disease of the bony framework of the chest, intercostal neuralgia, herpes zoster, aneurysm, mediastinal tumour, organic and functional heart disease. The diagnosis of most of these conditions will be more or less evident on a full consideration of all the signs and symptoms. Localized pain is in most cases a subordinate symptom.

(*b*) Pleuritic friction: This can often be felt as well as heard. In a doubtful case the tactile sense may settle the diagnosis.

(*c*) Rhonchal fremitus is common. It is not of much diagnostic importance.

(*d*) Splashing—*e.g.*, in pyopneumothorax—may be felt as well as heard.

(*e*) Fluctuation is a rare sign. It is occasionally observed in connection with old empyemata.

(*f*) Pulsations — cardiac, arterial, venous, aneurysmal: The position of the cardiac impulse must in all cases be determined with the utmost care. Many grave errors are the consequence of neglecting this simple rule.

The principal deviations from the normal position of the impulse found in connection with pulmonary disease are the following:

1. Apex displaced to the left—*e.g.*, right pleural effusion, old-standing left pleurisy involving cardiac adhesions, compensatory hypertrophy of the right lung depending on tubercular disease of the left lung, right pneumothorax, actinomycosis of right lung, hydatids of right lung.

2. Apex displaced to the right—*e.g.*, similar conditions on one or other side of the chest.

3. Apex displaced upwards—*e.g.*, tubercular disease and retraction of left upper lobe.

4. Apex displaced downwards—*e.g.*, tumour of left lung.

Let me emphatically endorse Kingston Fowler's dictum: 'The position of the cardiac impulse is the key to the diagnosis of many affections of the chest.'* The 'strange cases of pneumonia' which turn out to be pleurisy or empyema, and are not promptly recognised because the position of the apex is not carefully ascertained, are only too familiar. The beginner especially should recognise that until he has satisfied himself regarding the exact position of the cardiac impulse he can make no safe step in thoracic diagnosis.

Arterial and aneurysmal pulsations have not usually any direct association with pulmonary disease. Venous pulsation may be observed in advanced pulmonary emphysema.

* Fowler and Godlee, 'Diseases of the Lungs,' p. 54.

LECTURE II

PHYSICAL EXAMINATION OF THE LUNGS AND PLEURA (B)

SUMMARY:

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Auscultatory percussion.
Percussion sounds found in the various diseases of the lungs and pleura.

PERCUSSION.

Our knowledge of percussion is mainly due to Auenbrugger, Corvisart, Laennec, Piorry, Aran, Wintrich, Traube, and Gerhardt. It does not seem to have been known to the ancient or to the mediæval physicians.

Percussion is the most difficult mode of physical examination of the chest. Prolonged practice is necessary to acquire even a moderate degree of skill in the performance of percussion. A slight knowledge of music is a great assistance in interpreting the percussion sounds. Percussion is a method of examination of the highest importance, hardly inferior in value to auscultation. The inherent difficulties of the subject, which are neither few nor slight, have been much aggravated by the want of a clear and well-defined terminology, and by the use of obscure terms, not easily interpreted in the light of physical laws.

Mode of performing Percussion. — Pleximeters and hammers of all kinds are, in my judgment, of doubtful utility. The fingers are adequate for all purposes. It is often asserted that pleximeters and plessors are necessary for the demonstration of percussion sounds to a large class, but it will be found, I think, that the force employed in such demonstrations throws so large an area of the chest wall into vibration that no definite conclusions can be drawn as regards localizing signs, and that consequently percussion loses nearly all its diagnostic significance. It is a matter of indifference, or at least of individual preference, whether the first or the second finger of the left hand be used as pleximeter, and whether one, two, or three fingers of the right hand be used as plessor. A very satisfactory note can be elicited by using the first finger of the left hand as pleximeter and the second finger of the right hand as plessor. The pleximeter finger should be laid lightly upon the chest surface, perfect co-aptation being secured, while all pressure is avoided. The stroke given by the plessor finger must be in all cases delivered from the wrist, never from the elbow or shoulder. The blow must be given perpendicularly to the surface of the chest. As in pianoforte playing, only the pulp of the end phalanx of the plessor finger should strike the parts beneath. The hand and the fingers must not be held in a cramped or rigid position.

The force of the stroke must vary according to—

(*a*) The condition of the parietes. The thinner the parietes, the lighter must be the stroke; the thicker (*e.g.*, from muscle, fat, or œdema) the parietes, the heavier must be the stroke.

(*b*) The object aimed at in percussion—*i.e.*, whether we seek information regarding the state of the parts immediately under the parietes, or of parts more deeply placed. Light (superficial) percussion is indicated in the former, heavy (deep) percussion in the latter case. The difference between a light and a heavy stroke must not be great. In fact, using much force in percussion is always wrong; it is disagreeable to the patient, and tends to obscure the results obtained. Too vigorous percussion leads to many errors. It fails to detect slight, but significant, departures from the normal resonance, *e.g.*, in incipient phthisis; it tends to confuse the limits of cardiac dulness, and may lead to erroneous conclusions regarding the extent of the thin left lobe of the liver. Even a thin layer of pleural effusion may yield a resonant note if the percussion stroke be too forcible.

The stroke may be either 'staccato' or not. The staccato stroke is valuable in detecting the finer shades of dulness, *e.g.*, in the infraclavicular region in incipient phthisis.

It must be a fundamental rule always to compare the percussion note with the note elicited over the corresponding area on the other side of the chest, and care must be taken to compare rib with rib, and space with space. It is important, also, that the pleximeter finger should be laid in the same direction upon the chest on each side.

Percussion may be *immediate* or *mediate*—*i.e.*, the plessor finger may strike the chest wall without or with the intervention of a pleximeter finger. Mediate percussion is the usual method. The utility of immediate percussion is not great, but this method may be employed with advantage in testing the resonance of the clavicles, which is readily elicited by immediate percussion.

Percussion should be particularly gentle in the case of children, not only because of their greater nervousness as compared with adults, but because of the smaller dimensions of their chest surface, a fact which increases the difficulty of localizing percussion sounds and augments the probability of error if the percussion stroke be too heavy.

THEORY OF PERCUSSION.—The sound elicited by percussion depends upon two considerations:

(*a*) The condition of the chest wall as regards thickness and elasticity.

(*b*) The amount, tension, and distribution of air in the parts under the percussing fingers.

If the beginner will take an ordinary bladder and partially inflate it and percuss, then inflate it more fully and percuss again, then inflate it to the fullest extent and percuss once more, then make a small opening in the fully-inflated bladder and percuss yet again, he will learn in a few minutes more of the physical laws which regulate percussion sounds than can easily be conveyed by much argumentation and explication. He will find that as the bladder becomes more and more inflated, the percussion note becomes more resonant up to a certain point; then as inflation proceeds the note gradually loses resonance, until the note over a tensely-inflated bladder, which has no opening, is absolutely dull. When an opening is made the note again becomes resonant, while resonance diminishes as soon as a certain amount of air has been dislodged from the bladder. If the same experiment be performed by bladders of varying thickness and elasticity, the sounds elicited will be found to possess varying characters.

Percussion Sounds.—Much confusion has arisen from the want of uniformity in the use of the terms descriptive of percussion sounds. The subject is a difficult one, but it will repay study. It will be necessary to recall some of the historical landmarks of the question. The terms originally employed by Auenbrugger would suggest that he recognised only two elements—viz.:

(*a*) Varying degrees of pitch.

(*b*) Varying degrees of clearness or resonance.*

Skoda developed Auenbrugger's doctrine as follows:

Percussion-sounds may be divided into—

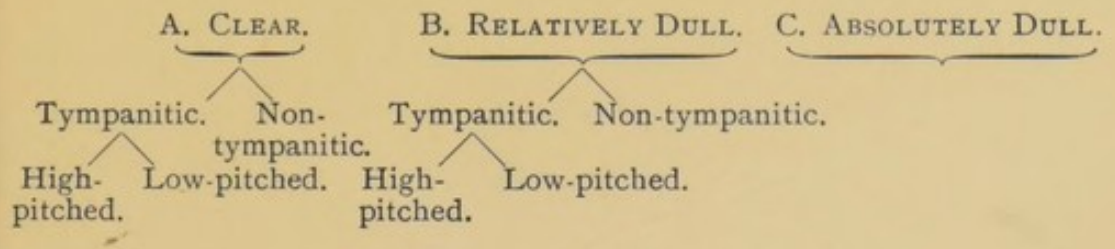
1. Voll und leer (full and empty).
2. Hell und dumpf (clear and dull).
3. Tympanitisch und nicht tympanitisch (tympanitic and non-tympanitic).
4. Hoch und tief (high-pitched and low-pitched).

Skoda added: 'A full percussion sound can be either clear or dull, tympanitic or non-tympanitic, high-pitched or low-pitched. So also can the empty (*leer*) sound.'

This classification is open to the objection that in practice *voll* (full) comes to be synonymous with *hell* (clear), and *leer* (empty) synonymous with *dumpf* (dull). Nothing is more fatal to precise observation than the employment of quasi-scientific terms, which are incapable of application at the bedside.

Gee lays down the following rules: 'The sound (*i.e.*, the sound produced by percussion) is a tone, clear or muffled, even to complete privation; this is the first and great distinction. And next, the tone is of a pitch higher or lower. Upon these two hang the whole theory and practice of percussion.'†

Sahli divides percussion sounds as follows:‡



* 'Sonitus vel altior, vel profundior; vel clarior, vel obscurior, vel quandoque prope suffocatus deprehenditur.' — 'Inventum Novum,' § 10, 'Scholium.'

† S. Gee, 'Auscultation and Percussion,' p. 70.

‡ Sahli, 'Lehrbuch der Klinischen Untersuchungs-Methoden,' p. 145.

The chief discrepancy between the view of Gee and that of Sahli is with regard to the 'pitch' of percussion sounds. In the strict acoustic sense pitch depends upon the swiftness with which the periodic sound waves follow on, *i.e.*, the number of vibrations per second made by the sounding body. Gee holds that pitch may be affirmed of all percussion sounds from the 'tympanitic' note obtained by percussing the stomach to the 'osteal' note obtained by percussion over a rib or over the sternum, the former representing the lowest, the latter the highest, pitch. Sahli, on the other hand, holds that pitch can only be truly affirmed of tympanitic sounds, and that, *e.g.*, the sound yielded by percussing the healthy chest has either no definite pitch, or one apprehended with great difficulty—in other words, that of the percussion sounds the tympanitic sound alone is a true 'tone,' the others being mere 'noises.'

This is a nice point, and depends mainly upon the strictness of our definition. The musically-trained ear has, I think, little difficulty in distinguishing variations in pitch in the sounds yielded by the chest according as the lungs are healthy, or the seat of moderate infiltration, or completely consolidated. These differences of pitch may sometimes prove of diagnostic value, *e.g.*, in investigating the percussion sounds in the infraclavicular regions in a case of suspected incipient phthisis. A rise in pitch may be sometimes recognised here before any loss of resonance is appreciable.

Practically, we investigate percussion sounds mainly with regard to the degree of 'resonance' or 'dulness' which they present. In a minor degree we attend to the pitch of these sounds. The relation of resonance to pitch is, as we have seen, a disputed point. I hold with those who maintain that pitch can be correctly affirmed of other sounds as well as of tympanitic sounds.

Skoda was probably right in maintaining that there is no such thing as a 'liver note,' a 'spleen note,' a 'heart note,'

or a 'stomach note'; that all solid organs yield the same percussion note; and that in air-containing organs the note has relation simply to the amount, distribution, and tension of the air which it contains. 'The different sounds which percussion produces over the regions of the liver, spleen, heart, lungs, and stomach do not depend upon any peculiarities in those organs, but upon variations in the quantity, distribution, and tension of the air present in the regions in which they lie, and upon the force of the percussion stroke.'*

We may recall that Piorry taught the contrary of this doctrine. I adhere to Skoda's view.

Percussion of the Normal Chest.—Percussion gives us information regarding the thickness and elasticity of the parietes of the chest, the amount and tension of the air in the lungs, and enables us to define the outlines of the various thoracic viscera.

The thinner and more elastic the parietes of the chest, the more resonant is the percussion sound which they yield, and *vice versâ*. In thin persons and in children the note is usually highly resonant, while it is feebly resonant in very muscular or very stout people. If ossification of the cartilages has taken place to any extent the chest loses elasticity and becomes less resonant. Resonance in the normal chest has also a relation to the degree of expansion of the lungs. In a patient who has been confined to bed for some time from any cause, the bases of the lungs will be found to be somewhat less resonant than in persons who are in full activity. The various regions of the chest present varying degrees of resonance. The infraclavicular regions are commonly the most resonant parts, while the supra-scapular regions and (in women) the mammary regions are the least resonant parts.

Definition of the Limits of Organs.—Normal pulmonary

* J. Skoda, 'A Treatise on Auscultation and Percussion,' Markham's translation, pp. 6, 7.

resonance extends from about 1½ inches above the clavicle to the upper border of the fourth rib on the left side, and the upper border of the sixth rib on the right side in the mammary line. In the axillary line the lower border of pulmonary resonance is at the eighth or ninth rib, in the scapular line at the tenth rib, and in the posterior dorsal line at the eleventh spinous process. Allowance must be made for the varying degrees of expansion of the lungs during inspiration and expiration. The pulmonary excursion during respiration amounts to 8 centimetres.

In localizing pulmonary signs the following landmarks are important: the vertebra prominens, the prominence on the sternum opposite the second costal cartilages, the base of the spine of the scapula (on a level with the upper portion of the lower lobe), the lower angle of the scapula, (opposite the seventh rib and seventh spinous process). The base of the ensiform appendix is a fixed point and corresponds to the lower border of the body of the ninth dorsal vertebra. The tip of the ensiform is a variable point.

Abnormal Percussion Sounds.—The terms commonly employed to indicate abnormalities of the percussion sounds are the following:

Dulness (varying in degree from a muffled note to one of complete wooden dulness).
Hyper-resonance (tympanites).
Skodaic resonance.
Amphoric resonance.
Box-tone (Schachtelton).
Cracked-pot sound (*bruit de pot fêlé*).

Dulness of varying degree is afforded by all those numerous conditions which either diminish the elasticity of the chest walls or reduce the amount of air in the air vesicles. Varying degrees of dulness are found in the following conditions:

thickened pleura; pleural effusion; hydrothorax; hæmothorax; consolidation, infiltration, compression, or induration of the lungs; atelectasis.

Where dulness is present it is of great importance to note the following points:

(*a*) The degree of dulness, both to light and heavy percussion.

(*b*) The precise outlines of the dull area.

(*c*) The effect upon the dull area of change of posture and of full inspiratory effort.

It is important to note that dulness may, in the progress of any given case, vary either as regards intensity or extent. Thus, as a large pleural effusion subsides, the dull note may either become more circumscribed or less intense. The dulness of pleural effusion is, in most cases, more complete, more absolutely 'wooden,' than that of infiltration or consolidation of the lungs.

In percussing the chest of children, it is important to bear in mind that their chest walls are more resilient than those of adults, and that hence dulness may, *ceteris paribus*, be less pronounced than in adults. Thus, in small pleural effusions in the child, if the beginner percusses with undue force, he may produce a fairly resonant note, and may be quite deceived as to the nature of the case before him.

The term 'tympanitic resonance' is used in different senses by different writers. I shall employ it in conformity with ordinary English usage.

Tympanitic percussion sounds may be present in the following conditions: emphysema; pneumothorax; pulmonary cavities—tubercular, bronchiectatic, or resulting from pulmonary abscess; relaxation of the lung in consequence either of changes in the lung itself or of pressure upon it from without either by tumours or pleural effusions; diaphragmatic hernia. These various conditions will engage our attention hereafter. One or two observations may suffice

in this place. In pneumothorax, if the tension of the air in the chest undergoes a progressive increase, the note previously resonant gradually loses its resonance, and finally becomes dull. To such a note the term 'tympanitic dulness' is sometimes applied.

Tympanitic resonance over a cavity indicates that the cavity is of considerable size, near the surface, and contains air. The precise cause of the tympanitic note obtained over 'relaxed' pulmonary tissue is not definitely known, but the fact is certain. Examples of this 'relaxed lung' note are found in the percussion at the edges of consolidated lung, or in some cases of tubercular infiltration, or above the level of the fluid in pleural effusion. This 'relaxed lung' note is a frequent stumbling-block to a beginner.

Tympanitic resonance in the case of diaphragmatic hernia is due to the passage of portions of bowel into the thorax.

Skodaic Resonance.—This is an important sign, attention to which was first drawn by the great Viennese clinician. It is a note of resonant quality and low pitch, heard under the clavicle on the affected side in many cases of pleural effusion. One of my correspondents* suggests that it is also present on the other side, but I cannot affirm this from personal observation.

Various views have been held as to the mode of production of this sound. Skoda regarded it as due to relaxation of the lung. 'That the lungs partially deprived of air should yield a tympanitic, and when the quantity of air in them is increased a non-tympanitic sound, appears opposed to the laws of physics. The fact, however, is certain, and is corroborated both by experiments on the dead body, and also by this constant phenomenon—viz., that when the lower portion of the lung is entirely compressed by any pleuritic effusion, and its upper portion reduced in volume, the

* Dr. James Young, of Invercargill, New Zealand.

percussion sound at the upper part of the chest is distinctly tympanitic.'*

C. J. B. Williams thought this sound was produced by vibration of the air in the large bronchi, but this is an improbable view.

The pitch of the sound in skodaic resonance is higher than in pneumothorax, because the vibrating area in the former condition is smaller than in the latter.†

Amphoric Resonance.—This is a variety of tympanitic resonance, sometimes heard in percussing over large and superficial cavities.

Box-Tone (*Schachtelton*).—A sound heard in emphysema, compared to the resonance of a paper-box. Light percussion must be used (Biermer).

Cracked-Pot Sound (*Bruit de Pot Fêlé*).—This well-known and interesting sign is a resonant note of variable pitch, metallic in quality, characterized by a peculiar 'chink,' fairly well imitated by placing one hand over the other, so as to form an air-containing space, and then smartly striking the knee with the under hand, the patient being directed to keep the mouth open. This sound is most often heard over cavities which are large, superficial, air-containing, and communicating freely with a bronchus. Most observers are agreed that a note indistinguishable from the cracked-pot sound is sometimes heard under normal conditions, *e.g.*, when percussing the back of a screaming baby. Skoda denied this. The cracked-pot sound may occasionally be heard over relaxed and partially collapsed lung, *e.g.*, above the level of the fluid in pleural effusion, and in cases of infiltration of the lung with tubercle or inflammatory material. The physical explanation of this fact is not known. A typical cracked-pot sound is, however, strongly suggestive of cavity. The sound is usually better appreciated in thin people.

* J. Skoda, *op. cit.*, pp. 13, 14.

† Bristowe and others.

Sense of Resistance felt in Percussion.—The plexi-meter finger conveys a sense of resistance which requires to be distinguished from the sound produced by percussion. This sign was regarded by Piorry as of more value than the percussion sound, a view which is not now maintained. This sense of resistance attains its maximum in pleural effusion, provided the effusion is sufficient to make tense the pleural walls, and in intrathoracic tumours. Massive and dense pulmonary consolidations come next. Lung infiltrated with tubercle and commencing fibrinous exudation gives a less sense of resistance. In pneumothorax and emphysema the distension of the pleural cavity or of the air cells may give rise to a certain degree of resistance. Healthy lung offers no sense of resistance. The amount of resistance in the various cases depends mainly upon the degree of distension of the pleural cavity with fluid or with air, or upon the density of a pulmonary consolidation.

Auscultatory Percussion.—This mode of percussion is advocated by some authorities as useful in mapping out the area of the heart and the liver. Its utility does not appear to be great.

I shall now give a synoptical view of the various varieties of percussion sound found in the different diseases of the lungs and pleura.

1. Pleural Effusion.—The degree of dulness depends not directly upon the amount of fluid in the pleural cavity, but indirectly upon the deprivation of the lung of air. An absolutely dull note shows that there is fluid present, and that the underlying lung is airless. In moderate effusions the percussion sound may be muffled, rather than dull. In children, the resiliency of the chest walls may modify the dulness to a degree which sometimes causes difficulties in diagnosis.

Distribution of the Dulness and Shape of the Dull Area.—There are some very important rules in this connection which demand our best attention. The fluid at first collects at the base posteriorly, and this appears to be the case even in ambulatory patients. Dulness frequently extends a considerable height posteriorly while the front continues completely resonant. The physical causes of this phenomenon are not well understood. It has been suggested that differences may exist in the elasticity of different portions of the lung area.* As the fluid increases in amount dulness extends to the axilla and the front, but the upper level of dulness is seldom horizontal. Much oftener it is higher behind than in front. In a certain proportion of cases the highest level of the fluid is in the axilla, and the upper level of dulness forms a curve, of which the highest point is in the axilla and the lowest point in front—*Ellis's curve.*

In left-sided effusions Traube's *Half-Moon Space* (*i.e.*, the space between the spleen, the heart and the liver, and the lower costal margin) often remains resonant, while the parts above it are dull. Ultimately, with an increasing effusion, this space also becomes dull.

Posture, as a rule, has no influence upon the shape of the dull area. The exceptions which occur to this rule are chiefly in the case of passive effusions. The percussion note above the level of the fluid is frequently tympanitic, and sometimes possesses the cracked-pot quality. Skodaic resonance is often found under the clavicle on the affected side.

The degree of dulness throws no light on the character of the fluid. It is the same whether the fluid be serous exudation, passive effusion, pus, or blood.

Dulness on percussion in pleurisy may be due to thickened pleura, and it is sometimes of considerable importance to distinguish this condition from effusion. The distinction is often difficult, and may be impossible. The following con-

* Sahli, *op. cit.*, p. 192.

siderations may be of assistance in a doubtful case: If the dulness is slight in degree and rather extensive in area, if the heart is not displaced, if the vocal fremitus is fairly well marked, we are probably dealing with a thickened pleura, and *vice versâ*. Another important consideration is this: Thickened pleura usually co-exists with some shrinkage of the lung on the affected side, while an effusion is more likely to lead to an increased measurement of the affected side, though this is by no means invariable.

Hydrothorax follows in the main the same laws as pleural effusion, but dulness is in this case more affected by posture; the upper level of the dulness is often horizontal, and the effusion is often bilateral, though unequal on the two sides.

2. PNEUMOTHORAX.—The percussion sounds are usually tympanitic over the upper portions of the affected side and dull at the base, owing to the presence of effusion, which may be either serous or purulent. If, however, the air-tension in the chest is increased beyond a certain point, the tympanitic sound loses its resonant quality, and may even become quite dull. If a broncho-pleural fistula forms, the note will again become resonant.

In pneumothorax with effusion the outline of dulness is much influenced by posture. Sometimes in these cases splashing may be heard, while no dulness can be demonstrated in percussion. The explanation is that the fluid collects under the lung.

3. PNEUMONIA—(*a*) *Stage of Engorgement.*—The percussion note is either normal, or more or less tympanitic. Skodaic resonance is occasionally present. There is no dulness.

(*b*) *Stage of Consolidation.*—Dulness of varying degree is usually present. The dulness is usually less 'wooden' than in pleural effusion. It is also less sharply defined than the dulness of pleural effusion, *i.e.*, the dull area fades gradually into resonant areas. The dulness has no fixed shape or definite limitations.

The dull note may be modified by—

(1) The presence of healthy lung tissue either above or beneath the consolidated area.

(2) The presence of patches of air-containing lung amongst the consolidated areas.

(3) The presence of air in a large bronchus beneath a thin layer of consolidated lung.

(4) The transmission of the stomach note through a consolidated left lower lobe anteriorly.

The above considerations account for the fact that a tympanitic percussion note (more rarely, a normally resonant note) may be present throughout the consolidation stage of pneumonia. On the whole, however, dulness is fairly constant. All parts of the chest should be carefully explored. 'Many pneumonias, both of the upper and the lower lobe, first show hepatization in the axillary region, and many pneumonias which begin centrally first reach the surface in front or at the sides, and in this way become accessible to the methods of physical examination' (Gerhardt).*

The lung tissue in the neighbourhood of the consolidated area often yields a more or less tympanitic note.

(*c*) *Stage of Resolution.*—Dulness becomes less marked, and gradually gives way to normal resonance.

A clear tracheal, tympanitic, or skodaic note is sometimes heard during this stage.

4. Broncho-Pneumonia.—The percussion sounds present in broncho-pneumonia are variable, and may be misleading. If the pneumonic foci are large and tend to coalesce, areas of dulness will be present. The dull areas are usually posterior and inferior in position, or they may be at the borders of the lungs in front or laterally, or, finally, they may be on either or both sides of the spine. Dulness may also be due to collapse, which is frequently present.

* Nothnagel's 'Encyclopædia of Practical Medicine,' English edition, article 'Pneumonia,' p. 459, quoted by Aufrecht.

On the other hand, over-distension of the air vesicles often gives rise to varying degrees of hyper-resonance, or it may happen that the percussion sounds present little departure from the normal.

Aufrecht is of opinion that dulness at the apices is sometimes due to lobular pneumonia confined to the bases. 'These changes,' he says, 'in the percussion note over the normal apex can be explained only by the fact that, under the influence of the changes at the base, the tension of the normal tissue at the apices had been modified, so that the percussion note, as compared with the high tympanitic quality over the lower lobes, appeared empty and almost dull.'*

If this view is sound, it is obvious that in cases of this description an erroneous diagnosis of tuberculosis of the apex might easily be made. Careful and repeated examinations of the sputum will be the chief security against error.

5. Passive Congestion and Œdema.—These conditions may be present without any alteration in the percussion note. If the lung becomes practically airless, the percussion note will be dull; but it is necessary to insist that a high degree of congestion and œdema is consistent with normal percussion resonance.

In some cases of œdema we get the tympanitic note characteristic of relaxed lung.

Hydrothorax often co-exists with passive congestion and œdema, and will, of course, cause dulness at the bases.

6. Pulmonary Induration (Fibrosis of the Lung).—Most of these cases are tubercular, but fibrosis may be due to irritative bronchitis, broncho-pneumonia, unresolved croupous pneumonia, pleurisy, neoplasms, pressure by aneurysm, foreign bodies in the bronchi, syphilis, and other causes.

* *Op. cit.*, p. 568.

Dulness is often present, and will present many variations as regards degree and extent, according to the nature of the primary disease. Pleural adhesions and pleural thickening may be present, and will affect percussion. A tympanitic or amphoric note may be present, depending on the existence of bronchiectatic cavities.

7. PULMONARY INFARCTION.—If the infarct attain a certain size, dulness (not usually intense) will be present. Infarcts are usually posterior and inferior in situation.

8. BRONCHITIS—(*a*) *Acute.*—In acute, uncomplicated bronchitis the percussion note is not necessarily affected. No amount of secretion in the bronchial tubes will cause dulness, provided the lung tissue remains unaffected.

Alterations in percussion resonance are, however, common in acute bronchitis, depending on one or other of the following conditions, viz., pulmonary collapse, acute emphysema, relaxation of lung tissue. Collapse causes a muffled or dull percussion note. Acute emphysema and relaxation of lung tissue give rise to varying degrees of hyper-resonance. In the acute bronchitis of young children the practitioner should be watchful for the supervention of collapse. Recession of the lower intercostal spaces is one of the most important signs of this condition.

(*b*) *Chronic Bronchitis and Emphysema.*—In these conditions the percussion note undergoes two changes, viz., it becomes more resonant, and it falls in pitch. The area of pulmonary resonance is increased upwards and downwards. The area of superficial cardiac dulness is diminished or obliterated. Resonance may extend as low as the seventh rib in front and the twelfth rib behind. In some cases there is dulness at the bases due to slight effusion. In very extreme cases of emphysema the percussion note becomes less resonant and acquires a certain flatness of tone. The percussion sounds in emphysema may be modified by rigidity of the chest walls, which is sometimes marked in old subjects.

9. New Growths in the Lungs or Mediastinum.—Dulness of a very intense degree is often present in these cases when the growth has attained to a certain size. The size of pulmonary neoplasms is variable, and no definite rules can be laid down; but such growths in their original seat and subsequent extension exhibit none of the topographical rules which are more or less characteristic of pulmonary tuberculosis. Mediastinal growths often cause a dull area in the superior sternal region. Pleural effusion is common in association with pulmonary neoplasms, and will produce its characteristic percussion sounds.

10. Actinomycosis of the Lungs.—Dulness is present, often basic in situation.

11. Hydatids of the Lungs.—The most typical percussion sound in pulmonary hydatids is dulness over a rounded area, absolute in the centre, and becoming gradually less marked towards the periphery. Deep-seated cysts may not cause any alteration in the normal resonance. A portion of lung compressed by a cyst may yield the tympanitic note characteristic of relaxed lung.

12. Atelectasis.—This condition, when depending on compression of the lung, causes marked dulness; when due to obstruction of a bronchus, dulness is less marked, and may be absent.

13. Tuberculosis of the Lungs.—Attention to the percussion sounds is of the utmost importance in the diagnosis of pulmonary tuberculosis. Every variety of percussion sound may be present in this disease, but certain more or less definite rules can be formulated.

(*a*) *Acute Miliary Tuberculosis.*—The percussion sounds may be normal, but a slight degree of dulness, rarely marked, or hyper-resonance due to over-distension of the air vesicles, may be present. The percussion sounds may vary from day to day.

(*b*) *Acute Pneumonic Phthisis.*—The percussion sounds resemble those of pneumonia.

(*c*) *Fibroid Phthisis.*—See under Pulmonary Induration.

(*d*) *Chronic Fibro-caseous Tuberculosis of the Lungs* (*Chronic Pulmonary Phthisis*).—The percussion sounds may be normal. 'The sound of a lung containing merely a few solitary tubercles does not differ from that of a healthy lung' (Skoda). Dulness varying in degree may be present, depending on infiltration of the lung with abundant tubercle; patches of pneumonia; fibroid thickening, especially round a cavity; a fluid-containing cavity. Hyper-resonance may be present, depending on relaxation of the lung tissue; a cavity containing air, and abutting on the surface; bronchiectatic dilatation of the tubes in relation with the tuberculous area. In incipient phthisis careful percussion of the supra-clavicular, infraclavicular, and suprascapular regions is of the highest value. Slight degrees of dulness in these regions are very significant. The pitch of the percussion tends, I believe, to rise on the side where there is commencing tubercular infiltration. Infiltration may proceed a considerable way before any dulness can be detected, and even extensive infiltration and softening may occur without any notable alteration of the percussion sounds. In some cases of infiltration and softening the heightened resonance of relaxed lung may be observed.

Percussion Sounds over a Pulmonary Cavity.—The following percussion sounds may be heard over a cavity:

(*a*) Normal resonance when the cavity is small or covered by a considerable layer of healthy lung.

(*b*) Dulness when the cavity contains fluid or is surrounded by a fibrous envelope. The dulness varies from time to time according to the amount of expectoration.

(*c*) Cracked-pot sound when the cavity is fairly large, near the surface, containing air, and freely communicating with a bronchus.

(*d*) Hyper-resonance when the cavity is large, superficial, and full of air.

The most characteristic percussion sounds over a cavity are dulness and the cracked-pot sound. The pitch of the percussion sound over a cavity is higher when the patient's mouth is open (Wintrich's sign). Percussion gives no information as to cause or type of cavity. It is conditioned solely by physical laws.

LECTURE III

PHYSICAL EXAMINATION OF THE LUNGS AND PLEURA (C).

SUMMARY:

Historical note on auscultation.

Methods of auscultation:

Immediate and mediate auscultation.

Accessory and extraneous auscultatory sounds.

Auscultation of the breath sounds:

Theories regarding the mode of production of the vesicular murmur.

Physiological variations in the character of the vesicular murmur.

Alterations in the quality of the breath sounds:

Laennec's classification of breath sounds, normal and abnormal.

Skoda's classification.

Vesicular breathing: Alterations in intensity; Alterations in rhythm.

Bronchial breathing (tubular breathing).

Cavernous breathing (amphoric breathing).

Adventitious sounds in the lungs and pleura:

Laennec's classification of râles.

Skoda's classification of râles.

Pleural friction—Rhonchus—Bubbling, non-consonating râle — Consonating râle — Crepitant râle—Gurgling (cavernous) râle.

Auscultation.

Auscultation, as a systematized art, is the creation of Laennec. Hippocrates had discovered the succussion splash, and probably also pleural friction, but no substantial progress was made from his day until that of the great French clinician. Laennec, having discovered the principle of mediate auscultation and invented the stethoscope, proceeded to work the subject to the bottom at the Hôpital Necker, Paris, and practically exhausted it, so far as the lungs are concerned. He was less successful with the auscultation of the heart. Since his day, auscultation has been further developed by Skoda, Wintrich, Traube, Williams, Bamberger, Weber, and Gerhardt. Skoda, who brought the laws of physics to bear upon the phenomena of auscultation, corrected and regularized Laennec's teaching. Skoda's work was, in the main, critical rather than creative.

Laennec's 'Traité de l'Auscultation Médiate,' published in the year 1819, will always remain one of the great landmarks of medicine, and should be read by every physician who interests himself specially in diseases of the lungs and of the heart. It is illuminated by profound insight and philosophic acumen, and is the fruit of vast experience and unwearied labour. It can never become antiquated, and subsequent observers are reduced simply to the position of critics. The great fundamental creative work has been done, once for all, by Laennec. Nevertheless, the work is open to criticism from, at least, two points of view. Laennec fell into two errors: he overrated the indispensability of mediate auscultation, and he formulated a terminology of auscultatory terms too elaborate for application at the bedside. If auscultation is to be made fully available for the practitioner who is not a pulmonary expert, it must not be overloaded with technical terms. The present-day terminology of auscultatory pulmonary signs is, in many respects, diffuse and

vague, over-elaborate and yet wanting in precision, confusing to the learner, and not readily handled by the expert.

METHODS OF AUSCULTATION. — Auscultation is either (*a*) *immediate, or* (*b*) *mediate.* In the former case the ear is applied directly to the chest wall; in the latter some form of stethoscope is employed.

Immediate auscultation, contrary to the opinion of Laennec, is a method of great value, and should be practised by everyone. For obvious reasons, it is not always available. It is more suitable in the case of children than in that of adults, and more applicable to the posterior than to the anterior surface of the chest. A very satisfactory examination of the chest can often be made by the direct application of the ear, a thin handkerchief or towel being allowed to intervene between the ear and the thoracic wall. Guttmann advises immediate auscultation in the case of feeble patients and in exhausting diseases, where it is important to go over the chest rapidly. Immediate auscultation is inferior to mediate in cases where precision is required, and where the definite localization of pulmonary signs may be essential. There are, moreover, certain regions of the chest, *e.g.*, the supraclavicular fossæ, where immediate auscultation is impracticable.

Mediate auscultation is the usual method. It is generally more convenient than immediate auscultation, more agreeable to the patient, more helpful in localizing topographical details, and it more readily lends itself to the exploration of difficult and obscure signs. Every form of stethoscope, however, modifies the sounds in some degree. The intensification of the sounds produced by some forms of stethoscope is not always an advantage. As to the form of stethoscope, the student would do well to familiarize himself with the wooden stethoscope, the binaural, and the phonendoscope. For ordinary purposes, the binaural stethoscope is the most convenient. It is immaterial which of the many varieties of binaural stethoscope we

employ. On the whole, the more complicated forms of this instrument are to be avoided. Care should be taken to see that the ear-pieces fit easily into the ears, and do not exercise any undue pressure. The phonendoscope, which I have found of real service in the exploration of obscure cardiac murmurs, is seldom of assistance in pulmonary cases. The chest-piece of the stethoscope should be applied lightly and evenly to the chest, without pressure. The patient is then instructed to open the mouth, to inspire and expire fully and deeply without violent effort, and not to make any noises with the mouth. A general survey of the chest should first be made, and then the auscultator should return to those areas where he suspects the presence of abnormal signs. It is important to examine both before and after coughing.

Accessory and Extraneous Sounds.—Our first care in auscultation must be to exclude or ignore certain sounds which do not arise in connection with the lungs or pleura. Sounds may be generated by imperfect coaptation of the chest-piece of the stethoscope to the chest wall, by inequalities on the surface of the chest, by contact with hairs or clothing, by contraction of the muscles of the chest. These sources of error can usually be obviated without difficulty. A little oil applied to the surface of the chest may be helpful. Extraneous sounds can usually be differentiated with ease by the practised ear. They are usually 'superficial,' rubbing or scraping in quality, and do not conform to any recognised variety of pulmonary sound. A comparison of the sounds produced over different areas of the chest often gives us the clue to extraneous sounds.

Auscultation must be conducted upon a definite method. The most convenient method is the following. We listen in order to:

A. The Breath Sounds.

B. Adventitious Sounds—râles, rhonchi, etc.

C. The Conduction of the Voice (Vocal Resonance).

It is most important to attend to these separately and in this order.

A. The Breath Sounds.

Normal vesicular breathing has a peculiar and well-known rustling or sighing quality. It varies much in intensity, according to the thickness of the parietes and the vigour of the respiratory act. The audible inspiration is four or five times as long as the audible expiration, although the actual length of inspiration compared with expiration is as ten to twelve. Expiration is sometimes weak and may even be quite inaudible under normal conditions. In normal breathing the inspiration overshadows the expiration. The contrary is the case in some states of disease.

The precise mode of production of the vesicular murmur is not certain. Three theories have been propounded on the subject, viz. :

1. That the vesicular murmur is produced in the alveoli by friction of the air against the walls of the alveoli at the moment of their dilatation (Laennec, Skoda).

2. That the vesicular murmur is produced at the glottis and is modified by conduction to the alveoli, (Beau, Baas, Gee).

3. That the vesicular murmur is due to vibration of the lung tissue, its tension being increased during inspiration (Gerhardt).

Whether the vesicular murmur is due to friction of the air against the walls of the alveoli or to vibration of the lung tissue is a point of no practical importance, however interesting from the physiological point of view—in other words, the point at issue between the first and the third of the above theories may be neglected, without much loss, by the clinician. It is, however, a matter of genuine practical importance to satisfy ourselves whether the vesicular murmur, as heard over the chest, is produced *in loco* or is a conducted

sound, propagated from the glottis. Our view of many pulmonary problems will be affected by the theory which we adopt on this subject.

I hold to the view that the vesicular murmur is produced in the small bronchioles and the vesiculi, and that it is not a sound produced in, and propagated from, the glottis for the following reasons:

(*a*) If the sound were of glottic origin it would undergo a progressive modification, probably a weakening of its essential characters, the further we go from the glottis. This is not the case. The vesicular murmur is practically, and, allowing for differences in the thickness of the parietes, as pronounced, *e.g.*, in the lower axillary region as in the mammary region in front. The bronchial breathing often heard under the clavicles, especially on the right side, does not conflict with this view, as we are here listening directly over a main bronchus.

(*b*) When any part of the pulmonary substance is acting with unusual energy (*e.g.*, in vicarious breathing in connection with pleural effusion, tuberculosis, large pulmonary infarct, and other conditions), the vesicular murmur over this area is intensified. It is difficult to see how this fact, which is certain, could be reconciled with the glottic view of the origin of the vesicular murmur.

(*c*) In stenosis of the larynx the vesicular murmur is weakened, while the sounds heard over the larynx are intensified.*

Variations of the Vesicular Murmur under Normal Conditions.—It is most important for the beginner to familiarize himself thoroughly with the variations in the vesicular murmur which are compatible with health. Many errors arise from confusing physiological with pathological variations. The vesicular murmur varies under normal conditions:

* See Sahli's excellent discussion of this subject, *op. cit.*, p. 213 *et seq.*

(1) According to the thickness of the parietes. *Ceteris paribus*, it is louder in thin than in stout or muscular subjects; louder where the chest coverings are thin, *e.g.*, in the infraclavicular regions, than where they are thick, *e.g.*, the supraspinous fossæ.

(2) According to the thickness of the subjacent layer of lung—louder, therefore, over the front and back generally than at the apices or the anterior or inferior borders.

(3) According to the activity of the respiratory processes.

The intensity of the vesicular murmur varies sometimes on the two sides of the chest generally. These variations may have their basis in anatomical peculiarities, *e.g.*, in variations in the shape, thickness, or direction of the bronchial tubes. The vesicular murmur is normally more distinct in the right than in the left infraclavicular region, and may be harsh or even bronchial in this situation, especially in the case of children. This is a fruitful source of difficulty to the beginner in auscultation, who will find it a useful rule to be chary of drawing conclusions from the breathing alone in the infraclavicular region, and to be careful to collate the other signs, especially those educed by percussion. The breathing of the child is more *shrill* than that of the adult. It is called *puerile* breathing. The explanation of this type of breathing is to be found chiefly in the thinness of the chest wall in the child; to a minor degree, perhaps, in the greater elasticity of the lungs and the greater activity of the respiratory processes in childhood. When we hear vesicular breathing of normal quality, we conclude not that the lungs are necessarily healthy, but that air is entering freely. Normal breathing is not inconsistent with disease, *e.g.*, a slight degree of bronchitis, a few scattered tubercles, the early stage of pneumonia. Upon the whole, however, when we have satisfied ourselves that the breathing is perfectly normal, we have gone a long way towards assuring ourselves that the lungs are healthy.

The breath sounds must be studied as regards :

Alterations in quality.
Alterations in intensity.
Alterations in rhythm.

ALTERATIONS IN QUALITY.—The quality of the breathing may be vesicular, broncho-vesicular, bronchial, tubular, cavernous, amphoric, or interrupted (wavy, cog-wheel, *respiration saccadée*).

Laennec's classification of breath sounds was as follows—

1. Respiration vésiculaire (normal vesicular breathing).
2. Respiration bronchique (bronchial breathing).
3. Respiration puérile (puerile breathing).
4. Respiration caverneuse (cavernous breathing).
5. Respiration soufflante (a variety of bronchial or cavernous breathing, in which the air seems to be drawn towards the ear of the observer during inspiration, and repelled from it during expiration).
6. Souffle voilé (veiled puff, described as a condition in which the air seems to set in motion a veil interposed between the ear of the observer and a cavity).

Skoda subjected the above classification to a trenchant criticism, and had no great difficulty in showing it to be unsatisfactory. He elaborated his own classification as follows :

1. Das Lungen-Respirationsgeräusch, oder vesiculäres Athmen (vesicular breathing).
2. Das bronchiale Athmen (bronchial breathing).
3. Der amphorische Wiederhall und der metallische Klang (amphoric echo and metallic tinkling).
4. Das unbestimmte Athmungsgeräusch (indeterminate respiratory murmur).*

Laennec's classification seems open to criticism from several points of view. His *respiration soufflante* and *souffle*

* J. Skoda, 'Percussion und Auscultation,' p. 107.

voilé are simply varieties of cavernous breathing. The latter term introduces a refinement which has no practical utility.

Skoda's classification also seems somewhat faulty. His 'indeterminate breathing,' by which he understood a variety of breathing neither purely vesicular nor definitely bronchial, is a mere negative term which has retained its place in German phraseology, but has never been definitely acclimatized in English practice. In his definition of 'indeterminate breathing' Skoda does not seem to have always clearly differentiated differences in the *quality* of breathing from differences in *intensity*.* If by this term we are to understand simply a variety of breathing which is intermediate in quality between vesicular and bronchial, it would be better to use the term 'broncho-vesicular.' Skoda's 'amphoric echo' and 'metallic tinkling' are comparatively rare sounds, which cannot be conveniently erected into types. Further, Skoda denied that there was any real distinction between bronchial breathing and cavernous breathing. In this view he seems to have been mistaken. Cavernous breathing has a definite quality of its own, which can be recognised, and from which important inferences may be drawn.

The quality of the breath sounds may be conveniently regarded as belonging to one of three types, viz.:

1. Vesicular (including broncho-vesicular).
2. Bronchial (including tubular).
3. Cavernous.

1. *Vesicular Breathing.* — Vesicular breathing is softly blowing or rustling in quality, of variable intensity, the inspiration much more pronounced than the expiration, which is often short, and may be quite inaudible. Over the root of the lungs in the interscapular region its quality may partake of the bronchial type, and it may then be denominated broncho-vesicular.

* J. Skoda, 'Percussion and Auscultation,' p. 114 *et seq.*

Vesicular breathing, normal in all respects, may be present in states of disease. More often it is altered either as regards (*a*) intensity, or (*b*) rhythm.

(*a*) *Alterations in intensity*.—We may recognise two types:

1. Exaggerated or harsh vesicular breathing.
2. Diminished or weak vesicular breathing.

Exaggerated or harsh vesicular breathing is found in the early stage of bronchitis, where it is due to slight swelling of the bronchial mucous membrane; in commencing phthisis, where it is due to localized catarrh, usually of one apex; in the early stage of pneumonia, before consolidation has begun;* over the sound side or sound portion of an affected side, when from any cause (pleural effusion, pneumonia, tuberculosis, infarction) one side, or a portion of one side, is deprived of its functional activity; in certain states of dyspnœa, depending upon disease in organs other than the lungs, *e.g.*, heart, larynx.

Diminished or weak vesicular breathing is found in pleural effusion; in bronchitis and emphysema; in pneumonia before consolidation has become established and during resolution; in phthisis; in neoplasms of the lung. The causes of weak breathing in the above cases are sufficiently obvious. They are as follows: Pressure upon the lung from without, as in pleural effusion; impediment to the free entry of air into the lungs, as in bronchitis, pneumonia, and phthisis; diminished elasticity of the lungs, as in emphysema; pressure upon the lung by solid growth in the lung, as in neoplasms of the lung.

Harsh breathing should suggest to us that there is either some impediment to the entry of air into the lung, that impediment being slight, sufficient to increase the labour of the lung without causing much actual obstruction, or that a portion of the lung or lungs is abnormally active, owing to a

* Stokes.

deficiency of activity in some other portion of the pulmonary area.

Weak breathing should suggest to us that there is either a considerable obstruction to the entrance of air into the lung, or that the elasticity of the lung is diminished, or that the lung is infiltrated or compressed.

(*b*) *Alterations in rhythm.*—(1) The inspiration may be delayed, *i.e.*, the inspiratory sound may follow the inspiratory movements after a definite interval. The causes of this phenomenon are not well understood.

(2) The inspiration may be prolonged. Prolonged inspiration is characteristic of cases in which there is some impediment to the free entry of air into the larynx and bronchial tubes, *e.g.*, acute laryngitis, œdema of the glottis, stenosis of the larynx, stenosis of the trachea, diphtheria.

(3) The expiration may be prolonged. Prolonged expiration may be general or local. The former type is characteristic of bronchitis, emphysema, and asthma; the latter of pulmonary infiltration (pneumonic or tubercular). Diminished elasticity of the lung tissue is the most usual cause of prolongation of the expiration.

(4) Inspiration or expiration, or both, may possess the wavy, cog-wheel character (*respiration saccadée*). The significance of cog-wheel respiration has been much debated. It would appear to be due either to swelling of the bronchial mucous membrane or to the presence of secretion there, or to irregular action of the respiratory muscles, especially the muscles of inspiration. As the causes of this phenomenon vary, so is also its significance variable. When it occurs generally over the chest it may be ignored. When it is limited to one or both apices the suspicion of commencing phthisis may arise; but, in the absence of other signs, no definite conclusion can be drawn. I have sometimes found this sign very fluctuating, appearing and disappearing in the course of the same examination. It

cannot be denied that it is a common sign in commencing phthisis, but it is not to be relied upon in any case of doubt.

2. *Bronchial Breathing.*—Bronchial breathing may be defined as a variety of breathing present in most healthy persons (especially in thin people) at the level of the seventh cervical and upper five or six dorsal vertebræ. Tubular breathing, strictly defined, does not occur in health. It is characteristic of somewhat dense consolidations, and resembles bronchial breathing with a superadded metallic quality. Bronchial breathing has a peculiar quality, readily appreciated, to which the terms 'hollow' and 'reverberating' have been applied. It may be loud or weak, high-pitched or low-pitched. In the large majority of cases the peculiar 'bronchial' quality is more evident in expiration than in inspiration. The expiration is often of a higher pitch than the inspiration. Whereas in normal breathing the expiration is short, weak, of a 'dying-away' quality, and of a lower pitch than the inspiration, in bronchial breathing these characteristics are reversed. Here the expiration is the pronounced element. It is usually well marked, prolonged, often low, frequently of higher pitch than the inspiration, and of a peculiar hollow quality, readily appreciated when the ear has become familiarized with the sound. In studying bronchial breathing, the beginner in auscultation should fix his attention in a special manner upon the expiration. Bronchial breathing is sometimes audible at some distance from its point of origin.

Two theories have been held regarding the mode of production of bronchial breathing, viz.:

(1) The theory of Laennec: that it is produced in the larynx and conveyed thence to the ear.

(2) The theory of Skoda: that it is due to *consonance*, the bronchial respiration being generated or magnified in the bronchi of condensed lung tissue or in cavities by the air in

the bronchi or cavities vibrating in consonance with that in the trachea.

The former view seems to me the more probable.

Bronchial Breathing in Disease.—Bronchial breathing heard outside the physiological areas is a sign of great importance. It is found in the following conditions:

1. In consolidation of the lung (whether due to fibrinous exudation, tubercle, induration, fibrosis, neoplasms, or infarct), provided the main bronchi remain open.
2. In pulmonary collapse.
3. In many cases of pleural effusion, especially in children.
4. Over some cavities, tuberculous or bronchiectatic.

(1) *Bronchial breathing in consolidation of the lungs.*—Bronchial breathing in pulmonary consolidation depends upon the increased conducting capacity of consolidated pulmonary tissue, and upon the fact that under such conditions the air conveying the sound is less dissipated through the side passages. The denser the consolidation, the more pronounced the bronchial breathing. In very dense consolidations, *e.g.*, in many cases of pneumonia, bronchial breathing assumes the tubular type, *i.e.*, there is a certain added metallic and hollow quality, suggestive of breathing through a metal tube. In loose consolidations, or where dense consolidations are in process of resolution, bronchial breathing tends to become vaguely blowing and weak. Patency of the main bronchi in cases of consolidation is a necessary condition for the production of bronchial breathing. If the bronchial tubes become filled with secretion, bronchial breathing ceases. This is frequent in pneumonia, and may give rise to the unfounded inference that a sudden pleural effusion has set in. Attention to the position of the cardiac impulse will help to obviate this error.

(2) *Pulmonary collapse.*—The bronchial breathing present in this condition is sometimes well marked, sometimes weak and 'distant' in quality. Diagnosis is assisted by the co-

existing retraction of the side, and by the frequently ill-marked vocal fremitus and vocal resonance.

Atelectasis from compression causes bronchial breathing, while atelectasis from blocking of the bronchial tubes with secretion causes silence over the part.

(3) *Pleural effusion.*—In the majority of cases of pleural effusion bronchial breathing is audible at some stage of the process in the interscapular region. The explanation is that the lung retracts towards its root, and, being compressed, its conducting power is increased. These cases are well known, and need give rise to no difficulty. Much more important are those cases, by no means rare, where bronchial breathing is heard over the entire affected side in pleural effusion, a phenomenon oftener observed in the child than in the adult. The explanation of bronchial breathing in these cases is not difficult. Fluid is a good conductor of sound, and if the large bronchi remain patent there is no reason why bronchial breathing should not occur in pleural effusion. Skoda held that the bronchial breathing present in such cases is indistinguishable from the bronchial breathing of consolidation. This may be so, but I have an impression that the bronchial breathing of pleural effusion has not usually the loud, insistent quality which is common in pneumonic consolidation, that it usually seems somewhat removed from the ear, and that the expiration has its peculiar characters less marked than the expiration in cases of consolidation. In such cases we must pay special attention to the state of the vocal fremitus and the position of the apex-beat of the heart.

(4) Bronchial breathing is heard over cavities of moderate size, near the surface, containing air, and communicating with a bronchus. It is sometimes best heard after coughing. The distinction between bronchial breathing and cavernous breathing is sometimes very difficult, and the most experienced ear may occasionally be in doubt upon this point.

The intensity and the rhythm of bronchial breathing present many variations, and definite rules upon these points can hardly be formulated.

3. *Cavernous Breathing.*—Cavernous breathing resembles bronchial breathing, but it is more hollow, of lower pitch, and it has a peculiar echoing quality, due to consonance in a cavity. To give rise to cavernous breathing, a cavity must be of considerable size, contain a considerable quantity of air, be somewhat superficial in position, and communicate freely with a bronchus. Some observers are of opinion that in cavernous breathing the expiration is always of lower pitch than the inspiration, while in bronchial breathing the reverse is the rule. Amphoric breathing may be considered a variety of cavernous breathing. It has a peculiar metallic quality, comparable with the sound produced by blowing into a large, empty bottle. It is characteristic of large cavities.

A type of breathing not easily distinguished from cavernous is sometimes heard about the mammary region in pleural effusion.*

Over bronchiectatic cavities the breathing is usually either cavernous or simply vaguely blowing.

In investigating the state of the breathing in any case of difficulty, we must bear in mind that the sound which we are observing may be a mixed sound, partly physiological, partly pathological, or that it may be partly a sound produced *in loco*, partly a sound conveyed from adjacent portions of lung. Complications of many kinds may thus arise. Our safety will lie in steadily bearing in mind the physical laws which regulate the production of the various forms of breathing, and in comparing the breath sounds with the information yielded by other methods of observation. Auscultation is not to be relied upon to the exclusion of other modes of examination.

* Gee, 'Auscultation and Percussion,' p. 221.

B. Adventitious Sounds.

The subject of adventitious sounds in the lungs and pleura is one of the most difficult in the whole range of medicine. A good deal of doubt hangs over the physical theories which are invoked to account for some of these sounds, and consequently the terminology of the subject is unsettled and confused, and the learner is bewildered by terms difficult of precise definition or of easy application at the bedside.

I shall approach the subject from the point of view of the physical mechanism or mode of production of the various sounds.

It is evident, I think, that we require terms to express the following conditions:

1. The rubbing of one pleural surface upon another.

2. The passage of air through a bronchiole of which the calibre is diminished by swelling of the mucous membrane or by the presence of mucus, or where, owing to irregular swelling of the mucous membrane, the entire wall of the bronchus is thrown into vibration by the current of air forcing its way through the narrow places.*

3. The passage of air through fluid in a bronchiole—large, medium-sized, or small—the surrounding lung being healthy.

4. The passage of air through fluid in a bronchiole—large, medium-sized, or small—the surrounding lung being infiltrated, condensed, or consolidated.

5. The opening up of collapsed infundibula or collapsed alveoli.

6. The bursting of bubbles of air in fluid contained in a cavity.

The following terms might be employed to express these ideas:

1. Pleural friction.

* F. A. Hoffmann, 'Nothnagel's Encyclopædia of Practical Medicine, English edition, article 'Diseases of the Bronchi.'

2. Rhonchus—sonorous when the large tubes are involved; sibilant when the small tubes are involved.

3. Bubbling, non-consonating râle—large, medium-sized, or small.

4. Consonating râle—large, medium-sized, or small.

5. Crepitant râle.

6. Gurgling râle (cavernous râle).

In practice, however, these distinctions are not strictly observed. A consonating râle and a crepitant râle are not usually differentiated, and it must be admitted that the distinction is often difficult.

There is the further difficulty of distinguishing 'dry' from 'moist' râles. A dry râle is taken by some writers as synonymous with 'rhonchus,' which is the more legitimate use of the term; while others take it to express a bubbling or crepitant râle of a certain sticky quality, suggesting that there is only a very little fluid in the bronchiole or alveolus.

Laennec classified râles as follows:

1. Le râle crépitant humide ou crépitation (crepitant râle).

2. Le râle muqueux ou gargouillement (mucous râle).

3. Le râle sonore sec ou ronflement (sonorous rhonchus).

4. Le râle sibilant sec ou sifflement (sibilant rhonchus).

5. Le râle crépitant sec à grosses bulles ou craquement* (dry crackling).

These types of râle are all easily identified, except the last. Laennec compared 'dry crackling' to the sound produced when we inflate a dry bladder. He regarded the sound as typical of emphysema.

Skoda's classification of râles was as follows:

1. Das vesiculare Rasseln (crepitant râle; râle originating in fine bronchioles and air cells).

2. Das konsonirende Rasseln (consonating râle).

3. Das trockene knisternde Rasseln mit grossen Blasen (dry, crackling râle).

* Laennec, 'Traité de l'Auscultation Médiate,' vol. i., pp. 98, 99.

4. Unbestimmte Rasselgeräusche (indeterminate râles).

5. Das Rasseln mit amphorischen Wiederhalle und metallischen Klange (râle with amphoric echo and metallic ring).

6. Das trockene sonore und das pfeifende Rasseln (dry, sonorous, and sibilant râle).

The classifications of Laennec and Skoda have formed the basis of all subsequent attempts to introduce order into this difficult department of medicine, but it is evident that agreement either in theory or in terminology has not yet been attained.

Without attempting any new classification, let us consider the various forms of adventitious sound, and endeavour to lay down, as far as may be possible, the guiding rules which are capable of application at the bedside. It will be found that our knowledge, although incomplete, is sufficient to meet most of the necessary indications for observation and diagnosis.

1. *Pleural Friction.*—Pleural friction has usually a rubbing, grating, or scraping quality. It is generally heard both during inspiration and expiration ; it is sometimes increased by pressure of the stethoscope ; it is usually (but not always) accompanied by the subjective sense of pain ; it may be heard over any part of the chest, but it is most often heard in the inferior lateral and antero-lateral regions ; it is due to the rubbing together of inflamed pleural surfaces. Most observers are agreed that pleural friction has occasionally a crackling or crepitant quality, which may lead to difficulty in diagnosis. We shall return to this last point in a future lecture.

2. *Rhonchus, Sonorous and Sibilant.*—A rhonchus is easily recognised. It is a snoring or sibilant sound, musical in character, usually heard both during inspiration and expiration, varying in pitch according to the calibre of the tube in which it is produced, sometimes changing position or disappearing after coughing. It is produced by narrowing of

a bronchiole, either from the presence of mucus or inflammatory swelling of the mucous membrane, or to irregular swelling of the mucous membrane, causing the wall of the bronchiole to be thrown into vibration as the air passes in and out.

Rhonchi may be heard in all pulmonary affections of which bronchial catarrh forms a part. They tell us nothing of the condition of the adjacent lung.

3. *Bubbling, Non-Consonating, Mucous Râle.*—This sound is due to the passage of air through fluid in a bronchiole. According to the size of the tube in which it is produced, this râle may be coarse, medium-sized, or fine. The term 'dry' should never be applied to this râle. Its non-consonating quality shows that the surrounding lung tissue is normal, or at all events has not undergone any change which can increase its capacity for conducting sound. This râle is heard both in inspiration and expiration. It may occur in any condition where there is fluid in the bronchial tubes and air is passing in and out, the surrounding lung tissue being normal, or approximately normal—*e.g.*, in bronchitis.

4. *Consonating Mucous Râle.*—This sound is due to the passage of air through fluid in a bronchiole, the surrounding lung tissue being infiltrated, condensed, or consolidated. The beginner should familiarize his ear with the ringing quality of this râle, which at once conveys information of importance. Like the non-consonating mucous râle, this râle may be large, medium-sized, or small, according to the size of the bronchiole in which it is produced.

The consonating mucous râle is heard in pneumonia, pulmonary cirrhosis, pulmonary infarction, tuberculosis, and other conditions.

This râle is heard both during inspiration and expiration.

5. *The Crepitant Râle.*—This râle is caused by the entrance of air into collapsed infundibula and alveoli, or to the opening

up of alveoli of which the walls have been glued together by inflammatory exudation. It is essentially inspiratory.

This râle is heard in the first stage of pneumonia*; in cases of pleural effusion when there is a small residue of pleuritic exudate; in pulmonary collapse and œdema. It may also be heard in bronchitis, whooping-cough, pneumonia, and phthisis, when the physical conditions necessary for its production, as above defined, are present.

It would be a great gain to clearness and precision in pulmonary terminology if the above definition of 'crepitant râle' or 'crepitation' were strictly observed, but in practice 'crepitant râle' and 'consonating râle' are often used indiscriminately.

6. *Gurgling Râle* (*Cavernous Râle*).—This râle is due to the bursting of air-bubbles in fluid contained in a cavity, and has a peculiar hollow quality, due to resonance. This râle presents many variations according to the following conditions: The size of the cavity; the condition of its walls; the amount and character of the fluid in the cavity; the degree of communication between the cavity and the bronchi. The larger the size of the cavity, *ceteris paribus*, the more hollow the râle; the thicker the fibrous envelope around the cavity, the more 'consonating' will probably be the character of the râle; the more abundant the fluid in the cavity, the 'moister' will be the râle. The râles produced in a cavity may be bubbling or consonating or 'croaking' in quality.

The subject of pulmonary adventitious sounds remains in a somewhat unsatisfactory state. The terms at our disposal are neither very happily chosen, nor strictly defined, nor rigorously applied. But even more important than the strict use of terms is a constant regard, so far as our knowledge goes, to the physical mechanism by which sounds are pro-

* Some observers believe that the so-called 'true crepitus' of the first stage of pneumonia is really a pleural friction sound. See Osler, 'Practice of Medicine,' sixth edition, p. 176.

duced. In a doubtful case we should ask ourselves such questions as the following:

(*a*) Does this sound suggest a narrowing of the calibre of a bronchiole, or the presence of fluid in a bronchiole, or the opening up of a collapsed infundibulum or air vesicle?

(*b*) If the sound suggests fluid in a bronchiole, is it produced in a large, medium-sized, or small bronchiole?

(*c*) Does the quality of the sound suggest that the adjacent lung is spongy and air-containing, or that it is infiltrated, condensed, or collapsed?

(*d*) Does the sound suggest the peculiar hollow quality due to resonance in a cavity?

With care the above questions can usually be answered with confidence.

LECTURE IV

PHYSICAL EXAMINATION OF THE LUNGS AND PLEURA (D)

SUMMARY:

Metallic tinkling.
Amphoric hum.
Succussion splash.
Bruit d'airain, or coin sound.
Post-tussive suction.
Vocal resonance:
  Bronchophony.
  Pectoriloquy.
  Ægophony.
Mensuration of the chest.
Radioscopy and radiography in pulmonary disease.

AUSCULTATION (*concluded*).

The following auscultatory phenomena remain for consideration:

(*a*) Metallic tinkling.
(*b*) Amphoric hum.
(*c*) Succussion splash.
(*d*) *Bruit d'airain*, or coin sound.
(*e*) Post-tussive suction.

The above sounds have this point in common, that they are heard only in connection with air-containing cavities, pulmonary or pleural.

(*a*) METALLIC TINKLING (TINTEMENT MÉTALLIQUE).—This sound was compared by Laennec to the sound produced

when a metal or porcelain cup is lightly struck by a pin, or when a grain of sand is dropped into it.* It may be heard when the patient breathes, speaks, or coughs. It indicates pneumothorax with either a serous or purulent effusion, or a large pulmonary cavity filled with liquid pus.

(*b*) Amphoric Hum (Bourdonnement Amphorique).—Laennec compared this sound to the sound produced by blowing into a carafe or pitcher. It may be heard in connection either with the cough, the breathing, or the voice. It indicates the presence of a large air-containing cavity.

(*c*) Succussion Splash.—This sound was known to Hippocrates. It is produced by seizing the patient and giving him a sudden shake, when a splashing sound is heard by the auscultator. It indicates the presence of air and fluid in the pleural cavity.

Succussion splash is usually easy of recognition. In exceptional cases splashing sounds in the stomach may give rise to difficulty.

(*d*) Bruit d'Airain, or Coin Sound.—This well-known sound is produced by placing a penny or half a crown in contact with the chest and striking it with another similar coin. A peculiar chiming sound is produced in presence of pneumothorax or large pulmonary cavity.

(*e*) Post-Tussive Suction.—This is a peculiar sucking sound, sometimes heard over cavities during inspiration after coughing. It is somewhat rare.

C. Vocal Resonance.

When we listen through the stethoscope while the patient speaks, the voice conduction may be modified in the following ways:

(*a*) It may be increased or diminished in loudness.

(*b*) It may be increased or diminished in distinctness.

(*c*) It may be altered in tone or quality.

* Laennec, 'Traité de l'Auscultation Médiate,' vol. i., p. 113.

It is impossible, in the present state of our knowledge, to keep these elements entirely distinct.

By *bronchophony* we understand that condition in which the voice is conveyed with increased intensity and heightened pitch, and with either increased or diminished distinctness.

By *pectoriloquy*, following Laennec and the French school, we understand that condition in which the voice is conveyed, as it were, directly into the tube of the stethoscope. The voice is not louder than in health—it may be much less loud—but it has always the quality of abnormal distinctness, and direct conduction to the ear.

By *ægophony* we understand a peculiar bleating, twanging, or quavering note occasionally heard in the interscapular region near the upper level of a pleural effusion.

Laennec distinguished bronchophony from pectoriloquy, and taught that this latter sign was pathognomonic of cavity. Skoda denied that there was any real distinction between bronchophony and pectoriloquy, or that the latter was a trustworthy sign of cavity.

Bronchophony.—The causes of bronchophony are identical with those of bronchial breathing, and the two signs are always associated. Bronchophony occurs under the following conditions:

1. In all conditions in which the lung parenchyma is deprived of air and rendered more or less solid, and in which the bronchial tubes remain patent—*e.g.*, pneumonia, tuberculosis, pulmonary fibrosis, pulmonary infarction, pulmonary neoplasm.

'Pneumonia in its first stage, inflammation confined to a few lobules of the lung, lobular hepatization, œdema of the lungs, or limited effusion of blood into the lung parenchyma, produce very little or no increase whatever in the strength of the thoracic voice; neither do solitary tubercles, however numerous, provided the intervening parenchyma remains pervious to air' (Skoda).*

* J. Skoda, *op. cit.*, p. 44.

2. In cavities of a certain size which contain air and communicate freely with a bronchus.

3. In certain cases of pleural effusion.

4. In pulmonary collapse.

I have included pulmonary neoplasm amongst the possible causes of bronchophony more on theoretical grounds than on those of experience. My experience of this condition is limited to eight cases, and in all weak breathing and diminished vocal resonance were present. Nor is this in any way surprising. Tumours of the lung, by the time they have given rise to signs and symptoms which render diagnosis possible, almost invariably compress a main bronchus, and hence give rise to the well-known symptom-complex of compression atelectasis. But it seems probable in theory that at an earlier stage pulmonary tumour might cause bronchial breathing and bronchophony. The bronchophony heard over a cavity does not call for comment. The bronchophony sometimes heard over pleural effusion depends on the same physical conditions as the bronchial breathing sometimes heard in that condition. The same remark is applicable to the bronchophony of pulmonary collapse.

PECTORILOQUY, as Laennec taught, is common over cavities, but it is not invariable in such conditions, while it is heard in some cases of consolidation. Pectoriloquy suggests the probability of cavity, but it cannot be relied upon in the absence of other signs. Flint thought that when bronchophony and pectoriloquy co-existed consolidation might be diagnosed, but that from pectoriloquy alone a cavity might be inferred.

ÆGOPHONY is a comparatively rare sign, which has been generally attributed to compression of the bronchial tubes by pleural effusion. Sahli is of opinion that any attempt to differentiate sharply bronchophony, pectoriloquy, and ægophony is futile.*

* *Op. cit.*, p. 241.

Speaking generally, increased vocal resonance suggests the probability of either pulmonary consolidation (pneumonic, tubercular, compression atelectasis) or of cavity. Diminished vocal resonance suggests the probability of pleural effusion; thickening of the pleura; consolidation of the lungs with blocking of the bronchial tubes; compression of the lungs by aneurysm or neoplasm with blocking of the tubes.

On the whole, vocal resonance is not one of the most trustworthy of the physical signs in the chest, and the beginner should beware of drawing conclusions from it without a careful collation of all the other physical signs present in any given case. He should also remember that the distinction drawn so confidently in many text-books between bronchophony, pectoriloquy, and ægophony does not rest upon any sure basis of observation, and is open to serious question.

In some cases of pleural effusion what we observe as regards voice conduction is not so much diminished vocal resonance—*i.e.*, a less loud voice conduction—as an alteration of tone. The voice sounds not so much feeble as flat and toneless.

I append a brief syllabus of the auscultatory signs found in the principal affections of the lungs and pleura.

1. Acute Bronchitis.—(*a*) The breath sounds retain their vesicular quality. Their intensity is variable—normal, harsh, or weak; normal in cases of slight intensity; harsh when there is a moderate obstruction to the passage of air through the bronchi; weak when there is much mucous obstruction, or when the breathing is superficial. The rhythm may be normal, but the expiration is often prolonged.

(*b*) Adventitious sounds of various kinds are usually present, but bronchitis may exist without adventitious

sounds. Sonorous and sibilant râles are frequent. Bubbling râles (large, medium, and small) may be present. Consonating râles do not occur unless there is some complication, involving condensation or consolidation of the lung tissue.

(*c*) The vocal resonance is usually unaltered, so long as the bronchial tubes remain patent. It is reduced when obstruction of the tubes takes place.

2. CHRONIC BRONCHITIS AND EMPHYSEMA.—(*a*) The breath sounds are vesicular in quality; their intensity is diminished, and may be very weak; the expiration is prolonged, and may even be longer than inspiration.

(*b*) Adventitious sounds are usually, but not invariably, present—generally rhonchi (sonorous or silibant) and bubbling râles (coarse or medium).

(*c*) Vocal resonance is unaltered.

3. BRONCHIECTASIS.—(*a*) The breath sounds are cavernous, amphoric, or vaguely blowing; of variable intensity; the expiration is often prolonged; the cog-wheel character may be present.

(*b*) Adventitious sounds are usually present—rhonchi and râles (bubbling, consonating, gurgling, croaking).

(*c*) Bronchophony and pectoriloquy are frequent.

4. BRONCHIAL ASTHMA.—(*a*) The breath sounds are vesicular; normal in intensity, or weak; the expiration is prolonged.

(*b*) Adventitious sounds are often present—rhonchi and râles (bubbling, coarse, or medium-sized).

(*c*) Vocal resonance is unaltered.

5. PLASTIC BRONCHITIS.—The auscultatory signs cannot be distinguished from those of acute and chronic bronchitis.

6. PNEUMONIA.—(*a*) The breath sounds may be vesicular, harsh, bronchial, tubular; of variable intensity; rhythm normal or expiration prolonged.

(*b*) Adventitious sounds are present—friction, crepitant râles, bubbling râles, consonating râles, rhonchi.

(*c*) Bronchophony is frequent, pectoriloquy sometimes occurs; the vocal resonance may be diminished.

7. Broncho-Pneumonia.—(*a*) The breath sounds may be vesicular, broncho-vesicular, or bronchial in quality; of variable intensity; rhythm normal or expiration prolonged.

(*b*) Adventitious sounds are present—rhonchi (usually sibilant) or râles (fine, crepitant, consonating).

(*c*) The vocal resonance is variable.

8. Passive Congestion and Œdema.—(*a*) The breath sounds are vesicular or feebly blowing; intensity weak; rhythm normal or expiration prolonged.

(*b*) Adventitious sounds are present—rhonchi and râles (fine, bubbling, crepitant, or consonating).

(*c*) Vocal resonance is normal or slightly increased.

9. Cirrhosis of Lungs.—(*a*) The breath sounds are cavernous, amphoric, or vesicular; intensity variable, but often weak; rhythm normal or expiration prolonged.

(*b*) Adventitious sounds are present—râles (bubbling, crepitant, or consonating).

(*c*) Vocal resonance is increased.

10. Tumour of the Lungs.—(*a*) Breath sounds vesicular or bronchial; intensity variable, often weak; rhythm normal or expiration prolonged.

(*b*) Adventitious sounds are sometimes present—rhonchi and râles (bubbling, crepitant, or consonating).

(*c*) Vocal resonance variable, but often diminished.

11. Tuberculosis of the Lungs.—(*a*) Breath sounds vesicular, broncho-vesicular, bronchial, or cavernous; intensity variable; rhythm normal or expiration prolonged.

(*b*) Adventitious sounds are usually present—rhonchi, râles (bubbling, crepitant, consonating, fine, medium, coarse).

(*c*) Vocal resonance variable, usually increased.

12. Pleural Effusion.—(*a*) Breathing suppressed, or vesicular, or bronchial; intensity variable, but usually weak; rhythm normal or expiration prolonged.

(*b*) Adventitious sounds—friction, râles (consonating).

(*c*) Vocal resonance variable, often reduced; bronchophony or ægophony may be present.

13. PNEUMOTHORAX.—(*a*) Breath sounds amphoric or tubular; intensity variable, often weak; expiration prolonged or absent; amphoric hum may be present.

(*b*) Adventitious sounds usually present—metallic tinkling, *bruit d'airain*, succussion splash, râles (consonating).

(*c*) Vocal resonance is diminished.

MENSURATION OF THE CHEST.

Mensuration is important as giving us a gauge of pulmonary capacity and expansion. Accurate measurements are not easily made, nor has the cyrtometer proved of much practical utility.

The semicircumference of the right side of the chest usually exceeds that of the left by from half an inch to an inch. The semicircumference of the affected side in pleural effusion may exceed that of the sound side; but this rule is by no means universal, and cannot be relied upon. The following passage from Gee's well-known book sets forth clearly the difficulties of drawing conclusions from chest measurements: 'Circumferential measurements of the two sides are often made; but be it remembered, first, that considerable increase of the sectional area of the chest may occur and the length of the periphery remain the same, by the passage of the elliptical form into the circular; and, next, that the displacement of the mediastinum, which accompanies unilateral enlargement, thrusts the heart into the unaffected side. And this consideration, too, that the walls of the healthy side must follow the antero-posterior projection of the diseased side, and then it will be plain why, as a matter of fact, the perimeter of the expanded side often

measures very little more—nay, even less—than that of the side which is not diseased.'*

The periodic measurement of the chest at the mammary line is to be recommended as a good routine practice in pulmonary phthisis. It gives valuable information regarding the progress of the case, and throws important light upon the amount of compensatory hypertrophy taking place in the sound (or comparatively sound) lung, upon which in considerable measure the prognosis of the case depends.

Increased expansibility in phthisis suggests the following possibilities:

(*a*) Compensatory hypertrophy of the sound lung.

(*b*) Emphysema in the neighbourhood of the diseased areas.

(*c*) Pneumothorax.

(*d*) Pleural effusion.

Diminished expansibility in phthisis suggests the following possibilities:

(*a*) Progress of tuberculization and softening.

(*b*) A tendency to fibrosis and chronicity of course.

The most extreme degrees of diminished expansibility are found in the cases of so-called 'fibroid phthisis,' which is only a very chronic form of tuberculosis, accompanied by much fibroid change.

In pneumothorax and in thoracic tumour the semicircumference of the chest is usually increased on the affected side.

THE RÖNTGEN RAYS IN THE DIAGNOSIS OF DISEASES OF THE LUNGS

By Dr. David Lawson

The application of the Röntgen rays to diagnosis in medicine depends upon two properties which they possess—viz. (1) their power of causing certain substances, such as

* S. Gee, 'Auscultation and Percussion,' third edition, p. 29.

barium platinocyanide and calcium tungstate, to fluoresce; (2) their capability of acting upon a photographic plate so that a negative can be obtained from it.

Certain bodies interposed in the course of the rays have the power of partially or completely intercepting them. That portion of a fluorescent screen—whose surface is commonly covered by a preparation of barium platinocyanide—corresponding to the intercepted rays will show a shadow, and a print from a radiographic negative (called a skiagram) similarly treated will present exactly the same appearance.

In applying the Röntgen rays to the diagnosis of lung conditions, it has to be borne in mind that whilst a healthy lung presents little or no obstruction to the rays, it is not so in the case of numerous diseased conditions. Where less air than normal is present in a portion of a lung—as in collapse or consolidation—its absence is revealed by a shadow. On the other hand, conditions associated with the presence of an excess of air over the normal—such as emphysema—are recognised by a lighter appearance than is usually to be found in the normal state.

Methods of Applying the Röntgen Rays to Pulmonary Disease.

These methods are four in number—viz.:

1. Stereoscopic.
2. Orthodiagraphic.
3. Radioscopic.
4. Radiographic.

The first two may be dismissed as being merely variations of numbers 3 and 4, of minor clinical interest and little practical value. But since it is upon the radioscopic and radiographic methods that the physician must rely, it will be necessary to deal with these methods more fully.

In employing the radioscopic method the observer uses a fluorescent screen, while in the radiographic method a photographic plate is substituted for the screen. By means of the radioscope he obtains a temporary, by means of the radiograph a permanent, impression. Most of the information obtained by the Röntgen rays is available equally by both methods. As, however, there are certain facts ascertainable by the radioscope alone which cannot be obtained by means of the radiograph, it will be well to refer to them separately. It will be convenient to deal with these in the first instance.

A. Information Obtainable by Radioscopy Alone.

1. Variations in the Illumination of the Chest.—Under normal conditions the appearance shown on the screen at the apex of the left lung is, in the majority of cases, somewhat lighter than that shown at the apex of the right lung. But, during the inspiratory phase of respiration, the two apices possess this characteristic in common—that both appear for the time being lighter than before the additional air has been inhaled. Failure on the part of one apex to light up during inspiration should always be considered a ground for suspecting the presence of early tubercular involvement. Moreover, it has been further observed that shadows due to the presence of old healed lesions withstand all effort to illuminate them in this way.

2. Movements of Solid Parts.—By far the most important of these are the movements of:—

(*a*) The Diaphragm. On the right side the diaphragm normally stands at a higher level—viz., about $\frac{1}{2}$ inch—than on the left. There is also a disparity in the diaphragmatic movements on the two sides, the maximum downward movement which takes place during forced inspiration on the right side being roughly $2\frac{7}{8}$ inches as compared with $2\frac{5}{8}$ inches on the left side. It is now admitted by general consent that one of the earliest changes which take place in the chest when a

tubercular process starts at the apex of one lung is impaired movement of the diaphragm upon this side. Hence, interference with the normal relationship of the diaphragmatic movements on the two sides is of cardinal importance. Deficient diaphragmatic movement on the affected side has frequently been detected at a stage so early that no shadow was discernible at the seat of the lesion. This sign has been explained as the result of reflex action, the different impulses passing by the vagus, and the efferent impulses by the phrenic nerve, (G. A. Gibson).

Complete fixation of the diaphragm on one side indicates the presence of an old pleurisy with adhesions.

(*b*) The ribs. The localized impaired movement of the ribs over a pulmonary lesion is readily recognised.

(*c*) Enlarged bronchial glands, occurring as they do in the vicinity of the aorta, have been mistaken for aortic aneurysm or for neoplasm. Where radioscopy is employed the characteristic pulsating movement of the shadow caused by an aneurysm when present at once settles the point.

3. Movements of Fluid.—At no time does the radiographic method more richly reward the efforts of the observer than when he employs it to differentiate between the presence of fluid alone and of fluid plus air in the pleural sac. In the former case, when the patient is examined in the upright posture, the upper level of the shadow caused by the fluid is somewhat indefinite in outline and takes the form of a curve, the concavity of which is in the centre. In the latter case the shadow is particularly definite and uniform, and instead of being curved it is absolutely horizontal. Moreover, if the patient be made to lean over at an angle of 45 degrees, whereas in the former case the upper level of the fluid will be observed to have moved with the patient, it is not so in the latter case. Where air is present with the fluid the level of the fluid remains horizontal, no matter what the position of the patient may be. Above all, a rhythmical, rippling move-

ment, corresponding to the movements of the heart, is observable on the surface of the fluid in the latter case, which is never present when the shadow owes its existence to the presence of fluid only. A close attention to these matters enables us definitely to distinguish between such conditions as ordinary pleural effusion, empyema, or hydrothorax on the one hand, and hydropneumothorax or pyopneumothorax on the other.

B. Conditions Recognisable Equally by Radioscopy and Radiography.

It is convenient to divide the conditions to be studied under this head into two classes—viz.:

(*a*) Intrapulmonary, and
(*b*) Extrapulmonary.

(*a*) Intrapulmonary Conditions—1. *Infiltration and Consolidation.*—In this case the affected portion of the lung is recognised by the presence of a shadow, and the characters of the shadow and of the surrounding parts depend upon whether the condition is acute or chronic. Whilst early or acute infiltration is recognised by a uniform and not altogether clearly-defined shadow, it is not so in the case of chronic infiltration, in which the shadow, though on the whole denser, is not so uniform, and is sometimes relieved by small light patches, so that the whole presents a mottled appearance. During the early stage of infiltration no appreciable change in the appearance of the ribs overlying the consolidated area is observed. But it is not so when the mischief is of older standing, for in this case a localized approximation of the ribs, termed by some 'roof-tiling,' from the suggestive appearance which they present, is usually to be found. A further point of difference between the two conditions consists in the disturbance caused in the relationship of the surrounding organs. Whilst solid organs in the

neighbourhood, such as the heart, tend to be drawn towards the older lesion, these organs do not suffer disturbance in the more acute conditions.

2. *Collapse.*—The appearance presented by a collapsed lung varies according to the duration of the condition. Thus, a temporarily collapsed lung caused by pneumothorax appears as a light speckled shadow located near the middle line close to the root of the lung and surrounded by the intensely bright appearance characteristic of pneumothorax. The overlying ribs in this case are seen usually far apart from each other. On the other hand, a lung permanently collapsed, as, for example, after a severe attack of empyema of long duration, is recognised by the presence of a very dense, dark, nearly black shadow, over which the ribs are lying so closely crowded together as to be nearly touching each other. Moreover, the transverse diameter of the radius of the chest upon that side is very much smaller that it is upon the healthy side.

3. *Cavity.*—Cavities occurring during the process of caseation appear as light areas in the midst of shaded portions, whose edges are not clearly defined. The appearance presented by a chronic cavity of long standing is quite different. The existence of a thick cover of dense fibrous tissue surrounding the cavity no doubt accounts for the clearly-defined wall which can be observed marking off the clear from the shaded portion.

4. *Emphysema.*—This condition is often present as compensatory emphysema. It is easily recognised by the strong contrast which the extremely light appearance of the emphysematous portions presents as compared with the dark portions corresponding to the disease in their vicinity, and also by the fact that the ribs overlying the emphysematous portion of the lung are widely separated from each other, as opposed to the 'roof-tile' arrangement of ribs in other portions of the same skiagram.

(*b*) EXTRAPULMONARY CONDITIONS—1. *Glands.*—The bronchial glands which lie near the bifurcation of the trachea and along the larger bronchi, when enlarged and calcareous, are easily recognised by the isolated dark patches, which may be as large as the size of an ordinary marble.

2. *Fluid in the Pleural Sac.*—The chief characteristic points are:

(*a*) The diaphragm is not discernible on the affected side.

(*b*) A uniform shadow is seen extending from the base of the chest upwards, and merging gradually in a clear area above.

(*c*) The upper border of this shadow is usually concave, with the concavity in the centre.

(*d*) The heart is usually displaced towards the opposite side.

(*e*) The appearance of the lung above the fluid corresponds to that of compensatory emphysema. A shadow caused by pus is believed to be darker than one due to serous fluid.

3. *Air in the Pleural Sac.*—Pneumothorax is recognised by—

(*a*) An abnormally light appearance on the screen or skiagram.

(*b*) A mottled, rounded area near the root, corresponding to the collapsed lung.

(*c*) Wide separation of the overlying ribs.

(*d*) Increase of the radial diametric measurements upon the affected as compared with the non-affected side.

THE RÖNTGEN RAYS IN THE TREATMENT OF PULMONARY DISEASE.

Some observers—*e.g.*, Hope Fowler of Edinburgh, Grubbe of Chicago, and Rudis-Jicinsky of Cedar Rapids—have claimed success in the application of the Röntgen rays to

the treatment of pulmonary disease, but their results have not been generally confirmed. It would seem that enlarged bronchial glands may be favourably influenced by the use of the rays, but erythema and ulcer of the overlying skin are frequently produced, a fact which constitutes a serious disadvantage. My own experience of the Röntgen rays as a therapeutic agent in lung conditions is not encouraging, and I no longer employ them in this connection.

LECTURE V

THE STUDY OF PULMONARY SYMPTOMS (A)

SUMMARY:

Modes in which pulmonary symptoms arise.

Classification of pulmonary symptoms:

A. Respiratory symptoms proper, viz., cough, dyspnœa; types of dyspnœa: the dyspnœa of pneumonia, œdema of the lungs, capillary bronchitis, pleurisy, heart disease, uræmia, anæmia, diabetic coma.

Stridor, loss of voice, pain in the chest.

Pulmonary disease may produce symptoms in any of the following ways:

1. By interfering with the ease, completeness, and rhythm of the respiratory movements.

2. By impeding the free passage of air to and from the bronchi and air cells.

3. By preventing the free interchange of gases in the air cells.

4. By indirect action upon other organs, such as heart, digestive system, kidneys, brain, blood.

The symptom-complex is thus in many cases many-sided. The blood condition which results from the above causes in pulmonary disease has as its leading feature deficient oxygenation of the blood. The relations of respiratory with circulatory disorder are close. Nutrition suffers comparatively little in acute respiratory disease, while it suffers much in chronic respiratory disease, such as chronic pleurisy, bron-

chiectasis, and, above all, pulmonary phthisis. The nervous symptoms of pulmonary disease are not usually amongst its most prominent features, but may be marked. Excretion by the kidney is only interfered with in a minor degree. The state of the bowels presents many variations, but on the whole exhibits few characteristic features. Abnormalities of the cutaneous system are occasionally present, but are only exceptionally of diagnostic importance. The pyrexia of pulmonary disease presents many types, some of them characteristic—*e.g.*, the temperature curve in pneumonia.

We may classify the symptoms of pulmonary disease on the following plan:

A. Respiratory symptoms proper—viz., cough, expectoration, dyspnœa, stridor, loss of voice.

B. Circulatory symptoms—viz., cyanosis, venous stigmata, malar flush, suffusion of the eyes, œdema, abnormalities of the pulse, epistaxis.

C. Nervous symptoms—viz., pain in the chest, rigors, insomnia, delirium, restlessness, convulsions, headache, somnolence, coma, tetany, subsultus tendinum, acute mania, hemiplegia, aphasia, photophobia, hiccough.

D. Cutaneous symptoms—viz., herpes, jaundice, erythema, pemphigus, purpura, sudamina, sweatings.

E. Digestive and nutritional symptoms—viz., nausea, vomiting, diarrhœa, constipation, emaciation, incurvation of the nails, clubbing of the fingers (*Trommelschlägelfinger*), tympanites.

F. General symptoms—viz., pyrexia, changes in the blood, fœtor.

A. Respiratory Symptoms Proper.

1. Cough.—Cough is an almost constant feature of pulmonary disease, but may be absent:—in young children; in cases of profound prostration of severe toxæmia; in terminal pneumonias; in the pulmonary complications of drunkards;

in some cases of pleural effusion; in some cases of incipient phthisis. In this last condition patients not uncommonly deny that they have any cough, while the relatives give a different story. Some patients deny that they have any cough, but admit that they bring up a certain amount of sputum.

The chief points to be noted about cough are the following:

(*a*) Its quality—whether hard, soft, brassy, ringing, barking.

(*b*) Its time—whether incessant or intermittent; whether occurring in definite paroxysms; whether worse by day or by night, in the early morning, after meals, after exertion, etc.

(*c*) Whether accompanied by secretion, which is the usual rule; or without secretion, which is the exception.

(*d*) Whether accompanied by pain, of which the two commonest types are substernal tenderness (bronchitis, emphysema, asthma), and 'stitch' in the side (pleurisy, pneumonia).

(*e*) Whether ever followed by vomiting, suffusion of the face, ecchymoses of the skin, convulsions, unconsciousness.

A cough is a reflex expiratory effort. The source of reflex irritation may be in—

(*a*) The upper air passages—pharynx, larynx, trachea.

(*b*) The lower air passages—bronchi, bronchioles, air vesicles, pleura.

(*c*) The ear and nose.

(*d*) The skin.

(*e*) The central nervous system.

(*f*) The stomach (?).

Whether cough ever occurs without some form of reflex irritation is doubtful. The well-known hysterical cough may depend upon some general condition of the nervous system,

or upon some obscure reflex irritation, magnified by a condition of abnormal responsiveness to stimulation. The 'stomach cough,' of which the laity speak so much, must be regarded as a very dubious phenomenon. It has often been my experience to find gross pulmonary lesions in cases where 'only a stomach cough' was alleged to exist. We cannot be too much on our guard against accepting the view that a cough is 'only nervous,' or 'only a stomach cough.' I have no doubt that in the immense majority of such cases there is some cause for the cough to be found in the respiratory tract. That cause is usually either swelling of the mucous membrane or the presence of secretion (mucus, pus, blood, necrotic or tuberculous material), or exceptionally of foreign bodies.

We shall briefly consider those qualities of cough which are of diagnostic significance, and then the characters of the cough which depend upon its cause or place of origin.

The Quality of the Cough.—The intensity of the cough depends partly upon the cause and seat of the cough, partly on the reflex irritability of the patient. The most intense forms of cough are due to the presence of a foreign body in the upper air passages, to laryngeal obstruction, to whooping-cough, and to extensive inflammation of the finer bronchioles or the lung parenchyma.

Swelling or ulceration of the vocal chords, or the presence of membrane in the larynx, causes a harsh, 'barking' cough, with hoarseness or loss of voice. A 'barking' cough is also found in hysteria, in pressure upon the recurrent laryngeal nerves (aneurysm, tumour), and paralysis of the abductors of the larynx.

A weak, 'toneless' cough is found in cases where there is imperfect closure of the glottis owing to ulcerative changes or paralysis of the abductors of the larynx; or where there is weak expiratory effort—*e.g.*, advanced phthisis, bulbar paralysis, myelitis, conditions of extreme debility and pros-

tration. A cough may be 'loose' or moist,' while no expectoration is voided, because the patient swallows it.

The Time of the Cough.—The chief points in this connection are whether the cough is markedly intermittent, and, if so, what rules govern the intermissions. No cough is absolutely incessant, for even in cases where a source of active reflex irritation is constantly at work—*e.g.*, a membrane in the larynx—the patient gets worn out at times, and ceases to cough from mere exhaustion; but a persistent cough, with few and brief intermissions, may be found in acute laryngitis, capillary bronchitis, pneumonia, or advanced phthisis. In all these cases, however, exceptions to this rule are not infrequent. A markedly intermittent cough is characteristic of whooping-cough and asthma, in which conditions the cough occurs in paroxysms at variable intervals. A cough confined to the morning, and absent throughout the day, is common in the slighter cases of bronchitis and in chronic cases of pulmonary phthisis attended by moderate or slight bronchial catarrh. A cough which disturbs sleep and awakes the patient is usually dependent on an accumulation of secretion in the respiratory tract, or on inflammation of the pulmonary parenchyma. A cough coming on after meals, or intensified by eating, is found in certain cases (*e.g.*, pulmonary phthisis) in which gastric catarrh exists as a complication of pulmonary disease.

The Character and Amount of the Secretion which accompanies the Cough.—The absence of secretion may be due to the fact that no secretion occurs, or, as usually in young children and not uncommonly in older patients, that the sputum is swallowed. The patient may swallow his sputum without being conscious of the fact. Sputum may be absent in a great variety of conditions—viz., in the so-called 'dry catarrh' of the larynx and bronchi, in affections of the nervous apparatus of the larynx, in the so-called 'hysterical cough,' in some cases of pneumonia, in many cases of

quiescent pulmonary tuberculosis. There is an important distinction between cases in which sputum has been absent throughout the attack and cases in which it has been abundant and has subsequently ceased. Thus, in pneumonia, the absence of sputum throughout the attack has no prognostic importance, while the disappearance of previously abundant sputum, unless convalescence is approaching, is often of grave augury. The disappearance of sputum previously abundant often signifies that the patient's capacity of expelling it has come to an end.

The characters of the sputum and the various inferences, diagnostic and prognostic, which may be based upon it are so numerous and important that a special lecture will be devoted to the subject.*

Symptoms which may accompany Cough.—Of these pain is one of the most frequent. It may take the form of either a substernal soreness, which is frequent in bronchitis; or of tenderness along the attachments of the diaphragm, which may occur in any case attended by severe coughing; or of 'stitch' in the side, which is characteristic of pleurisy.

Coughing often continues until the patient vomits. This is characteristic of the cough of whooping-cough, of certain cases of pulmonary tuberculosis, and of bronchiectasis. In this latter case this type of cough probably depends upon the effort to dislodge tenacious secretion from a cavity. Some diagnostic importance may be attached to coughing which culminates in vomiting. It is unusual in ordinary bronchial catarrh.

Suffusion of the face, ecchymosis on the skin, convulsions, the involuntary discharge of urine or fæces, and unconsciousness, occur in some forms of cough, and are to some extent a measure of its severity. These phenomena occur chiefly in children in connection with whooping-cough, asthma, capillary bronchitis, and broncho-pneumonia.

* See Lecture VII.

The chief localizing characteristics of cough have been indicated in the foregoing description. The beginner should accustom himself to distinguish carefully the cough of laryngeal obstruction; the cough depending on pressure on the trachea, bronchi, or recurrent laryngeal nerves; the cough due to the irritation of secretion in the bronchi; the cough arising from inflammation of the pulmonary parenchyma; the cough depending on pleural inflammation or pleural adhesions; and the functional or 'nervous' cough. These distinctions can be drawn with considerable confidence.

2. Dyspnœa.—The term 'dyspnœa' has been used in various senses, some authorities employing it in a wide sense to express abnormalities or difficulties of respiration in general, others defining it more strictly, and distinguishing dyspnœa from acceleration or slowing of the respiratory acts and from apnœa, and limiting it to cases where the breathing is laboured and forced, and where there is imperfect oxygenation of the blood. A further distinction has been sometimes drawn between *objective dyspnæa—i.e.*, dyspnœa attended by perceptible labour or difficulty of respiration—and *subjective dyspnœa*, where there is a consciousness on the part of the patient of difficulty or inadequacy of the respiratory act —*i.e.*, where there is a feeling of oppression of the breathing.

On the whole, these distinctions, although they can be easily drawn, seem to me of little practical value. It is impossible, I think, to define any one element as containing the essence of dyspnœa. The most general characteristic of these cases is imperfect oxygenation of the blood, but this is absent in 'hysterical' dyspnœa, a type of the condition which we cannot conveniently exclude from our definition. Acceleration of the respiratory act, though very frequent in dyspnœa, is not of the essence of the condition, because slowing is sometimes present. Nor does the subjective sense of respiratory distress on the part of the patient afford an

adequate basis for definition, as this is absent in the worst cases of dyspnœa, owing to extreme prostration or to poisoning of the respiratory centre with venous blood. I prefer, therefore, to take dyspnœa in a wide sense to indicate *all abnormalities in the frequency or rhythm of the respiratory acts, usually, but not always, involving the sense of respiratory distress, and usually accompanied by an imperfect exchange of gases in the pulmonary vesiculi.*

Dyspnœa, as might be expected, is one of the most constant of the symptoms of pulmonary disease, and its degree is a valuable gauge of the extent and severity of the morbid process in the lungs in any given case. In our discussion of the subject it will be convenient for us not to confine ourselves to the pulmonary causes of dyspnœa, but to deal with the subject in general. This is the more necessary, as many varieties of dyspnœa are most easily apprehended by attention to points of differential diagnosis.

Examination of a Patient suffering from Dyspnœa.—It is necessary to note the following points:

(*a*) The type, frequency, extent, and rhythm of the respiratory movements. We must observe whether the breathing is chiefly costal or chiefly abdominal; whether the excursion of the diaphragm is extensive or limited, painless or painful; whether the expansion of the chest is excessive or deficient; whether the respiratory movements are apparently attended by pain or not; whether inspiration and expiration preserve their normal relation; whether the movements of the chest are equal or unequal on the two sides; whether any part of one side shows any excess or defect of expansion; what is the number of respirations per minute; whether the respirations succeed one another at regular intervals or irregularly, and if irregularly, whether the want of rhythm conforms to any recognised type (*e.g.*, the Cheyne-Stokes variety); whether the regular muscles of inspiration and expiration are in normal or abnormal action; whether the accessory muscles

of respiration are being called into play; whether there is abnormal dilatation of the alæ nasi.

(*b*) The condition of the skin and mucous membranes—whether there is cyanosis or venous congestion, venous stigmata, œdema.

(*c*) The posture of the patient in bed—whether he sits or lies; if the latter, whether he lies by preference on one side or the other; whether he can lie back without distress or not; what posture he tends to assume while asleep.

(*d*) Whether there is excess or defect in the movements of the thyroid cartilage.

(*e*) Whether stridor—inspiratory or expiratory—is present.

Many of the foregoing points give us indications only as regards the severity of the dyspnœa. Some of them give us hints as to its cause.

The following are the usual characteristics of a severe type of dyspnœa: The respirations are notably altered in frequency, usually accelerated, exceptionally slowed; they are often shallow, occasionally more profound than normal; the rhythm of the respirations is altered, expiration being frequently, inspiration less frequently, prolonged; the accessory muscles of respiration are in action; the alæ nasi work strongly, especially in the child; recession of the lower costal spaces may sometimes be observed; cyanosis, more or less marked, may be present; the patient inclines to sit up in bed, so as to allow the normal and accessory muscles of respiration fuller play; there is an aspect either of anxiety or distress, or, in the worst cases, of apathy and diminished sensibility.

Types of Dyspnœa.—We may distinguish the inspiratory type, the expiratory type, and the mixed type. Inspiratory dyspnœa is characteristic of some obstruction to the entrance of air into the upper air passages, as in the following conditions:

Enlargement of the tonsils.

Adenoid growths in the naso-pharynx.

Retropharyngeal abscess.
Œdema of the glottis.
Acute laryngitis.
Foreign body in the larynx.
Spasm of the larynx, ('laryngeal crises' of tabes dorsalis).
Membranous laryngitis.
Tumours of the larynx or trachea.
Foreign body in the trachea.
Spasm of the adductors or paralysis of the abductors of the vocal cords.
Stenosis of the larynx.
Stenosis of the trachea.
Pressure on the larynx or trachea from without (tumours, enlarged glands, aneurysm).

Expiratory dyspnœa depends either on some obstacle to the exit of air from the air vesicles and bronchioles, or on diminished elasticity of the pulmonary parenchyma. It is found in its most typical form in chronic bronchitis, emphysema, asthma, uræmia.

Dyspnœa of mixed type is found in a great number of conditions—*e.g.*, pyrexia, heart disease, bronchitis, pleurisy, pneumonia, intercostal rheumatism, chlorosis, pernicious anæmia, whooping-cough, diabetic coma, cerebral hæmorrhage, some forms of poisoning, mediastinal tumour, pulmonary gangrene, carcinoma of the lungs, pulmonary œdema, aortic aneurysm, and other conditions.

We shall proceed to a more detailed description of the various forms of dyspnœa found in respiratory disease.

(1) *Dyspnæa depending on Obstruction in the Upper Air Passages.*—The causes of such obstruction have been already enumerated. The essential fact is the existence of some mechanical obstacle to the free entrance of air into the larynx and trachea. The characteristics of this form of

dyspnœa are stridor and either an increase or a diminution of the rate of respiration. If the former, the respirations will be superficial; if the latter, they will be deep, the organism seeking to compensate for the difficulty in the entrance of air either by an increased frequency, or an increased amplitude, of respiration. In consequence of the incomplete entrance of air into the thorax, there will be more or less recession of the supraclavicular and jugular fossæ, the epigastrium, and the lower intercostal spaces in the lateral regions. The downward movement of the diaphragm during inspiration is diminished.

Stridor is usually a marked feature of these cases, and is a point of great diagnostic significance. It is usually at first an exclusively inspiratory stridor, but as the case proceeds—*e.g.*, in œdema of the glottis—it may become expiratory also.

The urgency of the dyspnœa in this class of case is often great, but depends not only upon the degree of obstruction, but upon the rapidity with which it is developed. Thus, a foreign body suddenly gaining entrance to the larynx or trachea (a cause always to be borne in mind when inspiratory dyspnœa suddenly develops in an individual, especially a child, previously healthy) will cause a much more urgent dyspnœa in proportion to the amount of obstruction than such a cause as the gradual pressure of a tumour or aneurysm upon the larynx or trachea from without. Œdema of the glottis is another example of a cause which may develop rapidly, and speedily produce a very intense form of dyspnœa, chiefly inspiratory. A membranous laryngitis (diphtheria) often causes a form of inspiratory dyspnœa, which may be for a time slight or moderate, but is liable at any time to become urgent and to give rise to alarming symptoms.

In laryngismus stridulus we have a form of dyspnœa which develops very suddenly, and is characterized by a temporary arrest of respiration, followed by a series of deep, noisy, stridulous inspirations.

In adenoids of the naso-pharynx and retropharyngeal abscess we get a tendency to mouth-breathing and a stertorous type of respiration, which is apt to be specially marked during sleep.

The 'laryngeal crises' of tabes dorsalis are an important example of inspiratory dyspnœa. They depend upon spasm of the glottis, and are accompanied by signs of impediment to the entrance of air into the larynx, and by a cough resembling that of whooping-cough.

(2) *Dyspnœa depending on Obstruction in the Bronchi and Bronchioles.*—The obstruction may be either a swelling of the mucous membrane or the presence of secretion, or more commonly both combined. The amount of dyspnœa depends in part upon the rapidity with which the causal factors develop, but still more upon the amount of the bronchial tree involved. Involvement of the large or medium-sized bronchi causes only a moderate degree of dyspnœa, while a very severe dyspnœa may be the consequence of an extensive involvement of the fine bronchioles, as in the capillary bronchitis of children. In a simple bronchitis of the large or medium-sized tubes the organism in most cases easily compensates for the obstacle to the free entry and exit of air by a moderate increase in the frequency of the respirations. In capillary bronchitis the frequency of the respirations is usually increased to a marked degree. An actual slowing of the respiration, accompanied by an increased depth of the respiratory excursion, is not unknown in cases of bronchitis, but is rare compared with acceleration. Slowing is oftenest seen in the dry bronchitis of the medium-sized tubes.

In all cases where dyspnœa depends upon obstruction in the bronchi and bronchioles the tendency is towards prolongation of the expiration, diminished elasticity of the lungs being in most chronic cases a contributory cause.

Stridor is not usually a marked feature of these cases, but

may be present, not, as in laryngeal and tracheal obstruction, during inspiration, but during expiration.

(*a*) *The dyspnœa of emphysema.*—The dyspnœa of emphysema depends in part upon diminution in the area of breathing surface, in part upon diminished elasticity of the pulmonary tissue, in part upon co-existing bronchial catarrh and consequent obstruction in the bronchi. The breathing is usually accelerated, superficial, and the expiration is often much prolonged. In some cases, however, the breathing is slower than normal. The dyspnœa of emphysema is usually much intensified by exertion. It is aggravated by co-existing bronchitis and by dilatation of the right heart, which is a frequent complication.

(*b*) *The dyspnœa of asthma.*—The dyspnœa of asthma is characterized by its paroxysmal character, the attacks occurring at intervals, and often at night, by its severity, and by its expiratory type. Stridor is often present during expiration. The breathing may be accelerated or slower than normal. The accessory muscles of respiration are thrown into violent action. The subjective feeling of distress is often intense. Cyanosis, suffusion of the eyes, and the development of cutaneous petechiæ, frequently attend the attacks. Relief may come suddenly, but much oftener the attack gradually subsides.

(3) *Dyspnœa* depending upon diminution in the amount of breathing surface, either in consequence of inflammatory changes in the air cells, of pressure on the lungs from without, or of impediment to the free movements of the respiratory muscles:

In this class we may include the following cases: Pneumonia, capillary bronchitis, passive congestion of the lungs, œdema of the lungs, brown induration of the lungs, tuberculosis of the lungs, pleural effusion, pneumothorax, tumour of the lungs or of the mediastinum, spinal curvature, paralysis of the intercostal muscles, paralysis of the diaphragm.

Pneumonic dyspnœa falls partly into this class, partly into the next class.

(4) *Dyspnœa depending on Toxæmia.*—It is probable that many varieties of dyspnœa depend chiefly on the action of over-heated or imperfectly depurated blood on the respiratory centre in the medulla. That this is the main cause of the very intense dyspnœa of pneumonia is probable from the well-known fact that, after the crisis, when the temperature returns to the normal, the dyspnœa usually promptly disappears, although the physical signs may make it clear that the hepatization of the affected area has undergone little or no change.

This is the most convenient place for considering in some detail the dyspnœa of pneumonia.

(*a*) *Dyspnœa of pneumonia.*—The respirations are accelerated from the outset of the attack, and vary from about 30 to 35 per minute in a mild attack, to 40, 50, 60, or more in a severe attack. The number of respirations per minute is greater in proportion to the severity of the attack in children—in whom 80 or 100 respirations per minute are not unknown—than in adults, and marked acceleration of the breathing has less prognostic importance in the former case than in the latter. The respirations are shallow and usually painful, owing to co-existing pleurisy. The dyspnœa is of the mixed type—viz., both inspiratory and expiratory. The patient speaks with effort, and his words are broken up by involuntary pauses. In a severe case the patient seems, as it were, to be breathing for dear life—to have hardly time, thought, or energy for anything else. When he puts the drinking-cup to his lips he removes it in a second or two because of the urgent necessity to breathe. We get the impression that the patient does not take deep breaths simply because he cannot do so; not as in acute pleurisy, where it is evident that the patient could take a deep breath if he were not deterred from attempting to do so by fear of pain.

The subjective feeling of distress (*das subjective Gefühl der Athemnot*) may not be pronounced at the outset of an attack of pneumonia, but is usually prominent at the height of the attack. It usually abates suddenly at the crisis.

(*b*) *Dyspnœa of acute congestion of the lungs, œdema of the lungs, and capillary bronchitis.*—These forms of dyspnœa have much in common with pneumonic dyspnœa, and are probably dependent partly on diminished breathing surface in the lungs, and partly on toxæmia. In acute pulmonary œdema the dyspnœa may be quite as urgent as in acute pneumonia. In the capillary bronchitis of children recession of the ribs in the lower lateral regions is often present, and affords us a valuable index of the severity of the attack and of the degree of danger. With much recession the outlook is always grave.

(5) *Dyspnœa depending on Painful Muscular Movements during Inspiration.*—This form of dyspnœa occurs in fracture of the ribs, periostitis of the ribs, rheumatism of the inspiratory muscles, the presence of trichinæ in the intercostal muscles, pleurisy in the stage before effusion, peritonitis.

This form of dyspnœa is specially marked in diaphragmatic pleurisy.

The dyspnœa of pleurisy.—The dyspnœa of pleurisy is probably compounded of several factors. It is due, in some measure, simply to the pain produced by the rubbing together of inflamed pleural surfaces, but also in part to diminished expansibility of the lung on the affected side, and in part to the pyrexia present in the case. It is not so urgent as the dyspnœa of pneumonia, and the respirations rarely exceed 30 or 35 per minute. The respirations are shallow, the inspiratory expansion being checked by pain. The patient seems to us as if he could take a deep breath if he liked, but that he is afraid to do so owing to the suffering thus produced.

When effusion has taken place the dyspnœa usually abates,

especially if the patient is kept at rest. Even with one side full of fluid the patient may exhibit hardly any dyspnœa, unless he makes some effort. The dyspnœa in pleural effusion, according to the well-known law previously adverted to, is more marked in rapidly accumulating effusions, and *vice versâ*. In chronic and slowly accumulating effusions it is not uncommon for the patient to seek advice simply for debility or dyspepsia, and to make no allusion to his breathing until the matter is inquired into.

For the purposes of comparison, a brief description is appended of those forms of dyspnœa which are independent of pulmonary disease.

The Dyspnœa of Heart Disease.—The breathing is affected in practically every case of heart disease when compensation has begun to fail. The breathing is panting in character; the dyspnœa is both inspiratory and expiratory; it is much affected by posture and movement; the breathing is accelerated; in some cases it is deeper than normal.

In every case of heart disease attended by dyspnœa it is well to examine carefully for pulmonary complications, especially passive congestion of the lungs, œdema of the lungs, pulmonary infarct, and hydrothorax. Cardiac dyspnœa may be much aggravated by any of these conditions, but its essential cause is imperfectly oxygenated blood acting upon the lungs and upon the respiratory centre in the medulla.

The Dyspnœa of Uræmia.—This form of dyspnœa is usually paroxysmal in character and expiratory in type. It is well to inquire for bronchial catarrh, œdema of the lungs, failing compensation of the heart.

The Dyspnœa of Anæmia.—The dyspnœa of anæmia has much in common with the dyspnœa of failing compensation of the heart, with which it is often confused. It is a prominent feature of well-marked chlorosis, and still more of pernicious anæmia. The breathing is sometimes both

hurried and deeper than normal. The dyspnœa is much affected by movement and exertion.

The Dyspnœa of Diabetic Coma.—In this condition the breathing becomes deep and noisy; its rate may be normal or decidedly increased; there is a sense of *besoin de respirer* or *air-hunger*. The patient may be deadly pale or cyanotic.

Stridor.—This symptom has already incidentally engaged our attention. It is a symptom of great importance. It may be either inspiratory, which is the usual form; or expiratory, which is much less frequent; or both inspiratory and expiratory.

Inspiratory stridor depends upon some obstruction to the entrance of air to the upper air passages, or to pressure upon the larynx, trachea, or recurrent laryngeal nerves—*e.g.*, by an enlarged gland, a tumour, or an aneurysm.

Expiratory stridor depends upon swelling of the bronchial mucous membrane or the presence of secretion there. It occurs in bronchitis, emphysema, and asthma.

Loss of Voice may be due to inflammation, swelling, ulceration, or necrosis of the vocal cords; the presence of a false membrane in the larynx; or to feebleness of the expiratory effort, paralysis of the adductors of the larynx, or to hysteria.

It is not a symptom of much diagnostic importance in connection with pulmonary disease, except in the case of the laryngeal complications of pulmonary tuberculosis.

LECTURE VI

THE STUDY OF PULMONARY SYMPTOMS (B)

SUMMARY:

B. The circulatory symptoms of pulmonary disease:

Cyanosis.
Malar flush.
Venous stigmata.
Suffusion of the eyes.
Epistaxis.
Abnormalities of the pulse.
Œdema.

C. Nervous symptoms of pulmonary disease:

Pain in the chest.
Headache.
Restlessness.
Insomnia.
Delirium.
Convulsions.
Somnolence.
Coma.
Tetany.
Subsultus tendinum.
Hiccough.
Acute mania.
Hemiplegia.
Aphasia.
Photophobia.
Cheyne-Stokes respiration.

D. Cutaneous symptoms of pulmonary disease.

Herpes.
Jaundice.
Sudamina.
Erythema.
Acute acne.
Pemphigus.
Purpura.
Urticaria.

E. Digestive and nutritional symptoms of pulmonary disease:

Nausea.
Vomiting.
Diarrhœa.
Constipation.
Emaciation.
Incurvation of nails.
Clubbing of fingers.

F. General symptoms of pulmonary disease:

Pyrexia.
Changes in the blood.

B. The Circulatory Symptoms of Pulmonary Disease.

The relations of the respiratory and the circulatory systems are close. Pulmonary disease may affect the circulation in the following ways:

1. By hindering or restricting the blood circuit in the lungs—*e.g.*, in pleural effusion, pneumonia, tuberculosis, emphysema, œdema of the lungs, pulmonary infarction, tumour of the lungs, and other conditions.

2. By altering the conditions of pressure in the thorax, and so disturbing the normal influence of respiration upon the circulation, *e.g.*, excessive expiratory efforts, as in emphysema and asthma, increase the pressure in the thorax and hinder the entrance of blood into the right auricle. Excessive muscular respiratory efforts may cause the pulse to become very weak or even to fail altogether during inspiration. In certain cases violent respiratory efforts may cause emboli to be conveyed against the blood-stream.

3. By causing the passage of a portion of the blood in the bronchial veins directly into the pulmonary veins without passing through the lungs—*e.g.*, in emphysema.

4. By causing congestion of the coronary veins, and, in consequence, impaired nutrition and fatty change in the heart muscle.

Cyanosis.—Cyanosis is common in pulmonary disease, and is a symptom of great significance and often of much prognostic import. It shows that the essential function of the lungs—viz., the arterialization of the blood—is being seriously interfered with.

Cyanosis is common in all cases where an obstruction to the entrance of air into the upper air passages is present—viz., croup, œdema of the glottis, foreign body in the larynx or trachea, stenosis of the larynx or trachea, pressure on the larynx or trachea from without, tumours of the larynx or trachea. It is also common in capillary bronchitis,

emphysema, asthma, broncho - pneumonia, pneumonia, œdema of the lungs, paralysis of the muscles of respiration.

It is rare in pleural effusion, even when one side of the chest is full of fluid, and it is not often a prominent feature of pulmonary tuberculosis. Its absence in pleural effusion shows us what a large reserve of functional capacity the lungs possess—so long, at least, as the patient is at rest; while the absence of cyanosis in pulmonary tuberculosis may be attributed with probability to the progressive wasting in that malady, and the consequently lessened demands of the organism upon the circulation.

Cyanosis is often much aggravated by coughing and by the stooping posture.

Malar Flush.—A flush over the malar bones is common in pneumonia during the acute stage and in pulmonary tuberculosis, where active tuberculization is going on. It is occasionally present in empyema.

Venous Stigmata.—Venous stigmata may be present, especially in the cheeks, in any case of pulmonary disease in which venous congestion is a marked feature. They are often the result of violent coughing—*e.g.*, in capillary bronchitis, broncho-pneumonia, asthma, whooping-cough.

Suffusion of the Eyes occurs in much the same cases as those in which venous stigmata are found. It is frequently marked in all cases where violent and paroxysmal coughing occurs—*e.g.*, capillary bronchitis, broncho-pneumonia, asthma, whooping-cough.

Epistaxis.—Epistaxis is rare in pulmonary disease. It occurs occasionally in pneumonia, pulmonary tuberculosis, and whooping-cough.

Abnormalities of the Pulse.—The varieties of pulse in pulmonary disease are many, and the subject is a large one. Many of the phenomena will be more appropriately considered in subsequent lectures dealing with the various

pulmonary diseases in detail. Only a few of the more salient points will be dealt with in this place.

Pulse in Acute Bronchitis.—In mild cases in adults the pulse frequency is not much altered in acute bronchitis, and its volume and tension do not depart much from the normal. In severe cases the pulse is much accelerated, and may become small, weak, and even dicrotous. These changes are usually much more marked in children than in adults.

Pulse in Pneumonia.—Acceleration of the pulse occurs in most cases in proportion to the severity of the attack, and the degree of acceleration has much prognostic import. The usual rate of the pulse in an ordinary case in adults is from 90 to 120 beats per minute, while in the young child the pulse may rise to 160, 180, or more. In this latter case an extremely frequent pulse does not preclude a favourable issue. The pulse is abnormally frequent in patients with organic heart disease and in alcoholic subjects, and, according to many observers, in apical pneumonia. Slowing of the pulse is not unknown, even in cases which end fatally. Slowing of the pulse, sometimes to a marked degree, is often a feature of the post-critical stage of pneumonia. It has no unfavourable significance. At the outset the quality of the pulse is full and bounding, but later tends to become small and weak, especially in cases where an increase of cardiac dulness to the right and the presence of epigastric pulsation show that distension of the right ventricle has taken place. The most typical feature of the pulse of pneumonia is its altered ratio to the respirations—viz., 2 to 1, or 1·5 to 1, instead of the normal 4·5 to 1.

Intermittence of the pulse sometimes occurs, especially in old people, and is in my experience of grave import. Irregularity of the pulse in young patients does not seem to possess grave prognostic significance.

Pulse in Broncho-Pneumonia.—The pulse in this disease is in many cases very frequent, weak, and irregular. When

broncho-pneumonia supervenes upon pre-existing disease—*e.g.*, bronchitis, whooping-cough, measles—a marked increase in the frequency of the pulse, with lowered tension and altered rhythm, is usually a pronounced feature.

Pulse in Pulmonary Tuberculosis.—Increased frequency and diminished tension of the pulse are often among the earliest signs of incipient phthisis, and should be carefully noted. As the disease progresses, these characters become more and more marked, in proportion to the degree of asthenia present. The frequency of the pulse bears no fixed relation to the temperature. Cases of abnormally slow pulse, even when the disease is progressing towards a fatal issue, have been noted, but are rare. The frequency of the pulse has considerable prognostic value, and also throws some light on the question of the suitability of the high altitudes.*

A capillary pulse is sometimes observed in phthisis. Pulsation may also be sometimes noted in the veins on the back of the hand.†

Pulse in Asthma.—During an asthmatic paroxysm the pulse is small and weak, usually increased in frequency, but sometimes the reverse, often irregular.

Pulse in Pleurisy.—The pulse in pleurisy is, upon the whole, not very characteristic. There is usually a moderately increased frequency (90 to 100). The quality may be hard and bounding or soft and weak. Alterations in rhythm may occur, but are not characteristic. If pericarditis supervenes upon pleurisy, the pulse tends to become very frequent, weak, and irregular.

Pulse in Emphysema.—In slight cases the pulse may show no characteristic signs, but in advanced cases, when dilatation and hypertrophy of the right side of the heart and

* See Lecture XX.

† Osler, 'The Principles and Practice of Medicine,' sixth edition, p. 329.

tricuspid reflux are common, the pulse becomes frequent, weak, and sometimes irregular.

Œdema in Pulmonary Disease.—Œdema, either general or more frequently confined to the lower extremities, may occur in any form of pulmonary disease, accompanied by feeble circulation. It is not common in acute conditions, but occurs frequently in advanced emphysema, and towards the close of pulmonary tuberculosis. Œdema of the affected side is sometimes present in empyema, and is a sign of considerable diagnostic import. It is less common in serous or hæmorrhagic effusions. It is commoner in children than in adults. The precise cause of œdema in such cases is not clear, but it is probably local. One theory is that it is due to obstructed local circulation, in consequence of intrathoracic pressure. It is not easy to understand on this theory why œdema should be the rare exception, instead of being the rule, in pleural effusion, nor why a purulent effusion should lead to a greater increase of intrathoracic pressure than a serous effusion. Another view is that it is due to extension of inflammation through the intercostal muscles.*

C. The Nervous Symptoms of Pulmonary Disease.

Nervous symptoms are frequent in pulmonary disease, but, on the whole, are not very characteristic. They depend in the main upon the height of the pyrexia, the intensity of the toxæmia, and the amount of prostration, rather than upon the local pulmonary conditions. An exception might, perhaps, be made of the case of pneumonia, as in this disease nervous symptoms—headache, delirium, convulsions, tremor, retraction of the head, acute mania—are occasionally prominent, and may lead to grave errors in diagnosis. Nervous symptoms are usually more pronounced in children and young people than in adults or the aged. They are often very

* Wintrich.

prominent in the intemperate. Pain in the chest stands on a special footing, as it is a localizing sign, and has no necessary relation to the severity of the case.

Pain in the Chest.—Pain in the chest in pulmonary disease takes the forms of 'stitch in the side,' usually due to pleurisy; substernal pain, common in bronchitis; and soreness along the attachments of the diaphragm, common in cases characterized by severe coughing. As a rule, there is not much difficulty in identifying these forms of pain, or in attributing them to their true cause. Pleuritic pain is often erroneously attributed to pleurodynia. A distinction between these conditions may generally be drawn by attention to the seat and persistency of the pain, its relation to the breathing, the presence or absence of an audible 'rub,' and the state of the temperature. While precise rules on all these points can hardly be formulated, we may say that the pain in pleurodynia is less definitely localized than in pleurisy, more apt to involve several areas, more likely to shift about from hour to hour and from day to day, less closely associated with the breathing, usually unaccompanied by any rise of temperature. Tenderness on pressure may be present either in pleurodynia or in pleurisy. As previously pointed out, it is usually more marked in purulent than in serous effusions. Pain in the chest may also be due to periostitis of a rib, the pressure of an intrathoracic tumour or an aneurysm, or to trichinosis. Pain in the chest, persistent and gradually ingravescent, is a marked feature in most cases of malignant disease of the lungs.

Headache.—Headache may occur in any form of pulmonary disease, but does not usually possess much diagnostic value. The headache of early pneumonia is often intense and persistent, and sometimes leads to an erroneous diagnosis of meningitis. Persistent headache coming on in empyema may suggest the possibility of cerebral abscess; coming on in a case of chronic phthisis it may suggest the commence-

ment of tubercular meningitis. Headache in pulmonary disease often depends upon digestive disorder.

DELIRIUM.—Delirium may occur in any form of pulmonary disease attended by severe pyrexia. It is common in pneumonia, and has considerable prognostic import. The mortality is decidedly higher in cases attended by delirium than in those in which it is absent. It is said to be commoner in apical than in basic pneumonia. It is particularly common in the intemperate. It is comparatively common in acute tuberculosis. It also occurs in chronic phthisis, with or without tubercular meningitis.

CONVULSIONS.—Convulsions are not uncommon in the early stage of pneumonia in children. They may occur also in severe cases of capillary bronchitis and broncho-pneumonia.

COMA.—Coma is not a frequent feature of pulmonary disease. It occurs in pneumonia, acute tuberculosis, and chronic phthisis. It may be associated with meningitis—tubercular or pneumococcal. Coma is not a common terminal event in pneumonia.

ACUTE MANIA.—Acute mania occurs in some cases of pneumonia, especially in the intemperate. It may lead to serious errors of diagnosis.

CHEYNE-STOKES RESPIRATION.—This peculiar form of respiration, which has been attributed with much probability to diminished excitability of the respiratory centre in the medulla, occurs chiefly in myocardial disease, apoplexy, and renal disease. It is decidedly rare in pulmonary disease, but has been observed in acute miliary tuberculosis.

D. CUTANEOUS SYMPTOMS OF PULMONARY DISEASE.

These symptoms are not of great importance, but may sometimes give a hint for diagnosis.

HERPES.—Herpes is a common symptom in commencing pneumonia. It usually occurs about the lips and angles

of the mouth, but it has also been observed on the arms and on the trunk. It is an early symptom, but does not usually appear before the third or fourth day, and may develop at a later stage. Estimates of the frequency of herpes in pneumonia by different observers vary from 10 to 50 per cent. There is a difference of opinion whether this complication is commoner in the child or the adult. There is an old tradition in medicine that the occurrence of herpes in pneumonia has a favourable prognostic significance.

JAUNDICE.—Jaundice, varying from a slight icteric tinge to a pronounced yellowness, is a frequent symptom in pneumonia. It is commoner in pneumonia of the right than of the left lung. It is said to be more frequent in summer than in winter.

Jaundice has been observed, but very rarely, in pleurisy.

It is a very rare complication of other pulmonary diseases.

SUDAMINA.—Sudamina may occur in any pulmonary disease of which profuse sweating is a feature—*e.g.*, acute miliary tuberculosis, chronic pulmonary phthisis, and the post-critical stage of pneumonia.

ERYTHEMA, ACUTE ACNE, PURPURA, and URTICARIA have been described among the rare complications of pneumonia.

E. DIGESTIVE AND NUTRITIONAL SYMPTOMS OF PULMONARY DISEASE.

VOMITING.—Vomiting is common in pneumonia, occurring in nearly one-half of all cases. It is an early symptom, and in most cases ceases after the second day, but may persist longer. It is much commoner in children than in adults.

Vomiting is not uncommon in broncho-pneumonia, but it is much less frequent in this condition than in croupous pneumonia.

Vomiting in consequence of violent coughing is not uncommon in bronchitis.

Vomiting is common in chronic phthisis, and may be

either an early or a late symptom, more often the latter. It seems to depend either upon the presence of catarrhal gastritis or upon the irritation of coughing. When the story is that the patient often coughs until he vomits, it is a good clinical rule to think of two conditions—viz., whooping-cough and phthisis.

Vomiting is an almost constant feature of whooping-cough, and usually occurs at the end of a paroxysm of coughing.

Vomiting is sometimes a symptom of the invasion stage of pleurisy in children.

DIARRHŒA.—Diarrhœa is a frequent feature of many forms of pulmonary disease.

Diarrhœa is an occasional feature of bronchitis, but constipation is more common. Diarrhœa is common in broncho-pneumonia, either as a primary symptom or in consequence of the injudicious use of aperients. It is an occasional symptom of the invasion stage of pneumonia, or may arise at a later stage. It is not common in acute pleurisy, but is somewhat frequent in empyema. It is common in bronchiectasis.

Diarrhœa is a very common feature of chronic phthisis, and may depend upon one of three distinct causes—viz.:

(*a*) A catarrhal condition of the alimentary canal. In this case there will probably be disordered appetite, foul breath, discomfort in the epigastrium after meals, and a thickly-coated tongue, with in some cases prominent papillæ.

(*b*) Ulceration of the bowels. In this case the motions will probably be painful, and occasionally contain blood. There will be tenderness on deep pressure in the abdomen. The tongue is often raw-looking, clean, and unnaturally red.

(*c*) Amyloid disease of the intestines. In this case the motions will probably be very numerous, painless, and watery.

EMACIATION.—Emaciation is a prominent feature of many pulmonary diseases, both acute and chronic. It is a marked

symptom of acute miliary tuberculosis, of broncho-pneumonia, of some forms of chronic pleurisy, and of chronic phthisis. The emaciation of phthisis has a close relation to the pyrexia of the disease.

CLUBBING OF THE FINGERS (TROMMELSCHLÄGELFINGER).—This condition depends upon trophic changes in the skin and bones of the end phalanx of the fingers. It is found in many cases of bronchiectasis and of empyema, less frequently in phthisis.

F. GENERAL SYMPTOMS OF PULMONARY DISEASE.

PYREXIA.—Pyrexia is one of the most frequent symptoms of pulmonary disease. The subject, which is one of great importance, will be most conveniently dealt with in connection with the various diseases of which it forms part of the symptom-complex. Only points of a general character, or of importance for the differential diagnosis, will be adverted to here.

Pyrexia may depend upon the action of micro-organisms, and this is no doubt its most usual explanation. It is not always a safe index of the severity of the morbid process in any individual case, still less is its intensity a safe guide to prognosis. The reaction of the patient may be an even more important factor than the nature of the toxin at work, in determining the type and severity of the pyrexia in any instance. Many diseases—*e.g.*, pneumonia and some forms of pulmonary tuberculosis—have types of pyrexia which are characteristic, though a diagnosis based solely upon temperature is open to many sources of fallacy. In estimating the significance of temperature, age is a matter of much importance, and, to a less degree, idiosyncrasy. On the one hand, the temperature of the child is less stable than that of the adult, and in consequence departures from the normal possess less significance. On the other hand, with advancing years the element of defective reaction comes into play, and a low

temperature—as in the pneumonia of the aged—may simply be an index of feeble vitality, and hence highly unfavourable. Idiosyncrasy cannot be safely left out of account. There are many individuals in whom a smart pyrexia may be the result of trivial causes, and, therefore, devoid of the significance which under ordinary circumstances would attach to it. The estimation of the diagnostic and prognostic significance of pyrexia calls for much circumspection. It is easy either to underrate or overrate its importance. It is a safe rule to interpret pyrexia in the light of the other symptoms.

The impression, which prevails widely, that tuberculosis of the lungs is necessarily a febrile condition is without foundation. Pulmonary tubercle can exist for long periods, and make serious progress before the thermometer gives any warning—a fact by no means sufficiently recognised. That the temperature may be normal in quiescent or arrested cases is, of course, a familiar fact. It seems probable that the pyrexia of phthisis is due to a large extent to secondary infections by the micro-organisms of suppuration.

In studying the pyrexia of pulmonary disease, we shall do well to attend to the following points :

(*a*) The general course, severity, and daily oscillations of temperature.

(*b*) The conditions, such as exercise or nervous excitement, which tend to aggravate the pyrexia.

(*c*) The conditions, such as rest or cold applications, which tend to moderate the pyrexia.

(*d*) How far a sharp pyrexia is due to the severity of the attack or to vigorous reaction on the part of the patient.

(*e*) How far a low temperature is due to slight involvement of the lungs or to deficient reaction—*e.g.*, in the aged or intemperate.

(*f*) How far a severe initial pyrexia—*e.g.*, in pneumonia or pleurisy—throws light upon the probable type and duration of the subsequent illness.

(*g*) The relation of pyrexia to the condition of the circulatory, secretory, and nervous systems.

(*h*) The bearing of pyrexia on prognosis and treatment.

Most of these questions will engage our future attention in the course of these lectures. A few points may, however, be appropriately noted here:

1. The relation of pyrexia to other symptoms. One of the most important guides to the correct appraisement of the significance of pyrexia is to estimate it not so much *per se* as in relation to its effects upon the circulatory, secretory, and nervous systems. A slavish subservience to the mere readings of the thermometer without looking further afield is especially to be deprecated. There is a most significant difference between a pyrexia of a certain intensity which is accompanied by an unduly frequent and low-tensioned pulse, inability to absorb nourishment, restlessness, and insomnia, and a pyrexia of equal or even greater intensity where the pulse remains fairly good, the digestive condition satisfactory, and the nervous system is not seriously disturbed. Such differences exist and possess the highest significance. The reason why a high temperature has less importance in the child than in the adult is to be attributed largely, but not exclusively, to the fact that the circulatory and nervous structures of the child sustain a high temperature with more impunity than those of the adult.

2. It is most important to interpret fluctuations in temperature *in the light of the general clinical condition.* It is a not uncommon error to attach an importance to a fall in temperature which does not properly belong to it. The temperature has fallen, but the patient is not really better. His pulse remains frequent and weak, his nervous and digestive states are not improved. To be guided by the temperature too exclusively in such cases is to fall into one of the most obvious of pitfalls. In all such cases we should reason: Is the fall in temperature an isolated fact which

may have several interpretations, favourable or unfavourable, or is it the index to a general improvement? *Is the patient clinically better?*

There are, of course, cases in which a fall in temperature may have an unfavourable significance—*e.g.*, after a sharp hæmoptysis in phthisis. These cases are not so likely to be misinterpreted as those previously adverted to.

3. Casual fluctuations of temperature are to be interpreted with caution, and always in the light of the general course of the pyrexia. In most forms of pyrexia fluctuations occur which possess little significance. The patient is alternately depressed or encouraged by such fluctuations, which must not be allowed to mislead the medical attendant.

4. The relation of the maxima and minima of the pyrexia may be of much significance. Thus, in phthisis one of the worst forms of pyrexia is where a high afternoon temperature is conjoined with a very low, perhaps decidedly subnormal, temperature in the early morning.

5. The effect upon the pyrexia of cold applications, antipyretic drugs, exercise, rest, excitement, etc., gives us valuable information.

In some cases pyrexia is readily, in others but little, controlled by treatment. In the former cases it is important to note whether the improvement is transitory or more or less permanent, whether it holds good only so long as active medication is continued, or continues when the treatment is suspended.

The relation of the pyrexia of phthisis to rest and exercise is often close, and gives us one of the most important clues to treatment.

A temperature easily raised by slight nervous excitement, such as the visits of friends, is characteristic of the *eretische constitution*, and is one of the unfavourable characteristics of certain types of phthisis. It may depend on vaso-motor instability.

CHANGES IN THE BLOOD.—Our knowledge of the changes which take place in the blood in pulmonary, as well as in other, diseases has been much augmented in recent years, and the subject has assumed great practical importance. It can be dealt with only very briefly here.

Acute Bronchitis.—In an attack of bronchitis of moderate severity the changes in the blood are unimportant. In severe cases the white cells may be 15,000 per c.mm. or more. In capillary bronchitis a marked leucocytosis may be present, the white cells sometimes numbering from 20,000 to 40,000.*

Emphysema.—In slight cases, where there is no cyanosis, there may simply be a slight anæmia, due to co-existing conditions, such as cirrhosis of the liver or nephritis. When cyanosis is a marked feature, an increase in the number of red cells and an increase in the specific gravity of the blood has been observed.

Asthma.—An increase of the eosinophiie leucocytes, amounting to as much as 20 per cent. of the total leucocytes, has been observed during the asthmatic paroxysms by Leyden, V. Noorden, Sahli, and others. This increase appears to be confined to the time of the paroxysms, and not to be a constant feature of the blood of asthmatics. Whether it can be relied upon for assistance in diagnosis is still doubtful.

Pneumonia.—The changes in the blood in pneumonia are very important, and have been carefully studied by many observers. The following are the principal points:

The amount of fibrin is increased, except in certain very severe cases where leucocytosis is absent.

The total volume of the blood is somewhat reduced, owing to the pyrexia, the cyanosis, and the loss of cells and plasma in the exudate.

There is a diminution in the number of red cells, which some

* Ewing, Cabot.

observers believe is proportionate to the height of the temperature.* The diminution is usually slight, and there are few morphological changes in the red cells.

There is a well-marked leucocytosis in the great majority of cases of pneumonia. The number of white cells averages from 15,000 to 40,000 per c.mm. The increase is more marked in children than in adults. The degree of leucocytosis seems to vary directly with the amount of the exudate. Leucocytosis begins early in the case, attains its maximum just before the crisis, and diminishes a few hours before or just after the crisis. A secondary rise in the number of leucocytes points to the presence of some complication.

The pneumonic leucocytosis is characterized by the large percentage of the polynuclear neutrophile cells (80 to 95 per cent.), a marked reduction in the number of lymphocytes (2 to 4 per cent.), and the temporary disappearance in many cases of the eosinophile cells. Myelocytes are usually present.

Diagnostic and prognostic significance of the pneumonic leucocytosis.—The absence of leucocytosis is evidence against the existence of pneumonia; but, on the other hand, it is sometimes absent both in very mild and in the most severe cases. The presence of leucocytosis is evidence against the probability of certain diseases which might be confounded with pneumonia—viz., typhoid fever, typhus fever, malaria, acute pneumonic phthisis, influenza. In these conditions leucocytosis is unusual.† The grade of the pneumonic leucocytosis has no constant relation to the severity of the attack or the gravity of the prognosis, but in general it seems probable that a moderate leucocytosis is favourable, that a high grade of leucocytosis indicates a sharp attack or the presence of complications, and that a diminution of leucocytes (leucopenia) occurring in severe cases is unfavourable, though

* Böckmann.

† Ewing, 'Clinical Pathology of the Blood,' p. 254.

apparently less grave than was formerly thought. A well-marked leucocytosis with only relative increase of the polynuclear cells is believed to indicate a severe infection and a feeble reaction on the part of the organism.* A renewal of leucocytosis after its disappearance points to a fresh invasion of the lungs or to the presence of some complication. The reappearance of eosinophile cells indicates that the lesion has passed its acme.†

Tuberculosis of the Lungs.—The changes of the blood in phthisis are less marked than might have been expected from the degree of emaciation and the amount of the average pyrexia. Even in pale, emaciated patients with chronic phthisis and cavities the red cells may number 5,000,000 or more, and the white cells 5,000 to 10,000. A few cases are on record of severe anæmia consequent upon phthisis, but there are multitudes of cases in which 'the almost normal quantity of red cells and hæmoglobin stands in no ratio to the visible disturbance of the economy of the blood and to the extreme cachexia of the blood.'‡ The general rule would seem to be that a loss of more than 20 per cent. of red cells or hæmoglobin is unusual except in the presence of such complications as hæmorrhage, suppuration, or amyloid degeneration.§

The following are the views of Grawitz on this subject :‖

First stage of phthisis — stage of apical catarrh.—There is usually some diminution in the number of red cells; the white cells are variable; the solid residue of blood and serum and the specific gravity of the blood are diminished. Normal conditions may, however, be found.

Chronic phthisis with cavities, but with slight or no fever.—

* Sahli, *op. cit.*, p. 644.

† Ewing, 'Clinical Pathology of the Blood,' p. 254.

‡ Cornet, 'Nothnagel's Encyclopædia of Practical Medicine,' English edition, article 'Tuberculosis,' p. 381.

§ V. Noorden, quoted by Ewing.

‖ Quoted by Cornet, *loc. cit.*

The blood is often normal, as there may be a slight increase in the number of leucocytes.

Chronic phthisis with markedly remittent or intermittent fever.—The red cells and the hæmoglobin are decidedly diminished; the leucocytes are often increased; the solid residue of the blood and serum and the specific gravity of the blood are lowered.

The most singular fact about the blood conditions in phthisis is that the early oligocythemia is replaced by normal conditions as the disease progresses, and only returns at the end of the process. This has been attributed variously to concentration of the blood through loss of water, especially by profuse sweating and diarrhœa; to the absorption of certain extractives from tubercular areas; or to a general drying out of the tissues.*

The injection of tuberculin causes a temporary leucocytosis and an increase of eosinophiles.

An increase of leucocytes in phthisis points in most cases to the presence of suppurating cavities or areas of pneumonic foci, or is the result of hæmoptysis.

Purulent tuberculous inflammations of serous membranes cause usually a slight leucocytosis.

The state of the blood in tuberculous meningitis is apparently variable, some observers reporting a decided leucocytosis, others failing to find any important changes.†

A moderate or marked leucocytosis usually accompanies the involvement of bones and joints.

In acute miliary tuberculosis the number of leucocytes is usually normal.‡

Pleurisy.—In the acute stage some degree of leucocytosis is usually present, the white cells varying from 7,000 or 8,000 per c.mm. to about twice that number. The degree of leucocytosis has a relation to the height of the tempera-

* Cornet, *op. cit.*, p. 382. † Ewing, *op. cit.*, p. 302.

‡ Sahli, *op. cit.*, p. 646.

ture. Leucocytosis is most active during the exudative stage, and thereafter gradually subsides. In the majority of cases of pleural effusion when they come under treatment, the number of leucocytes has returned to the normal.

Leucocytosis is usually much more marked in cases of purulent effusion, but there are numerous exceptions to this rule, many cases of empyemata exhibiting no increase in the number of white cells.*

* Ewing, *op. cit.*, pp. 287, 288.

LECTURE VII

THE DIAGNOSTIC AND PROGNOSTIC INDICATIONS OF THE SPUTUM

SUMMARY:

Examination of the sputum.

A. Naked-eye characters of the sputum:

1. Amount.
2. Consistency.
3. Colour.
4. Presence of air.
5. Formation of layers.
6. Fibrinous casts, Curschmann's spirals, echinococcus membranes, concretions, foreign bodies.

B. Microscopical examination of the sputum:

Red blood corpuscles.
White blood corpuscles.
Epithelium.
Elastic fibres.
Charcot-Leyden crystals.
Connective tissue.
Parasites.

C. Chemical examination of the sputum.

D. The sputum in pulmonary disease.

The present lecture will be devoted to the Diagnostic and Prognostic Indications of the Sputum. Only points of definite clinical importance will engage our attention. For fuller pathological and chemical details you are referred to the special manuals.

THE SPUTUM consists of secretion or exudation from the respiratory mucous membrane from the nose and pharynx to the pulmonary alveoli; of pus or blood which may have found its way into the respiratory tract from adjacent parts—*e.g.*, the pleural cavity; or of foreign bodies derived from without. Portions of food from the mouth or regurgitated from the stomach are often intermingled with the sputum.

A. THE NAKED-EYE CHARACTERS OF THE SPUTUM.

QUANTITY.—The quantity of the sputum varies within the widest limits, and these variations often possess diagnostic importance. In some pulmonary diseases—*e.g.*, incipient phthisis, 'dry' bronchitis, pleurisy, pneumonia—the sputum may be either wanting or of the scantiest description. Young children usually swallow their sputum. In cases of advanced disease or extreme prostration the sputum is frequently wanting, because the patient no longer possesses sufficient expulsive power to eject it. The failure to expectorate in presence of physical signs showing the presence of abundant secretion in the bronchi or air cells is often of grave prognostic significance. On the other hand, a large quantity of sputum is frequent in some forms of chronic bronchitis (bronchorrhœa), bronchiectasis, advanced pulmonary phthisis, œdema of the lungs, rupture of empyemata or abscesses into the air passages. In cases of empyema discharging into the lung, as much as 800 to 1,000 cubic centimetres may be expectorated in twenty-four hours. We should distinguish cases where there is a persistent free expectoration of sputum from those where large quantities are discharged at considerable intervals of time. Bronchorrhœa is the type of the former class; bronchiectasis of the latter.

CONSISTENCY OF THE SPUTUM.—The sputum may be either extremely sticky and tenacious or of a watery consistence. As a rule, the smaller the amount of the sputum,

the more tenacious is its consistency, and *vice versâ;* but this rule has some important exceptions. The consistency of the sputum depends in part upon the amount of mucin which it contains, but also upon nuclein and nuclein derivatives. Extremely tenacious sputum is eminently characteristic of croupous pneumonia, in which condition we may often invert the spitting-cup without losing any of its contents. The viscosity of the pneumonic sputum is due to the presence of nuclein. Tenacious sputum is also common in the early stage of acute bronchitis, and in some cases of bronchial asthma, especially after a paroxysm. On the other hand, liquid and watery sputum is found in œdema of the lungs, in which affection the sputum has the general characteristics of blood serum, and is covered with a frothy surface layer. Sputum composed almost entirely of pus—*e.g.*, in acute pulmonary gangrene, pulmonary abscess, putrid bronchitis, and after perforation of an empyema into the lungs—is usually of a liquid consistence. The consistency of the sputum of chronic bronchitis and chronic phthisis presents many variations.

The Specific Gravity of the Sputum.—This varies within the widest limits and is not a point of any clinical importance.

The Reaction of the Sputum.—When freshly voided, the sputum is in the great majority of cases of an alkaline reaction. Through processes of decomposition it may in certain cases come to have an acid reaction.

The Colour of the Sputum.—The colour of the sputum depends partly on its microscopical, partly on its chemical, constituents, and may present the most various characters. In some cases the sputum is almost colourless, as in pure mucoid sputum and in some cases of pulmonary œdema. In proportion as pus corpuscles are added to mucus, the sputum tends to acquire a yellow or greenish tint. A red colour of the sputum usually depends upon the presence either of

unaltered blood—as in the various forms of hæmoptysis*—or of certain derivatives of the red corpuscles, as in the 'rusty' sputum of pneumonia.

The pneumonic sputum is not always 'rusty.' It may be yellowish or greenish, or prune-juice coloured, or its colour may be quite uncharacteristic. The green or yellow tint is most marked when pneumonia is accompanied by jaundice, but is not confined to such cases. It depends upon the presence of derivatives of the blood, which are closely allied to, or even identical with, the colouring matter of the bile. Green sputa may also be present in cases where bile pigment has become mixed with the sputa—*e.g.*, in cases of perforation of liver abscess into the lung. In cases of amœbic liver abscess with perforation into the lung, the sputa may present a characteristic colour resembling that of anchovy sauce.† Hæmorrhagic sputum may present many variations—*e.g.*, the bright-red tint of fresh arterial bleeding; the dark red or bluish-red colour of venous hæmorrhage; the dark or almost black clots sometimes seen in mitral disease; and the ochre-coloured or chocolate-brown colour seen in destructive processes in the lungs, such as abscess and gangrene. This ochre-coloured or chocolate-brown sputum is due to the presence of hæmatoidin or bilirubin. In some forms of pulmonary tumour a green-coloured sputum has been observed, of which the explanation is not known.

The various forms of pneumoconiosis sometimes present characteristically coloured sputum. Thus, the inhalation of particles of carbon or soot gives the sputum a grayish, or even black, tint; the inhalation of particles of flour or meal gives it a whitish tint; the inhalation of particles of oxide of iron gives it a reddish or brownish tint; while blue sputum is observed amongst workers in ultramarine.

Rust-coloured, punctate, or striped sputum is said to be

* See Lecture XXI.

† Simon, 'Clinical Diagnosis,' p. 247.

diagnostic of brown induration of the lungs. The sputum may, of course, be variously coloured by food, drink, or medicine—*e.g.*, fruit, milk, eggs, wine, chocolate, and so forth.

It is important to distinguish the colour of the sputum when voided from colours which may subsequently develop in the spitting-cup. These latter appear to be due in many cases to the action of chromogenic bacteria, and hence bear no necessary relation to the state of the lungs.*

The Odour of the Sputum.—Fresh sputum is almost odourless in most cases, but may acquire an unpleasant odour subsequent to its expulsion, owing to the action of putrefactive bacteria. In some cases, however, the sputum, when voided, has a powerful odour, which may be horribly fœtid, and render even a large hospital ward almost intolerable to other patients. Intensely fœtid sputum may occur in putrid bronchitis, gangrene of the lungs, bronchiectasis, pulmonary abscess, and empyemata discharging through the lungs. Sputum of this character may also occur in certain cases of phthisis where cavities are present, but in my experience this is the rare exception rather than the frequent rule. A slightly sweetish odour of the sputum is common in many conditions, and is attributable to the action of putrefactive bacteria. An odour like that of cheese has been observed in the sputum in cases of perforating empyema.

An unpleasant odour of the sputum often depends upon uncleanliness of the mouth, the presence of decaying particles of food, carious teeth, ulcers of the tongue, tonsils, or pharynx, and other such causes. Fœtor of this type is either removed or mitigated by free washing out of the mouth with a solution of permanganate of potash, a proceeding which, naturally, has no influence upon fœtor of pulmonary origin.

The odour of the sputum may depend upon certain articles of food or medicine—viz., wine, spirits, ether, turpentine,

* Simon, 'Clinical Diagnosis,' p. 248.

paraldehyde, creosote. In most of such cases it is the odour of the breath rather than of the sputum which is affected.

PRESENCE OF AIR IN THE SPUTUM.—The amount of air present in the sputum gives us some important hints as to the place of its origin, and hence may possess much diagnostic value. As a rough rule, sputum containing air floats in water; airless sputum sinks in water. Sputum containing much air usually comes from the bronchi or air cells, not from the mouth, larynx, or from pulmonary cavities. Airless sputum, which at once sinks in water, is characteristic of pulmonary vomicæ, but is not unknown in bronchitis. A purely purulent sputum, containing very little air, is found in cases of perforation of an empyema into the lungs and of rupture of a pulmonary abscess. The hæmorrhagic sputum of phthisis has often a frothy, churned-up appearance, due to a large admixture of air; but if the hæmorrhage has been very profuse and the blood quickly evacuated, there may be very little air present. In the hæmoptysis of mitral disease the sputum usually contains little air, is dark and clotty in appearance, and often sinks in water. In hæmorrhage from the upper air passages the sputum usually contains little air, as the blood is quickly evacuated. The amount of air in sputum has some relation to the activity of the respiratory processes, and is likely to be less during sleep and rest than during activity.

DIVISION OF THE SPUTUM INTO LAYERS.—In certain diseases—*e.g.*, bronchorrhœa, bronchiectasis, putrid bronchitis, and gangrene of the lungs—the sputum tends to become separated into three layers—viz.:

An upper layer, consisting of the watery elements of the sputum, and containing much air.

A middle layer, consisting of sero-pus or mucus.

A lower layer, containing pus cells, epithelial cells, and pulmonary débris.*

* Sahli, *op. cit.*, p. 582.

The sputum may be either homogeneous or heterogeneous.

The following forms of sputa are more or less homogeneous:

Mucoid sputa.
Purulent sputa.
Serous sputa.
Sanguineous sputa.

The following forms of sputa are heterogeneous:

Muco-purulent sputa.
Muco-serous sputa.
Sero-sanguineous sputa.
Sanguino-muco-purulent sputa.*

This distinction between homogeneous sputa and heterogeneous sputa is, however, not one of much practical value, as few types of sputa are absolutely uniform in appearance or quite homogeneous in character.

The 'nummular' sputum, frequent in advanced phthisis, but not unknown in other conditions, consists of roundish coin-like discs of almost pure pus embedded in mucus. This sputum sinks in water.

Small 'cheesy' particles are frequent in the sputum of tubercular cases.

Fibrinous Casts.—Fibrinous casts occur in the sputum in plastic bronchitis, of which disease they constitute the leading feature; in diphtheria, where the membrane sometimes extends from the pharynx and larynx to the bronchi; and in some cases of croupous pneumonia. These casts vary in size, from 12 centimetres in length and a few millimetres in thickness to minute fragments. They are whitish in colour, of variable but usually tough consistence, and branched. In pneumonia these casts are few in number and small in size. When they occur in this disease they

* Simon, *op. cit.*, p. 249.

cause much coughing and dyspnœa. Under the microscope they exhibit the usual structure of fibrin. These casts frequently contain Charcot-Leyden crystals embedded in them.

CURSCHMANN'S SPIRALS.—These are thick white bodies, worm-like in shape, from 1 to 2 centimetres in length and about 1 millimetre thick, either transparent or turbid, and generally of tough consistence, which are frequently present in the sputum of asthmatic patients. 'Under the microscope they display a remarkable variety of shape. In the usual arrangement there is a central thread disposed in a more or less zigzag manner, and around it a thick meshwork of very delicate fibres commonly looped round in spirals, but occasionally retiform. The spirals are often overlaid with epithelium, and sometimes also covered with Charcot-Leyden crystals.'*

These spirals are not composed of fibrin, as was formerly thought, but of a substance akin to mucin.† They have their origin in the finest bronchioles, and are believed to be due to a 'bronchiolitis exudativa.' While highly characteristic of asthma, they are also found in some cases of croupous pneumonia and capillary bronchitis. Their frequency in asthma tends to support the view that the essential condition in that affection is involvement of the bronchi, and not a general neurosis. According to Von Jaksch, in the earlier attacks of bronchial asthma, or at the beginning of a fresh attack, spirals are to be found, but no crystals; but crystals form in such cases if the sputum be allowed to stand for twenty-four to forty-eight hours, and in later asthmatic paroxysms crystals occur in the recent sputa.‡

ECHINOCOCCUS MEMBRANES. — Echinococcus cysts are rarely expectorated in a perfect state; usually the cyst is discharged in fragments. They appear to the naked eye as

* Von Jaksch, 'Clinical Diagnosis' (translated by J. Cagney), p. 116.
† A. Schmidt. ‡ Von Jaksch, *op. cit.*, p. 116.

whitish-yellow shreds. The hooklets may be recognised under the microscope, and, of course, establish the diagnosis of hydatids.

These membranes may come from a perforating cyst of the liver, kidney, or lung.

Concretions in the Sputum.—These are of a chalk-like consistence and aspect, and are sometimes called lung-stones (*Lungen-steine*). They are amongst the rarest of pulmonary phenomena. Only a few examples have come under my observation. While it is probable that these concretions owe more than one origin, their presence should always make us inquire carefully for quiescent or obsolescent tubercle.

Foreign Bodies in the Sputum.—Foreign bodies appear occasionally in the sputum, and may be coughed up months or years after they have found their entry into the air passages. Teeth, fish-bones, cigar-holders, and even more extraordinary articles, have been known to be retained for long periods in the trachea or bronchi, and ultimately expectorated. In any case where persistent hard cough, with little or no expectoration and no physical signs to explain it, is the leading feature, the practitioner would do well to weigh the probability of the presence of a foreign body in the air passages. In such cases examination by the X rays may sometimes throw light on cases which are very puzzling.

B. Microscopic Examination of the Sputum.

Leucocytes.—These bodies are always found in the sputum in considerable numbers, usually embedded in a viscid, stringy substance. They are usually polynuclear in character. They are often granular, and contain fat-droplets in their interior, or particles of carbon dust or hæmatoidin. Eosinophile cells are common in the sputum of asthmatic patients. In cases of purulent sputum, leucocytes may compose almost its entire substance.

Red Blood Corpuscles.—A small number of red corpuscles may be found in almost all sputa, and are without significance. In persons who exhibit a catarrhal condition of the buccal and pharyngeal mucous membrane, as for example in many smokers, the sputum may be streaked with blood.

The individual cells may be either quite intact, or may exhibit all degrees of departure from the normal. In some cases—*e.g.*, when blood has accumulated for some time in the bronchial tubes—the corpuscles may entirely disappear, only the pigment remaining.

A special lecture will be devoted to the subject of hæmoptysis.*

Epithelium.—The following types of epithelium may be found in the sputum :

Pavement epithelium—from the buccal cavity, pharynx, or upper larynx.

Cylindrical epithelium — from the nose, lower larynx, trachea, or bronchi.

Alveolar epithelium—from the air cells.

The so-called 'heart-failure' cells, consisting of alveolar epithelium, which contain altered blood pigment—found in cases of chronic venous congestion of the lungs.

Epithelial cells are often found in the sputum much altered in shape and character, and their diagnostic significance is not great.

Elastic Fibres.—Elastic fibres are frequently found in the sputum, and possess much diagnostic importance, inasmuch as they at once inform us that some destructive process is going on in the lungs. They vary in length and breadth, are usually curled up at the ends, and often exhibit an alveolar arrangement.

Elastic fibres are found in the sputum in phthisis, in abscess of the lungs, and in pulmonary gangrene.

The number of elastic fibres gives us a hint as to the

* See Lecture XXI.

probable intensity of the destructive process in the lungs. It is important to assure ourselves that elastic fibres found in the sputum are not derived from the food.

Elastic fibres may be present in the sputum even of incipient phthisis, and may give decisive help in diagnosis.

In some cases of pulmonary gangrene elastic fibres are absent — it is believed, owing to the action of trypsin ferment.*

Charcot - Leyden Crystals. — These are colourless crystals, octahedral in form, insoluble in cold water, ether, alcohol, and chloroform, but dissolved readily in acetic and mineral acids, alkalies, warm water, and ammonia. They are found in tumours, in the fæces, in leukæmic blood, in the bone-marrow, and in the blood.

Leyden believed that these crystals were the exciting cause of asthma, but other observers have found them in the sputum of bronchitic patients who had not suffered from asthma.

Connective Tissue in the Sputum.—Shreds of connective tissue have been occasionally found in the sputum in abscess of the lungs, pulmonary gangrene, and in sarcoma of the lung.

Parasites in the Sputum.—*Fungi.*—Of the three classes of fungi—moulds, yeasts, and fission-fungi—only the last possesses great clinical importance. The thrush-fungus is occasionally found, generally in consequence of admixture of the sputum with saliva.

The *Aspergillus fumigatus* has also been found. Yeasts are known to occur in the sputum, but nothing is understood regarding their significance.

Sarcinæ have been found in cases of ulceration of the lungs.

Leptothrix has been discovered in connection with putrid bronchitis.

The Bacillus of Tubercle.—The discovery of this organism

* Sahli, *op. cit.*, p. 590.

in 1882 by R. Koch constitutes one of the great landmarks of modern medicine. The methods employed for its detection depend upon the principle that the tubercle bacillus stains with aniline dyes in alkaline solution, and, unlike other micro-organisms occurring in the sputum, retains the dye in after-treatment with acid and alcohol. In the examination of sputum for the presence of this bacillus, it is advisable to pick out the small, friable, cheesy masses which are so frequently present in the expectoration of phthisical patients.

We may lay down the following axioms regarding the presence of the tubercle bacillus in sputum :

(*a*) The discovery of the bacillus of tubercle in the sputum is absolute proof of the presence of tuberculosis in some portion of the respiratory tract.

(*b*) Failure to find the bacillus after repeated examination does not preclude the existence of pulmonary tuberculosis. I have seen a case where the upper lobe of one lung became extensively infiltrated, with manifest signs and symptoms, although repeated examination of the sputum failed to detect the presence of the bacillus. In acute miliary tuberculosis the bacillus is commonly absent from the sputum.* Nevertheless, *in a case otherwise open to question*, the failure to find the bacillus where the sputum is abundant may justifiably throw doubt on the probability of tuberculosis being present.

(*c*) The bacillus may be found in the sputum before any physical signs can be detected by percussion or auscultation. I have never known a case where the bacillus was found in the absence of symptoms; but from the nature of the case such a discovery, even if possible, would rarely be made.

(*d*) The bacillus is sometimes, but not usually, present in the initial hæmoptysis of incipient phthisis.

(*e*) In the presence of active tubercular disease of the

* Sahli, *op. cit.*, p. 606.

lungs bacilli are almost always present in the sputum. Exceptions to this rule are found in some cases of acute miliary tuberculosis, in certain cases of cavity, and, as already mentioned, in a small minority of cases of ordinary progressive infiltration.

(*f*) The number of bacilli present in the sputum bears some relation to the activity of the tuberculizing process, but exceptions to this rule occur.

(*g*) In quiescent and arrested cases of phthisis bacilli are usually absent from the sputum, but they may be present in cases which are pursuing a quite favourable course. Their persistence in the sputum does not in itself warrant a gloomy prognosis.

The Pneumococcus (*Diplococcus*) *of Fränkel.*—This organism is present in the sputum of the great majority of cases of croupous pneumonia, of which most authorities regard it as the true cause. This diplococcus is also found in broncho-pneumonia, pleurisy, pericarditis, meningitis, peritonitis, acute synovitis, otitis, and endocarditis. An acute general septicæmia, without local symptoms, depending on diplococcus infection, has also been described.

The Diplococcus or Pneumo-Bacillus of Friedländer.—This organism is frequently present in the sputum of pneumonia, but it is doubtful whether it has any ætiological connection with the disease.

Actinomyces in the Sputum.—The characteristic granules and thread-like formations of this organism may sometimes be recognised in the sputum, and establish the diagnosis of actinomycosis.

The Influenza Bacillus of Pfeiffer.—This is a small bacillus, which is difficult to stain; it occurs in enormous numbers, sometimes in twos.

I may also mention the *Bacillus typhosus*, *Bacillus diphtheriæ*, *Amœbæ coli*, staphylococci, and streptococci.

When sputum has remained for some time in the spitting-

cup, many kinds of bacilli from the mouth may be found in enormous numbers.

C. The Chemical Examination of the Sputum.

The chemical examination of the sputum possesses but slight diagnostic significance. Serum-albumin, mucin, and nuclein are normally present. Peptone is found in pneumonic and purulent sputa. The 'prune-juice' expectoration of pneumonia is rich in serum-albumin.

The volatile fatty acids—acetic, butyric, propionic, and caproic—have been found in the sputum, especially in gangrene of the lungs.

Fat can be obtained from all sputa; those of tubercular patients contain much fat. The sputum of pulmonary gangrene contains indol, phenol, and scatol.

Glycogen has been found in the sputum.

The sputa of gangrene of the lung and putrid bronchitis have been shown to contain a ferment resembling trypsin.

The inorganic constituents of the sputum are the following: Chlorides, phosphates, sulphates, carbonates, phosphate of iron, and silicates.

D. The Sputum in Pulmonary Disease—Diagnostic and Prognostic Indications.

In describing the characters of the sputa in pulmonary disease, it will be convenient to consider together the diagnostic and prognostic indications.

Acute Bronchitis.—The sputum is at first scanty, viscid, transparent, and containing few cellular elements, the so-called *sputum crudum* of the old writers. It is often streaked with blood. Epithelial cells are present, a few leucocytes, and sometimes a small number of red corpuscles. Later the sputum becomes more abundant, the number of leucocytes increases, and the colour becomes a yellowish-green. Elastic fibres are never present.

The characters of the sputum do not afford much guidance for prognosis. A blood-stained sputum points to a severe attack. An increase in the amount of sputum and in its cellular elements occurs both in convalescence and in the passage from the acute into the chronic stage. A sudden cessation of a previously abundant sputum, the other symptoms continuing or becoming aggravated, is of grave omen, as probably pointing to failing power of expulsion on the part of the patient. The return of sputum in this connection would be of favourable augury.

Chronic Bronchitis.—The amount and character of the sputum in chronic bronchitis show the widest variations. In a large proportion of cases the sputum is abundant, yellowish-green in colour, and contains numerous pus-corpuscles. Some red corpuscles and epithelial cells and multitudes of non-pathogenic bacteria are usually present.

Variations in the amount and character of the sputum throw considerable light upon the progress of the case in chronic bronchitis. Usually the patient feels best when the sputum is moderate in quantity, while he is apt to feel oppressed either by a cessation or a decided increase of sputum.

Bronchiectasis.—The sputum in this condition is mucopurulent in character; variable in amount, but usually abundant; often fœtid; and usually voided in large quantities at considerable intervals of time. The posture of the patient has often an influence over the ease with which expectoration is accomplished. The expectoration, when allowed to stand for some time, separates into three layers—viz., a superficial layer of brown froth, an intermediate layer of thin mucoid material, and an inferior thick granular layer. Pus cells are very abundant, crystals of the fatty acids and hæmatoidin crystals may be present. Elastic fibres are rarely present. 'Nummular' sputum is uncommon. Hæmorrhage occurs in more than half the cases.

The character of the sputum gives some guidance as regards prognosis. Abundant, evil-smelling secretion, uncontrolled by remedies, is unfavourable. Hæmorrhage and the presence of elastic fibres point to ulceration. Variations in the amount and quality of the sputum afford indications as to the progress of the case.

PUTRID BRONCHITIS. — The sputum in this condition consists of almost pure pus. It is thick and greenish-brown in colour, variable in quantity. It has a sickly, sweetish, most disagreeable odour. It contains a profusion of micro-organisms of various kinds. It does not contain elastic fibres.

The prognosis has some relation to the amount and fœtor of the expectoration.

PLASTIC BRONCHITIS.—The sputum in this condition can be distinguished from that of catarrhal bronchitis only by the detection of fibrinous moulds of the bronchioles, already described. These coagula usually contain Charcot-Leyden crystals embedded in them. They are often voided after severe paroxysms of coughing, which have been mistaken for asthma.

The danger of the attacks has relation to the number and size of the coagula, and the intensity of the dyspnœa.

BRONCHIAL ASTHMA. — The sputum in this condition consists, at the beginning of the attack, of rounded gelatinous masses, consisting of moulds in mucus of the smaller tubes. Curschmann's spirals and the Charcot-Leyden crystals are usually present, and eosinophile leucocytes are abundant. Later the sputum becomes muco-purulent in character. Streaks of blood are not uncommon, but decided hæmoptysis is very rare. In some asthmatic paroxysms sputum is absent throughout.

The asthmatic paroxysm is usually free from danger. Patients usually find themselves relieved when expectoration becomes abundant and easy.

Croupous Pneumonia.—At the beginning of the attack and during convalescence the sputum may possess ordinary catarrhal characters which are quite uncharacteristic, but during the height of the attack, and often from an early stage, the sputum frequently exhibits remarkable characters which are pathognomonic of the disease. It is reddish-brown (rust-coloured); extremely viscid; contains either unaltered red blood corpuscles or blood pigment; leucocytes are present in considerable numbers; fibrinous moulds of the finer bronchioles may be seen; alveolar epithelial cells, often loaded with granules of pigment, fat, and myeline, may be present; calcium chloride is present in large quantity. The sputum is sometimes composed of almost pure blood.

The colour of the pneumonic sputum may be yellowish or greenish, with or without co-existing jaundice, or it may be of the 'prune-juice' type. Pneumococci (Fränkel) are nearly always present, and the pneumo-bacillus of Friedländer is frequent.

The prognostic significance of the pneumonic sputum is considerable:—

The absence of sputum throughout the entire attack has been occasionally observed, and seems to possess no special prognostic import.

A typical sputum—at first mucoid, then rust-coloured and viscid, later on muco-purulent—is, upon the whole, favourable, but has little weight in prognosis.

An abundant 'prune-juice' type is unfavourable, as pointing to a widespread congestion and œdema, and probably to failing power of the heart.

A pure hæmorrhagic sputum is somewhat unfavourable, but should not be interpreted as suggesting the probability of tuberculosis.

An abundant catarrhal sputum may point to the existence of a general bronchitis, which is, in my experience, a grave complication.

Fœtor of the sputum points to the existence of pulmonary gangrene, and is highly unfavourable.

The sudden cessation of sputum previously abundant is in pneumonia, as in other pulmonary conditions, of grave significance, as pointing to failing respiratory power.

While the sputum may give us a hint to prognosis in pneumonia, it is undesirable to base our prognosis upon the expectoration in any large degree.

The prognosis hinges essentially on age, previous habits, amount of lung involved, and staying power of the heart.

Broncho-Pneumonia.—In this affection the sputum may be either entirely absent, which is the rule with young children, in whom so large a proportion of the cases occur; or catarrhal in character; or more or less allied to the sputum of croupous pneumonia. A blood-streaked sputum is relatively common. Fränkel's pneumococcus is sometimes present, as are also the *Streptococcus pyogenes*, the *Staphylococcus aureus et albus*, and other organisms.

The prognostic indications are on general lines. The absence of sputum in the infant has, of course, no significance. A moderate expectoration, catarrhal in character, and with little or no blood pigment, is favourable. An abundant liquid hæmorrhagic sputum is unfavourable, pointing as it does to widespread congestion of the lungs and failing cardiac power.

Pulmonary Œdema.—In this condition the sputum is abundant, thin, and watery, and either white and frothy or of the 'prune-juice' type. It is often frothy and tinged with blood. It consists chiefly of pure or blood-stained serum, and contains a considerable quantity of albumin. It separates into two layers—a thin, frothy layer above, and a turbid layer below, which contains numerous formed elements, chiefly white corpuscles.

The character of the sputum in pulmonary œdema varies considerably, according as the antecedent condition is

capillary bronchitis, pneumonia, or pleurisy. The *expectoration albumineuse* of French writers—*i.e.*, the sputum which sometimes ensues upon the sudden expansion of the lung after aspiration in pleural effusion—depends upon pulmonary œdema.

Von Jaksch thinks that methæmoglobin is sometimes present.*

The prognosis is always grave in pulmonary œdema, and the amount and character of the sputum may give us a hint of the extent of the œdema in any given case. The acute œdema which in rare cases follows aspiration in pleural effusion is a very serious condition, but not always fatal.

Pulmonary Gangrene.—In this condition the sputum has a penetrating and horribly fœtid odour. It is usually thin, and of a dark, dirty, brownish-green colour. It contains pus cells and fragments of necrotic lung tissue. It tends to separate into three layers—putrefactive bacteria; fat crystals; crystals of cholesterin, leucin, and tyrosin. Pigment granules and pulmonary detritus are usually present. Elastic fibres are sometimes wanting, owing probably to the solvent action of a ferment akin to the pancreatic juice, but much oftener they are present in large numbers. Cases have been described in which fœtor of the breath was absent.

The presence of the typical sputum of pulmonary gangrene involves in the great majority of cases a fatal prognosis; but something depends on the nature of the antecedent condition (pulmonary gangrene is never primary), whether croupous pneumonia, aspiration pneumonia, bronchiectasis, or embolism of the pulmonary artery.

Pulmonary Abscess.—In this condition the sputum is chiefly composed of pus which has a stale odour. It may contain catarrhal elements. In cases where acute perforation has occurred the sputum consists of pure pus. Numerous bacteria, elastic fibres, hæmatoidin, cholesterin and fat

* Von Jaksch, 'Clinical Diagnosis' (translated by J. Cagney), p. 143.

crystals, and lung pigment are usually present. The characters of the sputum may establish the diagnosis, but do not afford basis for prognosis, which must depend upon a review of the case as a whole.

PULMONARY INFARCTION.—In recent cases single coin-shaped masses of blood, of a bright red colour and mixed with froth, are expectorated. Later the sputa become brownish, and contain epithelial cells and leucocytes undergoing fatty degeneration.

The sputum does not throw much light upon the prognosis, which is often grave, but depends upon the size of the infarct, the nature of the antecedent affection, and the capacity of the lung tissue for repair.

PULMONARY TUBERCULOSIS.—The sputum in tuberculosis of the lungs presents the widest variations, but often gives us valuable hints both for diagnosis and prognosis. The sputum may be entirely wanting, as in many early, and in a considerable number of arrested and quiescent, cases. The amount of sputum depends upon either the extent of bronchial catarrh present or upon the secretory activity of cavities. In general a diminution or disappearance of sputum points to quiescence of the disease, but a temporary increase of sputum may occur from intercurrent causes and possess no serious significance.

In the early stage of pulmonary tuberculosis the sputum may possess the ordinary 'catarrhal' characters, but even at this stage it is quite usual to find the bacillus of tubercle present, often in large numbers. As the disease progresses, the sputum tends to become more and more purulent in character. Tubercular sputum in a large proportion of cases presents small friable, yellowish or dull white, cheesy particles, which frequently contain many bacilli. When cavities are present the sputum has often the characteristic 'nummular' appearance. Elastic fibres are usually present.

Tubercular sputum has often a slightly sickly odour, but

decided fœtor has been rare in my experience—a fact which is somewhat difficult of explanation. The presence of the bacillus of tubercle is, of course, the most important fact in connection with the sputum of tuberculosis.

Blood is frequently present in the sputum of tuberculosis. Leucocytes, alveolar epithelium, hæmatoidin crystals, granules, and various fungi, in addition to the specific bacillus, may be found. Concretions of a chalk-like consistency, depending probably upon classification of caseous masses, are occasionally coughed up by tuberculous patients.

The prognostic importance of the sputum in phthisis is considerable :—

The sputum may be wanting, or almost wanting, even in acute cases with high fever and well-marked physical signs ; but this is quite rare, and in general active disease is accompanied by abundant expectoration ; while, *per contra*, a diminution or disappearance of the sputum indicates in the great majority of cases a tendency to obsolescence of the disease.

The number of bacilli in the sputum has a relation to the activity of the tuberculizing process, but there would appear to be exceptions to this rule.

The presence of blood in the sputum has little prognostic import. A 'nummular' or a foul-smelling sputum usually indicates the presence of excavation, and is, *pro tanto*, unfavourable.

Variations in the number of elastic fibres present are not necessarily of real significance.

LECTURE VIII

PLEURISY

SUMMARY:

- Ætiology of pleurisy:
  - Influence of chill.
  - Question of the existence of a 'rheumatic pleurisy.'
  - Relations of pleurisy and tuberculosis.
  - Pleurisy arising by extension of inflammation from adjacent parts.
  - Pleurisy in connection with the continued fevers.
  - Pleurisy in association with general dyscrasia.
- Bacteriology of pleural effusion.
- Varieties of pleurisy.
- Symptoms and course of pleurisy.
- Physical signs and diagnosis of pleurisy.
- Differential diagnosis of pleurisy.
- Prognosis in pleurisy.
- Treatment of pleurisy:
  - Rules for the performance of tapping.
  - Treatment of chronic pleurisy.

In accordance with the plan of these lectures, our account of pleurisy will be concerned chiefly with practical questions of diagnosis and treatment. The ætiology of the disease, however, involves so many important problems that we must consider it at some length.

Ætiology of Pleurisy.

Theoretically, pleurisy may arise—

(*a*) By the micro-organisms which are capable of exciting inflammation of the pleural membrane finding direct access to it—'primary pleurisy.'

(*b*) By the extension of inflammation from adjacent parts—*e.g.*, the pulmonary parenchyma, as in pneumonia.

(*c*) By general diseases involving the pleura as part of their manifestation—*e.g.*, septicæmia, Bright's disease, scarlatina, small-pox, cancer.

(*d*) By traumatism.

The distinction between primary and secondary pleurisy is sometimes difficult, and there are many sources of fallacy. It is probable that chill may act either as a predisposing or an exciting cause. In the latter case the primary cause may be an obsolescent tubercular focus in the lungs. In a series of seventy-four cases under my own observation there was a history of chill in twenty-six.* Such a history is, of course, in some cases misleading, the patient confusing the first symptoms of disease with its presumed exciting cause. But in many of these cases the history of chill is too definite to be misinterpreted—*e.g.*, a history of a specific wetting, lying in wet grass, rapid cooling when over-heated, and so forth. We may take it that a history of chill in this sense may be obtained in about one-third of cases of pleurisy which arise in practice. Strümpell is of opinion that 'we know positively of only one form of pleurisy—viz., the rheumatic—which is primary.'† Fiedler has emphasized the close relation which he regards as pertaining between pleurisy and articular rheumatism. On the other hand, Aschoff could not find any example of this connection in 200 cases of pleurisy observed by him

* 'Encyclopædia Medica,' vol. ix., article 'Diseases of the Pleura.'

† Strümpell, 'A Text-book of Medicine,' English edition, p. 280.

at the Urban Hospital, Berlin.* In my series of seventy-four cases of pleurisy rheumatism was noted as present in six, valvular disease of the heart in four. I have for many years enquired into the association of articular symptoms and cardiac complications with pleurisy, and I have not been able to convince myself that this association is more frequent than might be accounted for by accidental coincidence. If rheumatism is one of the essential causes of pleurisy, then pleuritic patients ought to exhibit a high proportion of valvular lesions of the heart. Is this so? Such has not been my experience. It is, of course, indisputable that a certain proportion of cases of acute rheumatism present pleurisy as one of their complications, but I do not think that we can reverse this proposition.

Relation of Pleurisy and Tuberculosis.—By far the most important question in relation to the ætiology of pleurisy is its relation to tuberculosis, which constitutes one of the most vital outstanding questions of practical medicine.

It is now generally admitted that the relation of pleurisy and tuberculosis is much more intimate than was formerly believed. This view rests upon the following chain of evidence:

(*a*) Clinical Evidence.
(*b*) Bacteriological Evidence.
(*c*) Evidence of morbid anatomy.
(*d*) Experiments with tuberculin.

(*a*) The *Clinical Evidence* is as follows: A large number of observers, amongst whom may be mentioned Fiedler, Barrs, Bowditch, Lemoine, Germain Sée, Coston, Dubville, Landouzy, Kelsch, Vaillard, Netter, Osler, and Schlenker, maintain that of patients who have passed through an attack of acute pleurisy a very large proportion (variously estimated at from a third to a half) subsequently develop phthisis.

* Ebstein and Schwalbe, 'Handbuch der Praktischen Medizin,' Band i. 1, p. 381.

Germain Sée is of opinion that 'the so-called simple pleurisy from a chill is only a tuberculous pleurisy, the nature of which has been misunderstood.' Landouzy holds that 98 per cent. of cases of pleurisy are tubercular. Netter puts the proportion at 70 to 80 per cent.

(*b*) *Bacteriological Evidence.*—Serous pleural effusions are usually sterile, and the bacillus of tubercle has been found only in very rare cases. But chiefly as the outcome of inoculation experiments, the opinion has gained ground that 'sterile' effusions are significant of tuberculosis. Aschoff found that a positive result was obtained in such experiments almost as constantly with 'sterile' effusions as with those which, on clinical grounds, could be definitely affirmed to be tubercular. Eichhorst found that inoculations of the guinea-pig with pleural exudate were followed by tuberculosis in 62 per cent. of cases.

(*c*) *Evidence of Morbid Anatomy.*—In fatal cases of pleurisy tubercle is often found in the pleura, the lungs, and the bronchial and retro-bronchial glands. The statistics on this subject are somewhat conflicting, but it may be safely taken for granted that in a large proportion of fatal cases of pleurisy the presence of tubercle can be demonstrated.

(*d*) *Experiments with Tuberculin.*—Koch found that 73·2 per cent. of cases of pleurisy reacted to tuberculin.*

This chain of evidence seems strong, and has been held by many authorities to be conclusive. Perhaps when we look a little more closely into the subject, we may see reason to question its absolute finality.

That a large proportion of fatal cases of pleurisy are of tubercular origin may be freely admitted. These fatal cases, however, constitute only a small proportion (about 6 or 7 per cent.) of the total number.

Much more important questions are the following:

* Transactions of the British Congress on Tuberculosis, vol. iii., p. 95.

(*a*) What grounds are there for supposing that an attack of acute pleurisy from which recovery is, or seems to be, complete was tubercular in origin?

(*b*) If a patient has made a good recovery from an attack of acute pleurisy, is the danger of subsequent tuberculosis slight, considerable, or serious?

(*c*) If a patient has made an imperfect recovery from an attack of acute pleurisy, is the danger of subsequent phthisis extremely grave, or only moderately serious?

These questions are of great practical importance. They meet us constantly in practice; they embarrass our forecasts in many pulmonary conditions; they complicate the problems of life assurance. Their solution would be welcome. At present a good deal of doubt hangs over the whole subject, and extreme views are current which a wider experience may ultimately modify. Without attempting to dogmatize upon a subject of great difficulty, I may suggest some reasons for withholding our assent to the view that all, or nearly all, or even the large majority, of cases of acute pleurisy are of tubercular origin:

1. The clinical evidence, strong as it looks, is open to several sources of fallacy. It is evident that we must not assume that every case of phthisis which follows at variable intervals of one, two, or several years upon an attack of acute pleurisy is due to the pleurisy. This assumption would be a naked example of the *post hoc, ergo propter hoc* fallacy. It is clear that in such cases phthisis may have arisen independently. Nor is it safe to conclude that when phthisis follows acute pleurisy—perhaps at a considerable interval of time—this sequence establishes the conclusion that the pleurisy in such cases was tubercular. The facts would be quite as easily explicable on the theory that the attack of pleurisy had left the pulmonary tissue in some way more vulnerable to bacillary invasion. Pleurisy often leaves adhesions behind it; the amplitude of pulmonary expansion is thereby cur-

tailed; respiratory efficiency is impaired. Now, everything which we know of pulmonary tuberculosis inclines us to the view that it is lungs whose functional activity is imperfect which most readily become the seat of the disease. That pleurisy should predispose to tuberculosis seems a natural and easy assumption.

2. The symptoms, course, and event of ordinary cases of acute pleurisy are not readily reconciled with what we know of the usual course of tuberculosis. No doubt this statement should be made with some caution, because our knowledge of tubercular processes is not complete, and we might arbitrarily assume that certain symptoms, and a certain type of clinical course, were inconsistent with a tubercular causation, when a wider knowledge might show that there was no real inconsistency. Allowing for such a source of fallacy, it will be admitted, I think, that, if ordinary acute pleurisies are in a large majority of cases of tubercular origin, we shall be obliged to revise our conceptions of tuberculosis of serous membranes. Without unduly straining the argument, the following points of contrast between acute pleurisy and tuberculosis give us some food for reflection:

(*a*) The onset in pleurisy is either acute or subacute. The onset of tuberculosis, either of the lungs or of serous membranes, is rarely acute, occasionally subacute, most often chronic.

(*b*) The temperature in pleurisy is uncharacteristic; the daily range is fluctuating; the total febrile period is often limited to two or three weeks. The temperature in tuberculosis, whether due to the toxins secreted by the bacillus of tubercle or, as some think, mainly to mixed infections, presents within wide limits certain more or less definite characteristics—viz., a certain type of daily range, a prolonged course, a gradual and irregular defervescence, when defervescence occurs.

(*c*) In the large majority of cases of acute pleurisy recovery, in most cases apparently complete, takes place in three or four weeks. Tuberculosis of the lungs, or of the peritoneum, or the meninges of the brain, is usually unfavourable in its course; most cases end fatally, and in the most favourable type of case the duration is very prolonged. We may fairly ask: Is it inherently probable that tuberculosis of a serous membrane will come on acutely in an individual apparently previously healthy, run a fairly-defined course of three or four weeks, end usually in complete convalescence without obvious impairment of the general health, and then in one, two, or more years burst out into fresh activity, involving the lung substance?

(*d*) The results of inoculation experiments are open to the fallacy that what is true of the guinea-pig is not necessarily true of man. Koch's famous therapeutic *non sequitur* should act as a warning.

(*e*) Pleurisy shows no preference, but rather the contrary, for the apices of the lungs, which are so constantly the seat of tuberculosis.

(*f*) Koch's experiments with tuberculin in cases of pleurisy seem to add much weight to the tubercular theory of the disease, but some remarkable experiments are on record tending to show that the number of apparently healthy individuals who react to tuberculin is large. In this connection we should do well to weigh the full significance of the well-known aphorism: *Jeder Mensch hat am Ende ein Bischen Tuberkulose.*

I do not wish to be understood as questioning the close relation which subsists between pleurisy and tuberculosis, but only as demurring to the extreme view of the subject which is now current in France and Germany. Some reaction against these views is already apparent. Unverricht, of Magdeburg, believes that the extreme view, above outlined, goes too far, and that 'a large number of patients after

cure of their pleurisy never show any symptoms which would justify the assumption of tuberculosis. Also that at later autopsies in such cases no signs suspicious of tuberculosis are found.'* E. Grawitz is inclined to postulate on idiopathic pleurisy not dependent on the presence of micro-organisms.†

In concluding our survey of this subject, we must not forget to note that acute pleurisy may supervene in patients who have a quiescent tubercular lesion at one or both apices. This is probably a not uncommon event. Patients suffering from pleurisy often give a history of previous indifferent health, and especially of a 'weak chest.' In such cases, it is well to weigh the probability of antecedent phthisis.

Pleurisy arising by Extension of Inflammation from Adjacent Organs.—Pleurisy is a usual complication of the following diseases: Pneumonia, pulmonary abscess, pulmonary gangrene, pulmonary infarction, pneumothorax, malignant disease of the lungs or mediastinum, caries of the ribs, sternum or spinal vertebræ, actinomycosis, hydatids, pericarditis. It may also arise by extension of inflammation —*i.e.*, by bacterial invasion from the abdominal viscera, as in subphrenic abscess; peritonitis; cancer of the stomach, liver, or intestine; appendicitis; uterine disease.

Pleurisy is not a common complication of simple or capillary bronchitis, but is frequent in association with putrid bronchitis.

The relations of pleurisy and pneumonia are close and important. In ordinary cases of croupous pneumonia the co-existing pleurisy is confined to the membrane covering

* H. Unverricht in Ebstein und Schwalbe's 'Handbuch der Praktischen Medizin': 'Zunächst ist festzuhalten, dass eine grosse Zahl derartiger Kranker nach Abheilung der Pleuritis nie wieder Erscheinungen darbietet, welche die Annahme von Tuberkulose irgendwie rechtfertigen Könnten. Auch bei einer später vorgenommenen Leichenshau finden sich Keinerlei für Tuberkulose verdächtige Befunde.'

† *Ibid.*, Bd. i. 1, p. 379.

the affected lung; it is a feature rather of the invasion stage of the disease, and commonly subsides before the crisis. In certain cases of pneumonia, however, pleurisy is a prominent feature; it extends over a large portion of the affected side and persists throughout, or even subsequent to, the pneumonic attack. In my experience such cases frequently end in empyema. When cases of pleurisy present symptoms suggestive of pneumonia, which is not very rare, a careful search should be made for a small pneumonic focus in some part of the pulmonary area.

The pleurisy accompanying pulmonary gangrene and pneumothorax is usually purulent and sometimes fœtid; that which accompanies infarction is often hæmorrhagic. Malignant disease of the lungs gives rise to an effusion which is sometimes hæmorrhagic, but not infrequently serous. Caries of any part of the bony case of the thorax commonly causes a purulent effusion.

Pleurisy in Connection with the Continued Fevers.—Pleurisy is a comparatively common complication of scarlatina, measles, and small-pox. It is not common, in my experience, in influenza or typhoid fever. It is sometimes present in diphtheria.

Pleurisy in Connection with General Dyscrasiæ.—Pleurisy is common in association with nephritis, gout, scurvy, and purpura. It is found also in diabetes; chiefly, however, in cases in which that disease is complicated with tuberculosis.

The Bacteriology of Pleural Effusion.

This subject has not yet been fully worked out, and the results obtained by different observers present many discrepancies. The following conclusions may be provisionally accepted:

1. Serous effusions are usually 'sterile.' Mitchell Prudden found micro-organisms in only two out of twenty-one cases

of pleural effusion. Aschoff found micro-organisms in only seven out of two hundred cases. Thue and Jakowski, however, found micro-organisms present in a much higher proportion of cases.

Many observers are of opinion that 'sterile' effusions are really tubercular.

2. Purulent effusions contain numerous bacteria, especially the diplococcus of Fränkel, the *Streptococcus pyogenes*, the *Staphylococcus albus*, and (rarely) the bacillus of tubercle.

3. Post-pneumonic empyema generally depends on the presence of the diplococcus of Fränkel.

4. Pyogenetic cocci are found both in serous and in purulent effusions.

5. Streptococcus pleurisy is much more unfavourable than pneumococcus pleurisy.

6. Primary acute pleurisy, not dependent on tuberculosis, is most often due to the pneumococcus.

7. Serous effusions which contain pyogenetic cocci have a marked tendency to become purulent.

8. A large lymphocytosis at an early stage of the effusion is believed to be relatively common in tubercular cases.

Varieties of Pleurisy.

The only satisfactory classification of pleurisies would be one founded upon an ætiological basis, which, as we have seen, is not feasible in the present state of our knowledge. The distinction often drawn between dry pleurisy (*pleuritis sicca*) and pleurisy with effusion (*pleuritis exudativa*) is valueless. It does not depend upon any difference in cause, nor does it give us any definite clue to the course and event of the disease in any particular case. It has been no uncommon event in my experience for a 'dry pleurisy,' which has persisted for a considerable time without effusion, to give rise to a sudden and large effusion.

Symptoms and Course of Pleurisy.

These are well described in all text-books, and need be only briefly adverted to here.

After an onset, which may be either quite sudden, or more commonly extending over two or three days, the patient seeks advice for 'stitch in the side' and increasing dyspnœa. His temperature is usually raised, but does not often exceed 102° or 103° F. He has a short, painful, unsatisfying cough, and the sputum is either absent or mucoid in character. His appearance is one of anxiety and discomfort rather than of prostration. He lies at first on his back, or on the sound side, often curled up in a constrained position in the bed, or leans forward and presses his hand to the affected side, being instinctively aware that relief comes from diminished movement of the thorax. In addition to the 'stitch in the side,' there may be a 'referred pain' in various parts of the thorax or abdomen. The breathing is shallow and ineffectual, but there is not the intense dyspnœa characteristic of pneumonia. The face is slightly flushed; labial herpes is rare, but not unknown; the pulse is moderately accelerated; the secretion of urine is diminished.

With the onset of exudation, which takes place usually in two or three days, the clinical picture undergoes important changes, which have relation partly to the presence of fluid in the pleural cavity, partly to its amount and the rapidity with which it collects. The 'stitch in the side' disappears, the patient's sufferings are mitigated, but he is conscious of increasing shortness of breath, especially on exertion. He now inclines to lie upon the affected side, in order to allow the lung upon the sound side free play. In some cases, but exceptionally, he shows some degree of cyanosis, occasionally a decided pallor, especially in cases attended by a hæmorrhagic effusion. The veins of the neck sometimes swell, owing to interference with the circulation in consequence of

the effusion. Œdema of the affected side sometimes appears, especially in connection with purulent effusions. This œdema is sometimes quite circumscribed, and does not seem to follow mechanical laws. For its detection, which in some cases is difficult, Wintrich recommends that a fold of the skin on the two sides should be pinched up, and their respective thickness compared.

Leichtenstern has noted the presence of the so-called *pulsus paradoxus* in cases of pleural effusion—*i.e.*, on deep inspiration the pulse is suspended.

The further progress of the case is variable. In a majority of instances absorption sets in towards the end of the second, or in the course of the third week, and proceeds uninterruptedly until convalescence is established in a month or longer. Delay in absorption is, however, common, and a recrudescence of the fluid after removal by aspiration frequently occurs. Convalescence is often protracted, and in this respect pleurisy presents a marked contrast with pneumonia. During convalescence the patient complains chiefly of weakness and shortness of breath, and frequently the disappearance of the fluid is attended by a return of the 'stitch in the side.' The temperature presents many fluctuations, and is not characteristic. It may be normal even in cases where absorption is delayed. After aspiration the temperature may either fall if it has been previously febrile, or rise again, having been normal before tapping, and this symptom will be found to possess some prognostic significance. Where convalescence is delayed, we may have to deal either with recurring effusion; collapse, or cirrhosis of the lung; bronchiectasis; or phthisis. The clinical picture presents many variations, according to which of these conditions is present. Chronic pleurisy is the usual explanation of those cases of 'people living with one lung,' which excite the wonder of the laity. The sound lung undergoes varying degrees, often much compensatory hypertrophy.

Physical Signs and Diagnosis of Pleurisy.

The diagnosis of pleurisy, whether in the 'dry' or 'exudative' stage, is in general an easy matter. It rests essentially upon physical signs, and only in a minor degree upon history and symptoms. Difficulties in diagnosis, however, sometimes arise and may be great, or even, prior to puncture, insuperable. We shall consider these difficulties from two points of view—viz.:

(*a*) Difficulties in connection with the recognition of pleural friction.

(*b*) Difficulties in connection with the recognition of pleural effusion.

(*a*) Difficulties in Connection with the Recognition of Pleural Friction.—Pleural friction has usually the following characters: It is rubbing, scraping, creaking, grating, or crepitant in character. It is superficial. It varies in intensity in the course of the same respiratory cycle. It is audible in the majority of cases, during both inspiration and expiration. It is increased in intensity by a deep inspiration. It is not affected by coughing, either as regards its intensity or its area of distribution. It is often accompanied by friction fremitus and by localized pain. It is heard more loudly through the stethoscope than by the application of the naked ear to the chest wall.* It is usually confined to a comparatively limited portion of the chest, but exceptionally is heard over a wide area. Its most usual seats are the inferior antero-lateral and posterior regions of the chest, but it may be heard anywhere. Apical friction is comparatively rare, except in association with obvious physical signs of phthisis. Pleural friction is entirely annulled when the breath is held, unless the slip of lung over the pericardium is involved. In this case it may be excited or modified by the

* Leube, 'Diagnose der Innern Krankheiten,' vol. i., p. 173.

movements of the heart, and difficulty may arise in the differentiation of pleural friction from pericardial friction.

Difficulty in diagnosis arises in the following ways: The history and symptoms may accord with a diagnosis either of pleurisy or pneumonia; the only physical sign present is a sound at one base which may be either crepitus or crepitant friction. This is no imaginary difficulty, nor, when we reflect upon the varying character and amount of the pleural exudate in different cases, is there any room for surprise that the sound produced by the rubbing together of the inflamed pleural surfaces should be sometimes of a 'crepitant' quality. In such cases the following rules may help us to differentiate 'crepitant' friction from 'crepitant' râle:

1. Pleural friction is more 'superficial' than crepitant râle. The term 'superficial' as applied to a sound in the lungs involves, of course, both an observation and an inference. We cannot directly observe the 'superficiality' of a sound. What we mean by the expression is this: I hear a sound of a certain quality, and from that quality I infer that the sound is produced on the surface of the lungs, and not in the ramifications of the bronchioles or in the air cells. It is obvious that error may lurk either in the observation or in the inference. Further, it must be admitted that changes in the lung which increase its capacity for conducting sound may give to a sound a character of 'superficiality' which does not really belong to it. Yet, after making all deductions, the superficiality of a sound is an important characteristic, which may be recognised with considerable certainty, and, being recognised, goes far to prove that a certain sound is of pleural, and not pulmonary, origin.

2. Pleural friction is in the great majority of cases audible both during inspiration and expiration; crepitant râle is in the great majority of cases limited to inspiration, frequently to the end of inspiration. There are exceptions to this rule—*i.e.*, friction may be limited to inspiration (this is compara-

tively rare), and crepitant râle may be audible both during inspiration and expiration (this is less rare); but, on the whole, the rule is a useful and usually trustworthy one. A sound confined to the end of inspiration is probably a crepitant râle.

3. Pleural friction is not altered in quality or area of distribution by coughing. Crepitant râle may be altered by coughing, both as regards quality and area of distribution.

4. Pleural friction is usually accompanied by local pain; crepitant râle is not.

5. Pleural friction is often intensified by pressure of the stethoscope; crepitant râle never.

6. Examination of the chest in the neighbourhood of the doubtful sound may reveal unambiguous pleural friction or crepitant râle.

7. Pleural friction may be attended by imperfect expansion of the side, owing to the pain caused by the respiratory movements; crepitant râle is not.

The distinction between pleural friction and coarse rhonchi may occasionally give rise to difficulty, especially as the tactile signs in the two cases may have much in common. The following rules may help to establish the distinction between the two sounds:

1. Pleural friction never possesses any definitely musical quality; rhonchus usually does possess such quality.

2. Pleural friction is unaffected by coughing; rhonchus is usually much affected by coughing.

3. Pleural friction has usually an interrupted or intermittent (*saccadierte*) quality; rhonchus has not.

Pericardial friction is usually distinguished without much difficulty from pleural friction. It is much more rapid than pleural friction. It has a peculiar 'to-and-fro' quality. It is confined to the cardiac area. It is definitely related to the cardiac movements, and is independent of the respiratory movements. It is usually loudest when the breath is held

after a deep inspiration. It is louder when the patient sits up or leans forward than in the recumbent position.

Miliary tuberculosis of the pleura may give rise to signs simulating pleural friction.

(*b*) DIFFICULTIES IN CONNECTION WITH THE RECOGNITION OF PLEURAL EFFUSION.—These difficulties, usually slight, are in exceptional cases great, and even insuperable. The best observers are agreed that the signs of pleural effusion and of pulmonary consolidation are in some cases indistinguishable. This is, however, rare.

The ordinary physical signs of pleural effusion are as follows:

Inspection.—The affected side has its mobility impaired, and lags in expansion behind the sound side. It may be more prominent than the sound side. It may be obviously larger than the sound side, but to this rule there are many exceptions. The intercostal spaces are usually, not always, flattened, while bulging of the intercostal spaces is extremely rare, and almost confined to cases of chronic empyema. The cardiac impulse is usually displaced towards the sound side—a sign often more evident in left-sided than right-sided effusions. In certain very rare cases visible pulsation may be seen over the area of the effusion. The excursion of the diaphragm is limited. Epigastric pulsation may be visible. The line in the inferior axilla marking the lower limit of the lung is obliterated (Litten's sign).

The patient lies upon the affected side.

A diagnosis of pleural effusion by inspection alone is sometimes possible.

Palpation.—Vocal fremitus is, in the large majority of cases, diminished or lost. As the thickness of the pleural exudate increases from above downwards, the diminution of vocal fremitus is usually more marked in the inferior portions of the chest. In some cases, however, of pleural effusion the vocal fremitus is normal or even increased. This

phenomenon is due probably to the presence of pleural adhesions, and Gerhardt is of opinion that when we can recognise islets of increased vocal fremitus in the midst of the dull area we may diagnose the presence of sacculated effusion and of pleural adhesions.*

At those portions of the chest where the compressed and collapsed lung comes into contact with the chest wall the vocal fremitus is usually increased.

Percussion.—Dulness is present according to the amount of effusion. The dulness is often wooden in quality and increases in intensity towards the base. The outline of the dull area is often characteristic. The highest point of the dull area is frequently posterior, even in ambulatory patients; sometimes it may be in the axilla, Ellis's curve being well marked. The lowest point of the dull area is nearly always in front. The upper limit of the dulness recedes in the neighbourhood of the spine, because the compressed lung in this situation does not yield a completely dull note. The outline of the dull area is little, if at all, affected by alterations of posture on the part of the patient; but in some cases, when the patient sits up, the dulness in front becomes more pronounced without altering its outline, owing to a limited motility of the fluid in its capsule. In passive effusions the dulness is much affected by changes of posture.

Capsulated effusions and interlobar effusions may cause the outline of dulness to present the widest variations, which may give rise to difficulty in diagnosis.

Above the level of the effusion we find important signs on percussion. The pitch of the percussion note is lower than normal, especially in the infraclavicular and mammary regions, because the tension of the lung on the affected side is reduced. The lung tissue has normally a certain tension, which in large measure determines the pitch of the normal

* Quoted by Leube, *op. cit.*, p. 176.

percussion note. If this tension is reduced, as happens in pleural effusion, the pitch of the percussion note falls. If the tension is still further reduced, as in large effusions, we find in many cases under the clavicle on the affected side that well-known form of tympany—*skodaic resonance*. In some cases, but in my experience very rarely, the cracked-pot sound may be audible in this area.

The percussion phenomena in Traube's Half-Moon Space are interesting and important. This space is situated in the left side of the thorax, its limits being—above, the lower margin of the left lung; on the right, the liver; behind, the spleen; below, the curve of the ribs. Normally, this area gives a tympanitic note on percussion, and the character of the note gives us information regarding the position of the diaphragm on the left side. If this space is enlarged vertically, we may conclude that the diaphragm on the left is higher than normal, and *vice versâ*.* In many cases of small pleural effusion the Half-Moon Space retains its tympanitic note, but with the extension of the effusion forwards this note is impaired, the diaphragm descends, and finally the note becomes absolutely dull.

Grocco has described a paravertebral patch of partial dulness obtainable on the sound side posteriorly. It is triangular in outline, with the apex of the triangle upward.

Percussion gives us information also regarding the displacement of organs. Cardiac dulness extends farther than usual towards the sound side. In right-sided effusions liver dulness extends below the right costal margin. With the subsidence of the effusion, these organs usually return to their normal position, but permanent displacement of the heart owing to adhesions is common.

The recognition of small effusions by percussion signs may present considerable difficulties. No obvious dulness may

* Unverricht in Ebstein and Schwalbe's 'Handbuch der Praktischen Medizin,' Bd. i. 1, p. 394.

be demonstrable, especially if the percussion stroke be forcible. Light percussion should be practised in these cases, and Gerhardt advises that we should first percuss the lower limit of pulmonary resonance on both sides from the nipples to the posterior scapular line while the patient sits; then direct the patient to lie upon the outstretched elbow on either side, when it will be found that the upper limit of dulness on the affected side will be above the line originally marked out.* Increased sense of resistance, often marked, is usually present.

Auscultation.—The breath sounds are usually weakened, often annulled, on the affected side, this condition being more marked as we auscultate from above downwards. This enfeeblement of the respiratory sound depends on three factors—viz., compression of the lung by fluid, the interposition of a layer of fluid between the chest wall and the lung, and the interference with the respiratory movements.

At a certain stage in the progress of the case, the air cells are collapsed while the bronchi remain open. Under these conditions bronchial breathing may be expected to be heard. It is usually weak and 'distant' in character, and has rarely, if ever, that loud insistent quality which we find in pneumonic consolidation. It is generally best heard in the interscapular region, but may be audible over the whole of the affected side, especially in children. As the effusion increases, the bronchial tubes usually become collapsed, and bronchial breathing gives place to weak breathing—often faintly bronchial in quality—or silence. In some cases, the breathing—especially in the mammary region—is almost cavernous in character. Over the unaffected side the breathing is harsher than normal, owing to compensatory hyperactivity (puerile breathing, vicarious breathing). There are no adventitious sounds until absorption

* Quoted by Leube, *op. cit.*, vol. i., p. 176.

has begun, when friction is usually heard, and crepitant râles, due to the opening up of collapsed air cells, are frequently present.

Vocal resonance is usually reduced, but under conditions similar to those which give rise to bronchial breathing it may be increased, even to the extent of bronchophony. Ægophony is often heard, especially posteriorly about the upper level of the effusion.

Mensuration.—The semi-circumference of the affected side is, in most cases, increased, but there are numerous exceptions to this rule.* Gerhardt has pointed out that the sound side also measures more than normal, owing to its increased activity in response to the respiratory requirements of the organism.†

The Blood in Pleurisy.—The following account of the state of the blood in pleurisy is given by Gibson :

The blood may be normal.

In early cases of dry and serous pleurisy there may be a distinct leucocytosis, and the predominant elements in such cases are the polynuclear neutrophiles. An absence of leucocytosis at an early stage in such cases is rather suggestive of tuberculosis. In pleurisy due to the pneumococcus high counts of polynuclear leucocytes are common. In almost all cases of purulent pleurisy the blood shows a considerable increase of the polynuclear neutrophiles, and usually a considerable diminution in the number of lymphocytes. Leucocytosis is the rule in cases of malignant pleurisy, and in such cases, together with an excess of polynuclear neutrophiles, coarsely granular eosinophiles are in excess, while myelocytes are not infrequently present.‡

* See p. 63. † Quoted by Unverricht, *op. cit.*, p. 395.

‡ G. A. Gibson, 'Some Thoughts on Pleurisy,' *British Medical Journal*, January 7, 1905.

Difficulties in the Diagnosis of Pleural Effusion.

Errors in diagnosis between pleural effusion and pneumonic consolidation often arise from forgetfulness of such well-known facts as—

(*a*) That bronchial breathing is common in effusion.

(*b*) That weak or suppressed breathing is common in pneumonia when the bronchial tubes are blocked by secretion.

The bronchial breathing of pleural effusion, as already pointed out, is usually weak or 'distant' in quality, often better heard near the spine than at the base posteriorly.

Consolidated lung, with blocking of the bronchial tubes by secretion, gives rise to a group of physical signs not distinguishable from those of pleural effusion. In such cases we find dulness on percussion, absence of breath sounds, and diminution of vocal resonance. The position of the cardiac impulse is here the best touchstone of diagnosis, while the history of the case, the character of the sputum, attention to the outline of the dulness, which in pneumonic consolidation never possesses the outlines characteristic of effusion, the degree of sense of resistance present, and the fact that the blocking of the tubes is only a temporary phenomenon in pneumonia, and may at any movement be relieved by expectoration, will usually keep us right. Puncture of the side may, however, be the only final test, but is not entirely free from fallacy, as we may from various causes fail to find fluid when it is clearly present; and, on the other hand, small effusions are not very rare in association with pneumonia.

Malignant disease of the lungs may give rise to physical signs closely simulating those of pleurisy—viz., wooden dulness, loss of vocal fremitus, and absence of breath sounds; and a further difficulty arises from the fact that malignant disease of the lungs is often accompanied by effusion, serous or hæmorrhagic. In such cases we should direct our atten-

tion to the signs and symptoms of pulmonary new growths, which are most helpful in diagnosis—viz., persistent intra-thoracic pain, increasing dyspnœa aggravated by exertion, local fulness or bulging, displacement of organs, loss of weight, progressive cachexia.

The condition of the vocal fremitus should always be carefully weighed in any case where the presence of pleural effusion is in question. In the great majority of cases of effusion it is diminished or absent; but, as already pointed out, the existence of adhesions may lead to its preservation, rarely to its exaggeration. Decided increase of intensity of the vocal fremitus raises a presumption against the presence of pleural effusion. Diminution of vocal fremitus must be interpreted with caution. This is the rule in pleural effusion, but is also common in pneumonic consolidation when the bronchial tubes are blocked by secretion.

Gee has given a good account of certain cases of pleural effusion in which the physical signs simulate those of pulmonary cavity. Several cases of this kind have occurred in my experience. As this point is not usually much dwelt on, I will state Gee's views in full: 'At places the breath sounds (*i.e.*, in pleural effusion) may be bronchial in all degrees of intensity up to a hollow resonance such as Laennec himself would have supposed to leave no doubt of cavity. Add to the physical signs hectic fever, and we cease to wonder that pleurisy of this kind is usually mistaken for phthisis more or less advanced. However, the pleuritic patients recover completely without a vestige of disease left behind, save, haply, a slight unilateral retraction of the side or a cup-like depression. Whenever the signs of a supposed phthisis are in some respects peculiar, whenever they indicate advanced disease limited to one side of the chest, whenever cavernous signs are heard in unusual places, it is well to weigh the possibility of a simple pleurisy. The most useful guide to diagnosis is this: that, as a rule, the signs of pleurisy are

more marked in the lowermost part of the chest, and that the signs of phthisis are more marked at the upper part.'*

I will add two comments on these cases, which, I believe are not excessively rare. Gurgling râles are not heard in the pleuritic cases, and the position of the cardiac impulse may give us an important hint for diagnosis.

The following diagnostic problem is common in practice: A patient is seen on the second or third day of a severe illness, characterized by sharp pyrexia, dyspnœa, and thoracic pain. The physical signs, for some of the reasons already adduced, are ambiguous. How shall we decide whether we are dealing with commencing pleurisy or commencing pneumonia, other alternatives being excluded? The following rules may help us in such cases:

1. A sudden onset may occur in either disease, but is much commoner in pneumonia. A gradual onset, extending over three or four days, is common in pleurisy, rare in pneumonia. A single severe initial rigor is the rule in pneumonia; a series of slighter chills is the rule in pleurisy.

2. Marked alteration in the pulse-respiration rate is strong evidence of pneumonia.

3. Marked physical prostration coming on early is suggestive of pneumonia rather than pleurisy. The pneumonic patient often lies like a log in bed, and raises himself with difficulty for examination. The pleuritic patient is restless and anxious, but not prostrate.

4. A respiration rate of 40 per minute or more, accompanied by marked *besoin de respirer*, is much more likely to occur in pneumonia than in pleurisy.

5. Mental symptoms—delirium, excitement, stupor—are much commoner in pneumonia than in pleurisy.

6. The pneumonic sputum is scanty, viscid, rust-coloured, or sanguineous. The sputum in pleurisy, when present, is catarrhal and uncharacteristic.

* Gee, 'Auscultation and Percussion,' third edition, p. 221.

7. The urine in pneumonia is deficient in chlorides, and often contains albumin. The urine in pleurisy is ordinary febrile urine, without significant characteristics.

Pain in the chest is common in both diseases, and has no weight in diagnosis. Labial herpes is relatively common in pneumonia, but is not to be relied on in a doubtful case. The course of the temperature as time goes on in most cases definitely differentiates the two diseases, but may be misleading at first. Pneumonia may have a temperature of only 101° or 102° for the first day or two, while the temperature in pleurisy may be 103° or 104° at the outset.

Some degree of pleurisy is always present in pneumonia, but genuine pleuro-pneumonia, which is common in cattle, is rare in man. The diagnosis of pleuro-pneumonia is usually the refuge of the weak diagnostician.

On the whole, I have more often suspected pneumonia where the subsequent development proved the case to be one of pleurisy than *vice versâ*. Sthenic cases of pleurisy in young people are apt to be misleading. The following axioms may sometimes be of service:

(*a*) In presence of a case which I suspect to be pneumonia, but which I recognise may be pleurisy, watch the position of the cardiac impulse, weigh symptoms.

(*b*) In presence of a case which I suspect to be pleurisy, but which I recognise may be pneumonia, watch the vocal fremitus and vocal resonance, note the pulse-respiration rate, ask for the sputum, examine the urine.

(*c*) Do not give a diagnosis of pleuro-pneumonia; it is usually a wrong diagnosis. In the great majority of cases the disease is either pneumonia or pleurisy, not a hybrid.

(*d*) If the case is really obscure, postpone a definite expression of opinion. A few hours may make the case quite clear.

Pleurisy and Phthisis.—The suspicion of phthisis sometimes arises in the course of pleural effusion. West speaks

as follows on this subject: 'If the lung be adherent at the apex, the physical signs may become puzzling. Thus, the breath sounds may be bronchial, there may be bronchophony and occasional crepitation. It will then be impossible to say that the patient is not suffering from phthisis, and it will be necessary to wait until the fluid has disappeared before this question can be settled. In a very large number of cases all these suspicious signs disappear when the fluid has disappeared, and no evidence of apex mischief remains.'* This account of these cases is quite in accord with my own experience. It is a good clinical rule not to give an opinion as to the existence of phthisis during the continuance of pleural effusion. The character of the fluid does not give us any decisive assistance. A sanguineous effusion lends support to the suspicion of tubercle, but a serous effusion may be present in any form of pleurisy. A purulent effusion suggests pneumococcal invasion rather than tubercle. Bacteriological examination of the fluid has so far failed to distinguish decisively tubercular from non-tubercular pleurisies.

Dry pleurisy (*pleuritis sicca*) may be mistaken for any of the following conditions: pleurodynia, intercostal neuralgia, periostitis of the ribs, trichiniasis of the intercostal muscles. As a rule, the differential diagnosis in such cases is not difficult. In pleurodynia the muscles are tender to the touch, movement is acutely painful, the temperature is normal, and there are no auscultatory signs. In intercostal neuralgia a careful attention to the seat, character, and radiation of the pain will usually prevent error. In periostitis of the ribs local tenderness, with, perhaps, redness and swelling, will probably be marked, and the general signs and symptoms of pleurisy will be absent. Trichiniasis may be recognised by the detection of the parasite in the muscles.

Dry pleurisy may be one of the earliest signs of tumour of

* S. West, 'Diseases of the Organs of Respiration,' vol. ii., p. 664.

the mediastinum, pleura, or lungs. In such cases we must be on our guard against recognising pleurisy as a final diagnosis, and have regard to the case as a whole.

Prognosis in Pleurisy.

The prognosis of pleurisy involves many interesting points, but the subject is much perplexed by the uncertainty which prevails as to the precise relation between pleurisy and tuberculosis.

The following are the points of most importance in prognosis:

(*a*) *The Mode of Onset of the Attack.*—An acute onset is more favourable than a subacute or a chronic onset.

(*b*) *The Character of the Fluid.*—The most favourable type of fluid is clear serum, not rich in corpuscular elements. When corpuscular elements are abundant, the chance of pus forming is proportionately greater. Empyema has its own prognosis. Dark, grumous, and sanguineous fluid is unfavourable. It points to the presence of some dyscrasia—Bright's disease, cirrhosis of the liver, scurvy, tubercle, cancer. Post-pneumonic effusions do well, the pneumococcus being usually present. Staphylococcus effusions are favourable.

(*c*) *The Course of the Pyrexia.*—Subsidence of the pyrexia in ten days or a fortnight is favourable; a hectic temperature is unfavourable. Recrudescence or exacerbation of the pyrexia after aspiration is unfavourable.

(*d*) *The General Condition and State of Nutrition of the Patient.*—Progressive anæmia, wasting, and asthenia are unfavourable, both from a general point of view and as tending to confirm the suspicion of tuberculosis.

The dangers of the acute stage are, in general, slight. In thirty-nine consecutive cases in my practice, death took place in two instances: in one, from meningitis; in the other, from heart failure consequent on delirium tremens. In

neither case did the fatal result seem to be directly dependent on the disease of the pleura. The gravity of pleurisy turns not so much on the dangers of the acute stage as upon the risks of a tedious convalescence, of the permanent disablement in more or less degree of the lung upon the affected side, of the development of empyema, or of ultimate phthisis. Sudden death in cases of large effusion—presumably from heart failure—has been occasionally recorded, but is rare. It has not occurred in my experience. In many cases after recovery from pleurisy, some degree of dulness on percussion and enfeeblement of the vesicular murmur remains permanently on the affected side. If we can satisfy ourselves that these signs are due simply to thickening of the pleura, they are without prognostic importance. The transformation of a limpid, serous effusion, not rich in corpuscular elements, into pus is probably somewhat unusual.

Our attitude towards tedious cases of pleurisy is often a difficult one, the chance of tuberculosis being always before our minds. It is not well to assume, however, that every intractable case of pleurisy is on the road to phthisis. Quite a considerable number of these cases ultimately make a good recovery. Much depends upon the patient's occupation and mode of life, something upon his intelligence and tractability, and not a little upon careful treatment.

The Treatment of Pleurisy.

The treatment of pleurisy from the point of view of cause is usually impossible. On the rheumatic theory of the causation of some cases of the disease, the value of the salicin compounds might be expected in certain cases to be evident. After some trial of these remedies in pleurisy, I have never seen a case of decided benefit from them. To have the possibility of tuberculosis before our minds in treating cases of pleurisy is a good general principle, but this consideration should not be allowed to dominate the field of therapeutics

in any exclusive fashion. The general principles of treatment would appear to be:

(*a*) To relieve symptoms, such as pain and dyspnœa, as they arise.

(*b*) To promote the absorption of the effusion.

(*c*) To prevent, as far as may be possible, pleural adhesions.

(*d*) To avoid all depressing and debilitating lines of treatment, and to give a preference, as far as possible, to tonic measures.

(*e*) To exercise special care during convalescence, and to protect the patient in every possible manner against the risk of tuberculosis.

The Relief of Pain.—This is the most urgent indication in the early stage of the disease, and can be best accomplished by a hypodermic injection of morphia or a few doses of Dover's powder, coupled with some form of local application, either a linseed poultice with some laudanum sprinkled upon it, dry cupping, the ice-bag, or anodyne liniments. Some limitation of the freedom of movement of the affected side is often advisable. Roberts recommends the fixation of the affected side by strips of plaster, a plan which sometimes succeeds well, but is found to be rather an embarrassment than a relief by some patients. It has the disadvantage that it interferes with percussion and auscultation, and hence with our observation of the progress of the case. Wet cupping, flying blisters, and iodine applications are better avoided.

Treatment of the Effusion.—In the early stage of exudation the treatment should be simple and on general lines, as there can be no doubt that most pleural effusions are spontaneously absorbed. A febrifuge mixture and gentle stimulation of the emunctory organs are all that is necessary. Such remedies as citrate of potash, bitartrate of potash, acetate of ammonia, sulphate of soda or magnesia, and an

occasional small dose of calomel may be employed. Severe purgation and active diaphoresis are usually unnecessary, and probably injurious. The diet had better be adjusted in relation to the temperature and the digestive capacity. A severe limitation of fluids is not advisable. I have not tried the salt-free diet which has been recently recommended. It is important to distinguish the treatment of effusions which show a tendency to spontaneous absorption—happily a large proportion of cases—from the treatment of effusions which tend to become chronic or to recur. In the former class of case our treatment may well be simple, and in the main expectant. In the latter class of case the therapeutic problems which arise are important, and sometimes difficult.

If absorption is delayed—*i.e.*, if no progress is manifest by the middle or end of the third week—it is probable that the case belongs to the less favourable type, viz., that in which spontaneous absorption is not going to occur, and more active measures become necessary. The choice now lies between freer stimulation of the emunctories on the one hand, or tapping upon the other. I have no hesitation in decidedly preferring the second alternative; it is easy, safe, prompt, and efficacious; while the former measures are of doubtful value, and tend to weaken the patient under circumstances in which, the possibility of consecutive tuberculosis being kept in view, all debilitating methods are undesirable. I have seen cases where, in spite of drenching perspirations and profuse purgation, the effusion remained absolutely stationary. If, however, at this stage tapping is for any reason discarded or postponed, our reliance will be chiefly in such remedies as digitalis, squill, diuretin, calomel, and saline purgation. Hay's method of giving salines in concentrated form before breakfast is spoken favourably of by some authorities. Pilocarpine is not a trustworthy drug, and hot-air baths, hot packs, and all similar measures are inadvisable. In most tedious cases of pleural effusion we

have to resort sooner or later—and better sooner than later—to tapping.

Tapping in Pleural Effusion.—Tapping, with or without aspiration, is a modern procedure, and it is well sometimes to ask ourselves the question: What became of pleural effusions in the days before tapping? Undoubtedly the great majority of them were absorbed. Nevertheless, in suitable cases, and performed with due precautions, tapping is a proceeding of the highest utility. It is immediately effective in a certain proportion of cases, while it starts other cases on the road to recovery. It is an operation which does not usually involve any difficulty, and is in the great majority of cases devoid of risk. A larger experience has falsified the fears regarding the operation at one time expressed by Besnier, Wilson Fox, and others.

Rules for Tapping.—The following are the rules by which I am guided in advising tapping:

1. Tap at any stage of the process of effusion if symptoms of immediate urgency are present—*e.g.*, much dyspnœa, marked interference with the action of the heart, cyanosis.

2. Tap in all cases of large effusion, either filling the entire pleural cavity or at least extending up to the second rib.

3. Tap in cases of moderate or small effusions if there are no signs of commencing absorption by the middle or end of the third week.

4. Tap if the effusion is bilateral.

These rules do not require much amplification. The advisability of tapping in presence of urgent symptoms is manifest, and the procedure is usually at once justified by the prompt relief which it affords. That large effusions should be tapped without delay is probably sound, though I have not chanced to see any of those cases of sudden death without warning in large effusions upon which Trousseau

laid so much stress. It is certain, however, that we are not justified in exposing our patients to such risks when we have in our hands a method whereby they can be promptly obviated. Tapping in delayed absorption of moderate or small effusions is a well-approved method, the only question being how long we should wait. Any limit must be more or less arbitrary, but delay beyond the end of the third week would seem to be inadvisable. Tapping in the presence of a double effusion is an obvious measure of precaution against serious risks.

There are no definite contra-indications as regards tapping. Pyrexia is not a contra-indication; indeed, it is not uncommon to find that the temperature falls promptly after tapping. Extreme weakness on the part of the patient is not a contra-indication. In such cases the prospect of benefit or relief far outweighs the slight risks of the operation. Old age is not a contra-indication. I have performed aspiration with good results in a patient aged eighty-seven. The probable presence of tubercle can no longer be held to be a contra-indication, in view of the probability that a large proportion of cases of apparently simple pleurisy are really tubercular in character. The chronicity of the case does not preclude tapping. West has seen complete re-expansion of the lung after tapping in a case of effusion of eighteen months' duration.* Many cases of benefit following tapping in cases of chronic effusion have come under my notice.

The question of the light thrown upon the propriety of tapping by the quality of the fluid, as ascertained by a preliminary puncture, is an important one. If the fluid be serous, the tapping, if otherwise indicated, may proceed. If the fluid be purulent, tapping should be performed, although a subsequent operation by free incision and drainage will in most cases be required. If the fluid be bloody, the question

* S. West, 'Diseases of the Organs of Respiration,' vol. ii., p. 685.

of tapping requires much consideration. A bloody effusion may be found in the following conditions: Tuberculosis, cancer, granular kidney, cirrhosis of the liver, hæmophilia, purpura, malignant fevers, pulmonary infarction, aneurysm. It is evident that in these several conditions the question of tapping is complicated by so many other considerations that no definite rules can be laid down. As a broad rule, tapping should be performed cautiously in bloody effusions, and we should be prepared at any moment to stay our hand. I have tapped a good many cases of bloody effusion, occasionally with benefit, oftener without definite influence upon the case, never with serious damage to the patient.

Mode of Performing the Operation.—A preliminary puncture with an exploring syringe is advisable. Aspiration is not always necessary. A trochar and an evacuating-tube are sometimes sufficient. If aspiration be decided upon, either Dieulafoy's or Potain's aspirator may be employed. In careful hands both of these are excellent instruments, but I am disposed to give the preference to that of Dieulafoy, inasmuch as, though somewhat more difficult to manage, it permits of a readier control of the amount of vacuum than the instrument of Potain. Strict antiseptic precautions should be observed. The entrance of air into the pleural cavity cannot always be prevented, but does not usually lead to any serious consequences. A large or a medium-sized trochar or needle should be employed. A small trochar or needle often leads to difficulties, especially in dealing with thick effusions, which may not flow readily through a fine tube, and in our desire to spare the patient a very slight additional pain we may hamper ourselves unnecessarily, and ultimately put the patient to far more serious inconvenience.

Some good authorities prefer a simple hollow aspirating needle attached to a gum elastic tube, which is filled with fluid before using, and the outer end discharges under some aseptic fluid. This method is slower than the use of the

aspirators of Potain or Dieulafoy, but it is a quite adequate method.

Place of Puncture.—The points of election for puncture are either—

(*a*) The mid-axillary line in the sixth space on the right side, and in the seventh upon the left; or

(*b*) Below the inferior angle of the scapula in the eighth space.

The advantages of the former site are that the chest wall is thinner there and more easily pierced; that the pleura is not usually much thickened; that the ribs are not much pressed down upon each other, and there is therefore ample room. The disadvantages of this site are that there is some (not much) danger of wounding important vessels; that there is a possibility of wounding the heart or liver; that the patient can watch the operation; and that encapsuled effusions may sometimes be entirely missed.

The advantages of the posterior scapular site are as follows: It is remote from important vessels and viscera; it facilitates the assumption of the correct posture by the patient; it prevents the patient from observing the details of the operation. The disadvantages of this site are that the chest wall may be thick and difficult to pierce, owing to muscle or fat; that the pleura is often much thickened at this point; that the ribs are sometimes pressed down, and the intercostal spaces consequently narrow.

In inserting the trochar or a needle, a point should be selected near the upper level of the rib and a vigorous thrust given. The intercostal artery is very seldom wounded.

It is not advisable to attempt to empty the pleural cavity. A partial evacuation is sufficient, as absorption proceeds subsequently.

Dangers of Tapping.—These are of two kinds—viz.:

(*a*) Dangers arising during the performance of the operation.

(*b*) Dangers arising subsequent to the performance of the operation.

(*a*) *Dangers arising during the performance of the operation.*—The chief of these is syncope. A slight degree of syncope is not uncommon. Severe syncope has, however, been rare in my experience, and I have never seen a fatal case.

Syncope may be averted by attention to the following points:

1. The posture of the patient. The patient should on no account be allowed to sit up during tapping. He should be semi-recumbent, with the shoulders and head well propped up with pillows, and with the affected side turned towards the operator.

2. If the pulse be weak, a little alcohol may be given before tapping is begun and $\frac{1}{20}$ grain of strychnine may be injected hypodermically. Digitalis and strophanthus are too slow in their action to be of service in this connection.

3. Anæsthesia, either local or general, should be avoided. It is unnecessary and not free from risk. The amount of pain inflicted by the operation is not serious.

4. If syncope threatens we should suspend the operation.

Coughing sometimes arises during tapping, and has various causes. Sometimes it is merely reflex. The patient should be instructed to restrain it as much as possible, and may take a little brandy. If the coughing is severe, the operation should be temporarily suspended, and if it proves uncontrollable, which is unusual, tapping should be abandoned.

Severe pain in the side is often complained of. It is not usually due to wound of the lung, and unless very severe, it is not a sufficient cause for abandoning the operation.

Hæmorrhage may supervene during tapping. It is most often due to rupture of vessels in the wall of the pleura, in consequence of them being deprived of their usual support. In such cases the operation should be abandoned. Hæmorrhage is hardly ever due to wound of an intercostal artery.

The case of sanguineous pleural effusion has been already considered.

(*b*) *Dangers subsequent to tapping.*—The most serious of these dangers is acute œdema of the lungs, which may set in within a few minutes or hours after tapping. The symptoms of this grave complication are urgent dyspnœa; cyanosis; rapid, weak, irregular pulse; and the expectoration of a large quantity of watery, albuminous, sometimes blood-stained fluid. The physical signs are fine, moist, crackling râles over the affected area. There is little, if any, dulness on percussion. This condition is a very grave one, but not always fatal. Alcohol, strychnine, the nitrites, oxygen, and dry-cupping may be tried, and the propriety of venesection may be considered.

Amongst the still rarer accidents occasionally subsequent to tapping may be mentioned embolism of the cerebral or pulmonary vessels; hæmorrhage from the lungs; monoplegia and hemiplegia; hemichorea; hysterical and epileptic attacks. These complications are seldom seen, and the bare possibility of their occurrence is no argument against the performance of tapping in any case where this procedure is indicated.

Treatment of Chronic Pleurisies.—Two types of case may be recognised:

(*a*) The case where, after pleurisy, there is much retraction of the lung, with little or no residual effusion. In such cases the chest wall falls in, the heart is much displaced, the patient is permanently short of breath, and probably more or less debilitated. The treatment in such cases consists in respiratory exercises, change to upland or mountain air, and attention to the general health and nutrition.

(*b*) Cases of recurring effusion. In certain cases the effusion persistently recurs after numerous tappings. Many of these cases are probably tubercular, and their management presents great difficulty.

We have to decide between the following courses:

1. To continue the tappings.

2. To incise and drain the chest, according to the method employed so successfully in empyema.

3. To try antiseptic injections.

4. To rely upon change of air and hygienic measures.

The first and third of these alternatives are probably inadvisable. The second has been favourably reported of by Aufrecht, Rosenbach, Osler, and West, but has not come into general operation. We have to weigh the gravity of freely opening the pleural cavity in cases where adhesions may be few, or weak, or even absent. In empyema we can reckon with confidence on the existence of firm adhesions; hence the pneumothorax resulting from free incision is partial in extent. The fourth plan may always be tried, and sometimes succeeds even in apparently unpromising cases. I have learnt by experience not to pass an absolutely unfavourable verdict upon even the most rebellious cases of chronic pleurisy, provided the patient's nutrition is fairly good and there is no evidence of progressive phthisis or of amyloid disease. Some of these cases can be coaxed back to a condition of comparative health by an occasional judicious tapping, suitable change of air, and attention to general tonic and hygienic measures.

LECTURE IX

EMPYEMA—PNEUMOTHORAX—HYDROTHORAX—HÆMOTHORAX—CHYLOTHORAX

SUMMARY:

Empyema:
- Bacteriology of empyema.
- Causes of empyema.
- Difficulties in diagnosis.
- Physical signs of empyema.
- Characters of the fluid.
- Distribution of the fluid.
- Complications, prognosis, mortality, and treatment of empyema.

Pneumothorax:
- Causes of pneumothorax.
- Varieties of pneumothorax.
- Symptoms and modes of onset of pneumothorax.
- Physical signs of pneumothorax.
- Diagnosis, prognosis, and treatment of pneumothorax.

Hydrothorax:
- Problems of diagnosis and treatment.

Hæmothorax:
- Problems of diagnosis and treatment.

Chylothorax.

EMPYEMA.

Empyema is often an accompaniment or sequel of pneumonia, and the pneumococcus is the most common organism present. The pneumonia may be only a small patch, and

the symptoms, though often distinctive enough, may be equivocal, and soon masked by those of the ensuing purulent effusion.

Next to the pneumococcus, the streptococcus is the most frequent micro-organism found; in the adult the latter is oftener present than the former. Streptococcal empyema is usually secondary to such conditions as pelvic peritonitis; subphrenic abscess; abscess of the stomach, liver, or spleen; otitis media; ulcerative endocarditis; general pyæmia.

In primary streptococcal empyema the original source of the disease is usually broncho-pneumonia, phthisical cavities, or pulmonary gangrene. Netter found the tubercle bacillus present in 12 out of 110 cases of empyema. The staphylococcus is relatively rare as the sole organism, but is common in association with the pneumococcus and the streptococcus. The typhoid bacillus and the influenza bacillus have been occasionally found. Most of the empyemata which occur in the course of the specific fevers—*e.g.*, scarlet fever—are of streptococcal origin. The empyema occurring in the course of phthisis is often due to the streptococcus or pneumococcus, and only in a small minority of cases to the tubercle bacillus. This fact is one more illustration of the doctrine which is becoming more and more generally recognised—viz., that a large part of the clinical picture usually attributed to the bacillus of tubercle is really due to mixed infections.

Empyema is common in infants and young children, in whom the pneumococcal variety is relatively frequent. It is a good rule always to withdraw a small quantity of the fluid in the acute pleurisies of young children. Pus will often be discovered in cases where the symptoms did not suggest its presence, and the discovery has an important bearing upon treatment.

THE SYMPTOMS OF EMPYEMA.—These are sometimes quite uncharacteristic, and not to be differentiated from those of serous effusions. The practitioner who relies upon symptoms

of septic infection for his diagnosis will be often mistaken. It has frequently been my experience to find limpid serum where a hectic temperature, perspirations, and malar flush had suggested the presence of pus, and, *per contra*, to find pus where the symptoms gave no hint of its presence. This fact requires to be emphasized, as the contrary view is taught, with more or less qualification, in some of the best text-books. It is, of course, true that in most cases of chronic empyema which have gone on for a considerable time unrecognised or unrelieved symptoms of septic absorption usually arise, but even to this rule there are exceptions.

The fact that pus may exist, even in large quantities, in various parts of the organism without causing any notable disturbance of temperature or evidences of constitutional involvement is one with which surgeons are quite familiar. In cases of chronic empyema the patients usually lose flesh to a marked degree, become anæmic, perspire freely, and have slight chills; but this entire symptom-complex may be present in serous effusions. The mode of onset will sometimes give a hint for diagnosis. The acute pleurisies of infancy and childhood which set in with a stormy or pneumonic onset are, in a large proportion of cases, purulent, and probably a small pneumonic focus is frequently present in the lungs in such cases. But the onset of empyema is even more frequently insidious in character, and marked rather by anæmia, cachexia, and debility than by respiratory symptoms.

Pain in the chest and cough may be entirely absent, but some degree of dyspnœa on exertion is always present. The temperature gives comparatively little aid in diagnosis. In many cases when they are admitted to hospital or come under treatment in private, the temperature has become, and remains, normal. In post-pneumonic empyema the temperature, which has fallen at the crisis, rises again, and remains for some time more or less hectic in character.

A difference of temperature in the two sides of the chest, the affected side being a degree or two higher than the sound side, has been noted by some observers. In a considerable number of cases marked tenderness is present on the affected side, and I am disposed to attribute some diagnostic importance to this symptom.

THE PHYSICAL SIGNS OF EMPYEMA.—These are in general the same as those of serous effusion, and in most cases no distinction can be drawn, the differential diagnosis being only possible by puncture, which should be performed in every case. In exceptional cases, however, we get certain physical signs strongly suggestive of the presence of pus in the pleural cavity—viz., local œdema, bulging of the intercostal spaces, 'pointing.' General œdema of the side occurs in serous effusions and pneumothorax, but is rare in these conditions. Local œdema of a part of one side is practically pathognomonic of empyema, if affections of the chest wall can be excluded. I have never seen bulging of the intercostal spaces in a case of serous effusion, but it is not uncommon in empyema, suppurative inflammations having a tendency to involve the subjacent tissues. According to Baccelli, whispering pectoriloquy is less well heard over purulent than over serous effusions. Senator believes that displacement of the heart and liver is more marked in empyema than in serous effusions, owing to the greater weight of the fluid. 'Pointing' is, of course, practically pathognomonic of empyema if we can exclude abscess of the chest wall, but is, fortunately, rarely seen in these days. A case of empyema can only be allowed to reach the stage of 'pointing' by gross negligence, if it has been under medical supervision. Pulsation over the seat of the effusion, a rare phenomenon, is relatively more frequent in empyema than in serous effusions.

CHARACTER OF THE FLUID.—This may consist either of ordinary creamy pus or may be sero-purulent. The colour varies from a greenish-yellow to a chocolate-brown. The

reaction is alkaline unless decomposition has taken place. The odour is slightly disagreeable, sometimes sour or acid, occasionally fœtid. The bacteriology of the fluid has been already considered.

Distribution of the Fluid.—The fluid is usually basic in seat, but loculation is the rule, and collections may occur almost anywhere. 'When the signs of a localized effusion are present in a peculiar place, the probability is greatly in favour of empyema.'* Some very puzzling cases arise by collections of fluid in portions of the pulmonary area not readily accessible to percussion and auscultation—*e.g.*, between the lobes of the lungs. In such cases puncture is the only method of establishing the diagnosis, and may not be successful at the first attempt. When an empyema lies between the base of the lung and the diaphragm great difficulty may be encountered in reaching it with the needle.

Diagnosis of Empyema.—Puncture is the final, and often the only test. Failure to find pus on a first attempt, or even after several attempts, is no proof of its non-existence, and some courage is required on the part both of physician and surgeon to persevere in spite of negative results. Perseverance is, however, in many cases absolutely essential, and will usually have its reward. The amount of pus found is often much less than the extent of dulness would have led us to expect, owing in most cases to extensive thickening of the pleura and collapse of the lung on the affected side.

Progress of the Case.—If from any cause a case of empyema is left untreated, the following results may occur:

(*a*) The pus may be spontaneously absorbed. This is probably a rare result, but there is no reason to doubt that it occasionally takes place. The chest wall falls in, the pleura is much thickened, and the pus undergoes calcareous change.

(*b*) The pus may rupture through the lung, giving rise to pyopneumothorax.

* S. West, *op. cit.*, vol ii., p. 702.

(*c*) The pus may rupture externally, usually in front, about the fourth or fifth space.

(*d*) The pus may penetrate the diaphragm and open into the peritoneum, liver, or colon, or pass down the spine along the psoas muscle into the iliac fossa, and simulate a psoas or lumbar abscess.

Many of these complications have, happily, become quite rare, owing to more prompt diagnosis and treatment.

Complications of Empyema.—If cases of empyema are promptly recognised and submitted to proper treatment, complications are rare. Complications are, however, numerous and important in connection with chronic cases of empyema, which have for some reason or other been allowed to go unrelieved. Amongst the most important complications are the following: Pericarditis, which if purulent involves a fatal prognosis; abscess of the brain; meningitis; phthisis; pulmonary cirrhosis; bronchiectasis; amyloid disease. It has not chanced to me to see any case of septicæmia, pyæmia, or gangrenous pneumonia as the result of empyema, but such cases have been described. Phthisis and amyloid disease, either separately or in association, are among the probable final developments of unfavourable cases of empyema.

Prognosis of Mortality.—West gives the general average mortality at 21·5 per cent., and states that the mortality is highest in early childhood and after forty. In young adults he estimates the mortality at about 8 or 9 per cent.* My experience of empyema in young children is rather more favourable than the above, when diagnosis has been prompt and appropriate treatment carried out. One of the worst types of empyema in my experience has been that occurring in parturient women. Thick, curdy, foul-smelling pus points to an unfavourable issue.

* S. West, *op. cit.*, vol. ii., p. 713.

TREATMENT OF EMPYEMA.—This is in the main a surgical question, but the physician is often called upon to advise with regard to it, and to decide whether tapping only or free incision and drainage, with or without resection of a rib or ribs, shall be performed. While it is not to be denied that tapping alone, repeated if necessary, will suffice for the cure of a certain number of cases of empyema—especially, I think, the pneumococcal empyemata of young children—it has been my uniform practice to advise the larger operation, which, in the hands of an expert operator, is one of the most successful in the whole range of surgery. It is highly unsatisfactory to tap an empyema repeatedly, and finally be compelled to fall back upon free incision and drainage. The delay cannot be for the patient's benefit, and the chance of complications arising may be seriously increased by a temporizing policy. Nevertheless, our hands are sometimes forced in this matter. The patient, or, in the case of young children, the parents of the patient may decline to permit the performance of the larger operation. Our attitude under such circumstances should be to consent to tap, warning those most concerned that we are doubtful of the adequacy of this procedure. How often paracentesis should be performed before we pronounce this operation insufficient to ensure a cure must be left to the discretion of the practitioner. Long delays and a too optimistic attitude are to be deprecated.

If free incision and drainage be resolved upon, a preliminary paracentesis is usually advisable. It gives temporary relief, affords a slight chance of cure, gives time for the study of the case, and perhaps in a small number of cases for such tonic and roborant measures as may serve to put the patient in a more fit state to bear the major operation. On the whole, however, the plan of waiting to fatten a debilitated patient, whose chest is, perhaps, full of pus, is to be condemned. Delay in such cases involves certain dangers and

uncertain benefits. A preliminary paracentesis is favoured by many surgeons for another reason—viz., that it renders the after-operation easier.

When free incision has been decided upon, we should in all cases make a preliminary puncture at the spot of the proposed incision, in order to establish without doubt the presence and the precise localization of the pus. Many embarrassing situations have arisen from neglect of this simple rule. The preliminary puncture should in all cases be made just before the larger operation, and we should not rest satisfied with the fact that pus had been found a few days or a week or two previously. Pus in the chest sometimes plays us strange tricks; loculation is sometimes complicated and peculiar, and there is no reason for inviting difficulties which are so easily obviated.

The question of the use of anæsthetics in these cases is a further question which it usually falls to the physician to answer. My rule is to dispense with them if the patient is willing to suffer a considerable amount of pain, but where an anæsthetic is required, to advise a moderate chloroform narcosis, or a mixture of nitrous oxide and oxygen may be tried. Ether is to be avoided as causing irritation of the bronchial tubes; freezing of the skin with ether or ethyl chloride is of little avail in the larger operations, and injections of cocaine or morphia locally are to be deprecated. I have seen a good many operations done without anæsthetics, and although the suffering must be severe, I have often found it well borne.

The details of the operation for empyema must be settled by the surgeon. I believe the removal of a portion of a rib is, at least in recent cases, often unnecessary; but the surgeon must, at all costs, make an adequate exit for the pus. The problems to be solved are mechanical, and each case must be judged upon its merits. Drainage is often a tedious affair, but we must have much patience with these

cases, and avoid an attitude of premature discouragement. In one of my cases a tube was worn for six months, but cure was in the end complete, and the patient remains well, after the lapse of many years. Unhappily, in some of these cases the discharge persists in spite of all our endeavours, and phthisis or amyloid disease is the ultimate goal.

Pneumothorax.

By pneumothorax we understand the presence of air in the pleural cavity, usually accompanied by more or less fluid—either serum, pus, or blood. The distinction between pneumothorax, hydropneumothorax, pyopneumothorax, and hæmatopneumothorax is not of much practical value. The proposal of Wintrich and Fräntzel to distinguish cases where the presence of fluid is primary and that of gas secondary as pyopneumothorax, and the much more frequent cases where the presence of gas is primary and that of fluid secondary as pneumopyothorax, does not seem a happy one.

Causes of Pneumothorax—(*a*) *Pulmonary Causes.*—By far the most frequent of these is tuberculosis, which is believed by some authorities to account for 90 per cent. of all cases.* The normal pressure in the pleural cavity is a negative pressure of 3 to 5 millimetres of mercury; hence, upon any rupture of the pulmonary substance, as may happen in tubercular lungs as the result of coughing, air rushes in to equalize the pressure within and without the pleural cavity. The occurrence of pneumothorax in phthisis has been in my experience almost confined to advanced cases of the disease, with active softening or excavation, but it is well known to occur also in the earlier stages. It must be remembered that, as phthisis progresses, adhesions form between the lungs and the chest wall, and that these

* Walsh, West, Unverricht, and others.

adhesions afford some protection against the occurrence of pneumothorax. I am disposed to think that pneumothorax is relatively common in cases where active caseation is in progress, relatively rare in cases of chronic and quiescent cavities.

Other pulmonary causes of pneumothorax are abscess of the lung, gangrene of the lung, and pulmonary infarct. Cases are also on record where the presence of echinococci in the lungs immediately beneath the pleura has caused pneumothorax. Cancer might conceivably act in the same way. Emphysema is usually mentioned among the causes of pneumothorax, on the theory that dilated vesicles situated immediately under the pleura occasionally rupture. No case suggestive of such causation has come under my notice.

Pneumothorax as a consequence of the violent coughing in whooping-cough seems more probable, though no undoubted case has come under my observation. It is evident that the lung will stand an enormous increase of intravesicular pressure when it is not the seat of infiltrating or necrotic processes.

A case has recently been under my care of a young gentleman, aged twenty-five, who developed typical pneumothorax as the result of moderate physical strain, but in whom no evidence of tubercular or other disease of the lungs was forthcoming, and who seemed in perfect health at the time of the occurrence of pneumothorax. He made a complete recovery in a few months, which lends probability to the view that the lungs were healthy.

(*b*) *Pleural Causes of Pneumothorax.*—Cases of empyema which have not been instrumentally relieved often rupture into the lung. Serous effusions never rupture in this way, so far as I am aware. Pneumothorax also occasionally ensues upon the performance of aspiration in ordinary cases of pleural effusion, and this accident is believed to be usually

due to the creation of too strong a vacuum during the employment of Dieulafoy's or Potain's aspirator, or by want of care in the control of the cocks. Pneumothorax is said to arise sometimes by the spontaneous development of gas in a pleural exudate.

(*c*) *Parietal Causes of Pneumothorax.*—Wounds of the costal parietes, fracture of ribs, diseases of the ribs, are among the well-recognised causes of pneumothorax. Their mode of operation requires no elucidation.

(*d*) *Abdominal Causes of Pneumothorax.* — Ulcers and tumours of the stomach and bowel may perforate the diaphragm and give rise to pneumothorax. In one case in my series a gastric ulcer gave rise to a subphrenic abscess, which ruptured into the left pleural cavity, and ultimately gave rise to left pneumothorax. The patient positively declined operation.

VARIETIES OF PNEUMOTHORAX.—Cases of pneumothorax may be conveniently classified as—

(*a*) General, where air occupies the general pleural cavity;

(*b*) Local, where air occupies a loculus in the pleural cavity; and

(1) Closed—*i.e.*, where the fistulous opening between the lung and the pleural cavity has become healed;

(2) Open—*i.e.*, where there is a free opening between the lung and the pleural cavity.

In some cases we get the peculiar condition described as valvular pneumothorax, in which the fistulous opening between the lung and the pleural cavity is of such a nature that air can freely enter the pleural cavity, but cannot find an exit from it. This type of case obviously approximates to 'closed' pneumothorax.

The above types of pneumothorax may not always be distinguishable during life, but as the physical signs present in any individual instance are much modified by the special features of the case, the practitioner will find it helpful to

keep the following considerations carefully in view in any case of pneumothorax :

1. Does the air occupy the whole, or nearly the whole, of the pleural cavity (allowing for the usual presence of a certain amount of fluid), or is it circumscribed in a loculus of limited size ?

2. Is there evidence of free communication between the pleural cavity and the lung or not ?

Symptoms and Modes of Onset of Pneumothorax.—When air enters the pleural cavity the lung on the affected side becomes speedily collapsed and airless. If there be no adhesions, the lung lies shrunken about its root and flattened along the vertebræ. If adhesions be present, the contraction of the lung will be irregular in fashion and dependent on the seat of the adhesions, which are most often apical. The heart and mediastinum are displaced towards the sound side. The sound lung is pressed upon, and its respiratory capacity is seriously reduced. The occurrence of pneumothorax is in the vast majority of cases sudden, and hence the lungs have no time, as they have, for example, in slowly accumulating pleural effusions, to accommodate themselves to altered respiratory conditions. The above considerations account for the fact that in the majority of cases the onset of pneumothorax is well marked and the symptoms definite and pronounced. But it must be borne in mind that a pneumothorax may be local, and hence involve much less grave consequences on respiration than in cases of general pneumothorax; also that the symptoms have a definite relation to the previous functional activity of the lung on the affected side. If this lung has been previously the seat of extensive disease, and hence of small functional efficiency, the occurrence of pneumothorax will make much less difference to the respiratory functions than if this lung has been fairly healthy previous to the occurrence of pneumothorax. Weighing these various considerations, which might easily be further

amplified, it is in no way surprising that while many cases of pneumothorax present a mode of onset and a symptom-complex which are highly characteristic, others are quite untypical.

The following are the chief features of a typical case: The patient, who may have been feeling previously fairly well, is seized with a sudden agonizing pain in the side, and feels as if something were burst or torn in the chest. The attack may ensue on active movement or severe coughing. Intense dyspnœa, perhaps amounting to orthopnœa, is set up. The patient gasps and pants for breath; he becomes rapidly cyanosed; cold perspiration breaks out over the body; his aspect is one of acute anxiety and distress; he feels as if death were impending. The veins of the neck stand out as distinct cords; the face is swollen; œdema of various parts begins to appear; the voice is weak or lost; signs of carbonic acid intoxication begin to show themselves; the patient is tormented by a hacking cough, often without expectoration.

The above clinical picture is almost pathognomonic, but confusion might arise with any of the following conditions—viz., embolism of the pulmonary artery, an asthmatic paroxysm, or large pulmonary infarction. The previous history of the case and the physical signs make the differential diagnosis of these various conditions a comparatively simple matter. In embolism of the pulmonary artery, some probable source for the embolus will be present, and, in spite of the intensity of the symptoms, there are no characteristic signs in the lungs. In asthma there will be a history of previous attacks, pain in the chest will not be a prominent feature, the dyspnœa is definitely expiratory in type, and the physical signs are those of general bronchial catarrh, with more or less emphysema. In large pulmonary infarct there will probably be antecedent heart disease, hæmoptysis is likely to be present, an area of dulness may be discoverable at the periphery of the lungs, and the symptoms are usually much less intense and acute than in pneumothorax.

In not a few cases, however, the onset of pneumothorax is much less acute and stormy. Especially in advanced phthisis, when the patient is pursuing an easy, restful life, and when the respiratory needs of the organism are at a minimum, the attack may be more or less latent in character, and only reveal itself by physical signs. In cases of local pneumothorax it is not difficult to understand that the disturbance of respiration may be moderate.

A drop in the temperature of a case previously pyrexial is usual in pneumothorax, and is probably due to shock or collapse. A subsequent rise may occur, dependent on some of the consequences of pneumothorax. The pulse is usually accelerated and weak, but is, on the whole, less violently disturbed than the respirations. During the persistence of urgent dyspnœa, the patient either sits upright or takes a semi-recumbent attitude.

The further development of symptoms depends on the progress of the case, whether towards death or recovery.

PHYSICAL SIGNS OF PNEUMOTHORAX.—As already pointed out, a considerable diversity of physical signs is to be expected, according as the pneumothorax is general or local, and according as there is free communication or not between the pleural cavity and the lungs or outside air. We shall first describe the physical signs of the most typical variety of pneumothorax—viz., when the entire pleural cavity is more or less filled with air, and the communication with the lung or outside air is imperfect.

Inspection.—The affected side is distended, the shoulder is raised, the sternum is thrust forward, the intercostal spaces are obliterated and show little or no alteration during inspiration and expiration. The impulse of the heart is displaced towards the sound side. Epigastric pulsation may be visible, especially in left-sided pneumothorax. In thin persons the downward displacement of the liver may be visible. General œdema of the side may be sometimes observed.

Palpation.—Vocal fremitus is much diminished or entirely lost. The sense of resistance is increased. The apex of the heart is felt to be displaced. The lower edge of the liver and the spleen are usually to be felt.

Percussion.—The percussion sound is hyper-resonant and of lower pitch than normal. It has been compared to the sound produced by percussing a horse-hair cushion or a band-box. The area of hyper-resonance may extend an inch or two beyond the sternum, owing to the displacement of the mediastinum, to or below the costal arch in front, and as low as the twelfth rib behind. Cardiac dulness is much displaced towards the sound side, and perhaps diminished in area. Liver dulness is displaced downwards, and may not extend higher than the margin of the ribs. The displacement of the heart is much more marked in left, displacement of the liver much more marked in right, pneumothorax. The hyper-resonant note is general over the affected side, but may be modified by the existence of adhesions or by the presence of fluid. Dulness due to the presence of fluid is much influenced by posture. It is generally held that if the tension of the air in the pleural cavity exceeds a certain point, the percussion note loses some of its resonance, and acquires the quality of 'tympanitic dulness,' or even actual dulness. This is denied by West.* I incline to the usual view.

Auscultation.—The auscultatory signs of pneumothorax are in many cases highly characteristic and interesting. The breath sounds may be almost inaudible, but oftener, in my experience, they are faint and distant, and possess a highly characteristic hollow metallic quality. In the interscapular space over the seat of the collapsed lung they may be definitely bronchial in quality. At the apex they are occasionally, but rarely, exaggerated. In rare cases the breathing over the whole side is bronchial or amphoric in

* S. West, *op. cit.*, vol. ii., p. 786.

quality and of considerable intensity. The bell-sound (*bruit d'airain*), metallic tinkling, and succussion splash are frequently present.* If râles are present they have a peculiar metallic quality. Vocal resonance is diminished or absent.

Mensuration.—The affected side is usually increased in its semi-circumference, but there are many exceptions to this rule, as retraction of the side sometimes occurs.

Variations in the Physical Signs in Pneumothorax.—These variations, which are important, depend mainly on the following considerations—viz., whether the air is in the general pleural cavity or in a loculus; whether there is free communication between the pleural cavity and the lung or outside air or not, (in other words, the degree of tension of the air in the pleural cavity); whether pulmonary adhesions exist, and if so, their extent and distribution; the amount and character of the fluid present in the pleural cavity. It is evident that these considerations afford room for wide variations.

Physical Signs of a Loculated Pneumothorax.—These may be uncharacteristic and leave us in doubt. The hyper-resonant note on percussion will often be found to pass beyond the sternal line. The breathing will probably be amphoric. Most of the physical signs enumerated as characteristic of pneumothorax will probably be absent. The history and symptoms may throw important light upon the existence of a loculated pneumothorax.

If a free opening exist between the pleural cavity and the lungs, the signs depending on increased pressure in the pleural cavity will obviously be wanting. There will be no distension of the side, perhaps even a retraction. The intercostal spaces will not be obliterated, the displacement of the heart and liver will be slight or absent. The percussion sound will be hyper-resonant, and probably the cracked-pot sound will be audible.

* See Lecture IV. for a discussion of these signs.

THE RÖNTGEN RAYS IN THE DIAGNOSIS OF PNEUMOTHORAX. — The following account is based upon the descriptions of Unverricht: In healthy individuals the lungs present a clear zone under the X rays. From the hilus we can recognise a row of dark lines ramifying a variable distance into the clear zone and corresponding to the branches of the bronchial tree. In pneumothorax we observe that the pulmonary field is much clearer than usual, and that the above-mentioned dark lines are absent. The greater part of the pulmonary field on the affected side is luminous, and it extends beyond the normal limits on that side. The collapsed lung is in the middle region, and throws a shadow. If it has been previously healthy its collapse will be all the more complete, while if it has been the seat of caseated masses or fibrous thickenings its collapse will be less complete. Hence the extent of the shadow cast by the collapsed lung may throw important light upon the previous state of the lung and possess diagnostic value. The bony structures of the thorax stand out in the skiagram of these cases more prominently than usual. The variations in the area of dulness, which, as before mentioned, attend changes of posture in cases of pneumothorax with effusion, find their analogue in corresponding changes in the skiagram. When succussion splash is present visible waves can be recognised on the surface of the fluid. Slighter movements in the fluid, synchronous upon the contractions of the heart and apparently dependent on them, can also be seen. The upper level of the fluid can be seen to fall with inspiration and rise with expiration.

These observations are of much interest, and confirm, if they do not materially supplement, our knowledge of the physical conditions which accompany pneumothorax.

DIAGNOSIS OF PNEUMOTHORAX.—In the presence of the typical symptom-complex the diagnosis of pneumothorax is not difficult, but many cases are obscure, especially if fluid is

not present, and the diagnosis of loculated pneumothorax may be impossible.

The points to be chiefly relied upon in diagnosis are the following:

(*a*) A previous history of tuberculosis, pulmonary abscess, pulmonary gangrene, or perforating wound of the chest.

(*b*) The sudden advent of symptoms, with pain, distress, and dyspnœa.

(*c*) The sudden development in the pulmonary area of physical signs pointing to the existence of a large air-containing cavity.

(*d*) The extension of the zone of hyper-resonance beyond the middle line, and the marked displacement of organs.

Unverricht attaches considerable importance to the use of the exploring needle, and believes that the freedom of movement which the inserted needle shows in pneumothorax can be distinguished from the much more limited movement which it possesses in normal lung.* As the air in a pneumothorax cavity is almost always at higher pressure than the air of the outside atmosphere, the insertion of a trochar is usually followed by a puff of air, which is very characteristic; or the tube connected with the trochar can be introduced into fluid in a vessel, when the passage of air from the cavity within the thorax will be manifest by the development of bubbles in the fluid.

When fluid is present and the characteristic succussion splash audible, the diagnosis is in general easy. Splashing sounds may be produced in the stomach, but the differentiation of these sounds from those present in pneumothorax, as a rule, presents little difficulty. The history of the two cases is quite different, and the absence in cases where stomach splashing is heard of all thoracic signs is conclusive.

Large pulmonary vomicæ may give rise to most of the signs of pneumothorax, but rarely in quite typical form.

* Unverricht in Ebstein and Schwalbe's 'Handbuch,' Bd. i. 1, p. 433.

Careful percussion will nearly always demonstrate the presence of a layer of fibroid thickening round a cavity. The signs of cavity are often much modified from time to time by cough and expectoration. I have never heard the succussion splash over a cavity.

The diagnosis of loculated pneumothorax may, however, as already stated, present great, or even insuperable, difficulties. The history and mode of onset are probably the most helpful points. The physical signs may be inconclusive. The character of the fluid present in pneumothorax can only be determined by puncture.

Prognosis of Pneumothorax.—The best outlook is in traumatic cases or in those where pneumothorax has supervened on physical strain, the lungs being presumably healthy. This is a small group of cases, but affords in general a favourable prognosis. Pneumothorax from ruptured empyema affords a somewhat unfavourable, but not definitely bad, prognosis. My own personal experience of this type of case is such as to lead me to think the outlook somewhat better than has usually been assumed. I have had three cases of the kind in the last few years under my observation. All three made a good recovery at the time. One became phthisical after a long interval; the remaining two have been lost sight of, but were well when last seen.

Pneumothorax depending on tuberculosis involves a very grave prognosis, 90 per cent. of these cases, according to West, dying within four weeks.* Douglas Powell found the mean duration of thirty-nine cases to be twenty-seven days.† Nevertheless, a minority of cases survive the accident, and in exceptional cases the activity of the tubercular mischief in the lungs becomes less marked.

Pneumothorax depending on pulmonary abscess or pul-

* S. West, *op. cit.*, p. 799.

† Douglas Powell, 'Diseases of the Lungs,' p. 141.

monary infarct involves a grave and doubtful, that depending on pulmonary gangrene a definitely bad, prognosis.

TREATMENT OF PNEUMOTHORAX. — The treatment of pneumothorax immediately after the accident is on general lines. A hypodermic injection of morphia will usually be advisable. Stimulating or soothing applications to the chest; injections of ether, camphor, or strychnine; the free exhibition of stimulants; the application of leeches, are amongst the points most worthy of consideration.

The after-treatment in cases which survive involves some important problems. Unverricht advises that all cases of recent pneumothorax should be treated by the formation of a wide external fistula, with the view of preventing the air in the pleural cavity attaining high pressure, of allowing pleural adhesions to form, and of facilitating closure of the fistula between the pleural cavity and the lung. He lays proper stress on the importance of securing that this external fistula shall not be allowed to close. In cases of long standing he thinks a similar procedure the best at our disposal, though the results cannot be expected to be so favourable as in recent cases.* Repeated aspiration of the air seems to lead to only temporary relief, the over-pressure of air in the pleural cavity being speedily renewed.

When pneumothorax has followed upon a ruptured empyema, the problem of treatment is somewhat different. The question here is whether, with a free exit for the pus *per vias naturales*, there is any advantage in incising the chest. It is well known that these cases in not a few instances undergo spontaneous cure, but their after-history is not well established. The general opinion is in favour of operating in these cases. Rosenbach says: 'If it can be positively determined that an empyema has ruptured through the bronchi, an empyema operation is to be advised, in spite of the fact that spontaneous cure in these cases not rarely

* Unverricht, *op. cit.*, p. 437.

occurs; because operation, provided the lung retains some power of expanding, is certain to hasten recovery, and is often the only means of curing the patient. To depend on spontaneous cure in such cases is not rational, as the usual results of pleurotomy present better chances for recovery than the uncertainty of internal evacuation, which fails to guarantee thorough elimination of the pus and permanent asepsis.'*

It would be interesting if we had fuller details of these cases of 'spontaneous cure' following pneumothorax consequent on empyema. They are not uncommon, but their after-history is not clear. Is the cure complete or incomplete? Do any considerable proportion of these cases become phthisical? Would that tendency have been obviated by a timely operation? How far might a phthisical tendency in such cases be eliminated by hygienic treatment or residence at high altitudes? These are interesting and important questions, but they can hardly be fully answered in the present state of our knowledge. The balance of evidence is at present in favour of treating these cases on the same lines as empyema without fistula—*i.e.*, by free incision and drainage.

Hydrothorax.

By hydrothorax we understand the collection of non-inflammatory serous fluid in the pleural cavity. The serous fluid present in such cases differs from the serous fluid of inflammatory collections by containing little or no fibrin and few red or white blood corpuscles. The cause of hydrothorax is in the great majority of cases general, not local, and depends upon certain states of the circulatory or excretory organs or upon the existence of some constitutional dyscrasia. It may also arise from local new growths or from

* Rosenbach in 'Nothnagel's Encyclopædia of Practical Medicine,' article 'Pneumothorax.'

aneurysm. As a rule, hydrothorax does not arise as an isolated phenomenon, but is part of some general disease—cardiac, pulmonary, renal, or hæmic. It is worthy of separate study, however, inasmuch as certain problems of diagnosis and treatment arise in connection with it.

HYDROTHORAX IN HEART DISEASE.—Hydrothorax is a very common feature of cases of heart disease attended by general dropsy. As a rule, it is not an early symptom, but supervenes in cases where subcutaneous œdema has been for some time present. Its development often runs *pari passu* with dropsy of the peritoneum. It is nearly always bilateral, but it is remarkable how often the amount of fluid present in the two pleural cavities shows such differences in quantity, so much so that we may have a large effusion in one side, while we may have some difficulty in demonstrating the presence of any fluid in the other. Unverricht thinks that in certain cases when one pleural cavity has been the seat of previous disease hydrothorax may be confined to this side.* It is probable that postural influence may have something to do with the usual excess of fluid in one pleural cavity, but this has not seemed to me to be the usual explanation. The practitioner would do well to remember that exacerbations of dyspnœa in cardiac disease often depend upon an increase of fluid in the pleural cavities, and may be relieved by tapping.

HYDROTHORAX IN PULMONARY DISEASE.—Emphysema is the principal case in point. Hydrothorax in association with emphysema depends upon failure of the right heart, so common in advanced cases of that disease. Hydrothorax in these cases is a grave symptom, and often presages the beginning of the end. It can be best relieved by cardiac remedies—digitalis, strophanthus, strychnine, caffein.

HYDROTHORAX IN RENAL DISEASE.—Hydrothorax arises usually in connection with parenchymatous nephritis and

* *Op. cit.*, p. 441.

amyloid disease. It is quite rare in granular kidney. Its significance is, on the whole, less grave in renal than in cardiac cases, and the prospects of relief and cure are proportionately better. The treatment must have regard to the general renal condition, but cardiac tonics are often required.

HYDROTHORAX IN THE VARIOUS DYSCRASIÆ.—Hydrothorax may be found in severe anæmias, scurvy, cancer, marasmus, and in all conditions attended by extreme prostration and a feeble state of the circulation. It is usually a grave symptom in such conditions, and significant of profound debility. The treatment must have regard to the primary condition, and will be only too often ineffectual.

DIAGNOSIS OF HYDROTHORAX.—The physical signs of hydrothorax are identical with those of pleural effusion, and need not be further dwelt upon. The influence of posture is notably greater in hydrothorax than in inflammatory effusions. On removal of a portion of the fluid it will be found to possess a specific gravity under 1015, to contain a relatively small amount of albumin, little or no fibrin, and few red or white blood-corpuscles.

HÆMOTHORAX.

Ordinary serous effusions contain some red blood corpuscles, but not usually in sufficient numbers to give a red tint to the effusion.

The principal causes of hæmorrhagic effusion are the following—

1. Intense inflammatory effusions in persons of low vitality.
2. Tuberculosis.
3. Cancer.
4. Pulmonary gangrene.
5. Granular kidney, scurvy, hæmophilia, cirrhosis of the liver, certain of the infectious fevers, puerperal fever.
6. Traumatism.

The cases of hæmorrhagic effusion (excluding cases where

a serous effusion was merely flecked with blood) which have come under my observation have nearly all belonged to two categories—tubercle or cancer. Hæmorrhagic effusion in tubercular cases has seemed to me to be characteristic rather of low resisting power on the part of the patient than of any special type or stage of the disease. Its presence is often not suspected until revealed by puncture. The blood is usually well mixed with serum. When the character of the effusion is revealed in these cases, it lends confirmation to the suspicion which may have been previously entertained of tubercle, and hence may necessitate a reconsideration of the treatment of the case. If the effusion is large in such cases, it should be tapped like other effusions, but the tapping should be done with more than usual caution, and the operator should be prepared at any moment to suspend the operation if pallor, faintness, or a poor quality of the pulse suggests that fresh bleeding is taking place into the pleural cavity.

In cancerous cases the fact of a hæmorrhagic effusion adds nothing fresh, either from the point of view of prognosis or treatment. It is highly important to remember that in the majority of cases of cancer the effusion is serous and not hæmorrhagic. Tapping in the hæmorrhagic effusions of malignant disease must be governed by general considerations. It is often necessary as a palliative measure.

In the other cases enumerated—pulmonary gangrene, scurvy, granular kidney, cirrhosis of the liver, puerperal fever, etc.—the question of tapping hæmorrhagic effusions must be decided on general grounds. We have to weigh, on the one hand, the probable relief to be gained by the operation, and, on the other, the possibility of further hæmorrhage into the pleural cavity after removal of the effusion.

Chylothorax.

An effusion into the pleural cavity of pure chyle is one of the rarest of events, and practically confined to cases of rupture of the thoracic duct. In a much larger proportion of cases, however, we find a pleural effusion which contains fat and possesses a more or less milky appearance. This type of effusion has often no relation to admixture of chyle, but depends upon fatty degeneration of pus corpuscles and epithelium. In such conditions, chylothorax is a misnomer, but as the differentiation of the two types of fat-containing and milk-like effusion is a matter of extreme difficulty, it is useless to attempt a separate terminology for the two varieties. The presence of any considerable amount of sugar in the effusion would favour the view that there was a genuine admixture of chyle.

Tubercle and cancer are believed to be the two most frequent causes of 'chylous' effusion. How far these causes operate by causing erosion of the thoracic duct, how far by producing fatty degeneration of white corpuscles or epithelium, must be in many cases doubtful.

The prognosis depends on cause, and is, on the whole, decidedly unfavourable. Traumatic cases may be expected to do well in a certain proportion of instances.

Treatment must be on general lines.

LECTURE X

PNEUMONIA—CROUPOUS AND CATARRHAL

SUMMARY:

Croupous pneumonia.
Nosology of the disease.
Bacteriology of pneumonia.
General ætiological factors.
Mode of onset and clinical course.
Symptoms of special diagnostic import.
Physical signs of pneumonia.
Types of pneumonia.
Differential diagnosis of pneumonia.
Complications and sequelæ of pneumonia.
Prognosis of pneumonia.
Treatment of pneumonia.
Broncho-pneumonia:
  Symptoms and course of the disease.
  Differential diagnosis of broncho-pneumonia.
  Treatment of broncho-pneumonia.
Primary congestion of the lungs.
  Maladie de Woillez.
  Views of French clinicians.

PNEUMONIA.

Pneumonia, understood in the most general sense, connotes all inflammatory affections of the proper parenchyma of the lungs. The most general pathological feature of these cases is the presence in the air cells of an inflammatory exudate, which interferes with the free entrance of air.

When the exudation involves a lobe or a large part of a lobe we speak of lobar pneumonia; when only small portions of the lung substance are affected we speak of lobular pneumonia. When the exudation is mainly fibrinous, we speak of croupous pneumonia; when it consists of mucoid and cellular elements, we speak of catarrhal pneumonia; when it is mainly pus, of a septic pneumonia. When the inflammatory processes involve mainly the interstitial connective tissue of the lungs, we speak of an interstitial pneumonia.

Pneumonia may be a primary condition, or it may be secondary to other affections—*e.g.*, bronchitis, pulmonary tuberculosis, influenza, one of the specific fevers, and many other conditions.

We shall first deal with acute croupous or lobar pneumonia, and subsequently with the other varieties.

Acute Croupous (Lobar or Fibrinous) Pneumonia.—Acute croupous pneumonia is one of the most remarkable, most frequent, and most fatal diseases in the range of medicine. It has been known from the earliest times, but it was only when modern methods of physical examination—percussion and auscultation—came into vogue that it could be distinguished with certainty from pleurisy. The peripneumonia of the older writers included both pneumonia and pleurisy, and the distinction between the two diseases was at one time impossible. Pneumonia accounts for about 3 per cent. of all diseases in man. It comes next to tuberculosis as the most frequent cause of death; the mortality varying from 1 to 2 per 1,000 living. The number of cases shows considerable fluctuations, being abnormally high, for example, in 'influenza' years. According to Lenhartz, pneumonia accounts for 6·6 per cent. of all deaths.* Osler states

* Lenhartz in Ebstein and Schwalbe's 'Handbuch der Praktischen Medizin,' Bd. i. 1, p. 228.

that the deaths from pneumonia in some of the leading American cities, such as New York and Chicago, vary from 14 to 19 per cent. of deaths from all causes.

Pneumonia is a disease deserving of the closest study, because the difficulties of diagnosis are sometimes great, and errors are frequently made; because the course of the disease and the tendency to spontaneous cure involve the possibility of many therapeutic fallacies; because there is reason to fear that certain lines of treatment, founded upon erroneous pathological assumptions, have been worse than useless. Pneumonia has been one of the great battle-grounds of physic for centuries, and it is somewhat melancholy to note how often the advocates of depletion or of stimulation have boldly founded their practice on the most slender and questionable of data, instead of humbly observing the course of nature, and seeking to intervene in order to supplement the *vis medicatrix naturæ*. No disease has been oftener the sport of confident theories; in none, perhaps, has treatment availed less or been more frequently perverse and ill-judged.

The nosological position of pneumonia has been difficult to establish. Since Laennec's time the morbid anatomy of the affection has been well understood, and until recent years little doubt has existed in the minds of physicians that the disease was a genuine fibrinous inflammation of the pulmonary parenchyma. But, since the dependence of the disease on micro-organisms has become probable, there has been a tendency to regard pneumonia as a general infective disease, of which a pulmonary inflammation—a pulmonitis—is simply the local expression. Some leading writers, indeed, go so far as to remove pneumonia from the category of pulmonary diseases altogether, and include it amongst specific infectious diseases. Now, it must be freely admitted that the classification of disease is still in a very rudimentary state, and that we must always be prepared to amend or remodel

our classifications, as the gaps in our knowledge are gradually filled up. The question at present at issue as regards pneumonia is this: Is it more fruitful and more helpful on the whole to study pneumonia as a variety of pulmonary disease or as a disease of the entire organism in which pulmonary symptoms happen to be usually prominent?—as, for example, bowel complications are common in typhoid fever, though no one proposes to describe typhoid fever as a local disease of the intestines. V. Jürgensen was the first to describe pneumonia as a general disease, though he was unacquainted with its bacteriology, and this point of view has been adopted by many subsequent authorities, such as Osler, who describes pneumonia amongst the specific infectious diseases.

In favour of this view it is urged that the cause of pneumonia is almost certainly a micro-organism, the diplococcus of Fränkel being the organism present in the great majority of cases; that the constitutional symptoms of the disease cannot be explained by the local lesion; that remedies directed to the lungs are for the most part futile. That these propositions are sound in the strict scientific or academic sense may be admitted, but it is less clear that the conclusion drawn from them is valuable or practically convenient. To describe pneumonia among the infectious diseases is to lay stress upon the point in its clinical history which is the most doubtful of all. Probably most practitioners would agree that they had never seen a clear case of infection, apart from those cases of epidemic pneumonia which probably belong to a special category. Nor can it be truly said that the relation between the local lesion in the lungs and the constitutional manifestations of the disease is vague or ill defined. Of pneumonia without pulmonary lesion, if such a condition exists (which I think highly improbable), we know nothing. The amount of lung involved usually bears a very distinct relation to the severity of the attack. Double pneumonia is notably more severe and more fatal than single. The char-

acter of the sputum frequently affords a most valuable index of the type of the attack and the progress of the case.

The nosology of pneumonia thus remains a controverted question, but I incline to the view that we gain nothing in clearness of thinking or in assistance either as regards diagnosis or treatment, by separating pneumonia from other diseases of the lungs and transferring it to the class of general infective diseases. It would seem also that some of our time-honoured distinctions as regards the types of the disease are of very doubtful validity. Lobar pneumonia is not strictly synonymous with croupous pneumonia, nor is lobular pneumonia synonymous with catarrhal pneumonia. The pneumonia following measles, for example, is always catarrhal in type, but may be either lobar or lobular in distribution.* Perhaps the same might be affirmed of the pneumonia which so often complicates influenza. As proof of this proposition we have only to look to the enormous inflammatory hyperæmia of the smallest vessels and capillaries of the finest bronchioles which is present in such cases.† Even when the pulmonary lesion is lobar in extent, important differences are found on closer examination, some cases exhibiting the characteristic granular appearance, others rather a smooth-surfaced splenization.

Bacteriology of Pneumonia.—This subject has been worked out by Klebs, Ebert, Koch, Leyden, Friedländer, Fränkel, Weichselbaum, Washbourne, Gamalia, and others. The results obtained are highly interesting and important, but not yet final.

The two organisms to which pneumonia has been attributed are the pneumo-bacillus of Friedländer and the *Diplococcus lanceolatus* of Fränkel. As regards the former, its presence can be demonstrated in only a minority of cases

* Aufrecht, 'Nothnagel's System of Practical Medicine,' English edition, article 'Pneumonia.'

† *Ibid.*

of croupous pneumonia, and the results of experiments on animals have been quite indecisive. Fränkel's diplococcus is present in the large majority of cases of croupous pneumonia. Weichselbaum found it in 94 out of 129 cases. Injected into the rabbit this organism gives rise to virulent inflammation, the blood being filled with characteristic diplococci. If the injection be made through the wall of the thorax, a pleurisy, a fibrinous pericarditis, or occasionally a lobar pneumonia, is the result.* The diplococcus of Fränkel is not confined to pneumonia, but is found in pleurisy, pericarditis, peritonitis, endocarditis, otitis media, meningitis, and even in the mouth and bronchial secretions of healthy individuals.

Other organisms have been found in cases of croupous pneumonia—*e.g.*, the *Streptococcus pyogenes*, the staphylococcus, the *Bacillus typhosus*, the *Bacillus diphtheriæ*, and the influenza bacillus.

The limits of these lectures do not permit an exhaustive discussion of the questions arising out of the pathology of pneumonia. The practitioner would do well to accept at least two conclusions—viz., that the disease is due to a micro-organism or micro-organisms, and that the distinctions between the different types of the disease have only a provisional validity and are not final.

General Ætiological Factors.—The most certain fact in this connection is the influence of season and of cold, variable, and windy weather. Pneumonia is notably most frequent in the first quarter of the year, attaining its maximum in March, and remaining comparatively frequent until the end of May, when there is a rapid decline, the minimum being reached in August. There is a gradual but slight increase during September and October. This increase becomes more marked, but still moderate, during November and December, while in January and February the rate rapidly

* Aufrecht, *loc. cit.*

rises, attaining its maximum in March. Hirsch believes that while spring is the pneumonia season *par excellence* in the higher latitudes (Russia, Sweden, Denmark, Germany, England, Northern States of America), the disease attains its maximum in winter in the warmer and subtropical regions (Italy, Mediterranean Islands, Greece, Algiers, Southern States of America, Chili, Peru).* This statement would tend to support the view of the importance of weather in the causation of pneumonia, as in the higher latitudes spring is especially the season of variable weather and sharp alternations of temperature and humidity, while in more southern regions it is the winter which affords these conditions, the spring being early and on the whole genial.

Pneumonia not uncommonly follows upon a definite exposure or wetting. This was the case in eleven out of forty-one cases under my care in the Royal Victoria Hospital during the last few years. Exposure or a wetting seems to be much more effectual as a predisposing cause to pneumonia if combined with unusual muscular exertion or prolonged fatigue. The disease is relatively very common among young recruits during their first year of service.

A serious discrepancy exists amongst some of the best observers as to whether pneumonia is more common amongst those confined indoors or those who spend much of their time in the fresh air. V. Jürgensen is of opinion that the disease is commoner amongst indoor workers, while the contrary opinion is held by Fräntzel, Aufrecht, and others. There is no doubt that pneumonia is relatively frequent in prisons, barracks, asylums, and similar institutions; but there is some doubt whether the form of pneumonia which prevails under these conditions, and which sometimes assumes epidemic proportions, is identical with the ordinary sporadic pneumonia.

It is not clear that occupation, as such, has much influence

* Hirsch, 'Geographical Pathology,' vol. iii., p. 95.

upon the incidence of pneumonia. It does not appear to be relatively frequent in farmers, gardeners, or agricultural labourers. It is when to an outdoor life are added the elements of undue exposure, heavy work, hardship, and intemperance that the pneumonia rate seems to rise. It does not appear, however, that the influence of alcoholism is very marked as a predisposing cause, though it undoubtedly has much influence upon the mortality from the disease.

Pneumonia is much more frequent in the male than the female sex, a fact probably explicable by the greater liability to exposure, hardship, and unfavourable conditions of life and labour on the part of men. This contrast between the sexes is not seen in infancy and early childhood, when the conditions of life of the two sexes are in the main similar, but after the age of fifteen, when the male sex becomes more liable to those conditions of life which tend to increase the susceptibility to pneumonia—viz., over-exertion, over-heating, vicissitudes of atmospheric pressure, wet, cold, intemperance, and injury.*

I have not seen anything in my own practice to lead me to attach much importance to traumatism as a cause of pneumonia; but Litten, Stern, and others have emphasized this cause, and have attributed to it from 2 to 4 per cent. of cases of pneumonia. My series does not contain any certain example of traumatic pneumonia.

Mode of Onset and Clinical Course.—In a large majority of cases the onset of pneumonia is sudden, and occurs in the midst of apparently good health. In an important minority of cases, however, the onset is spread over three or four days, and want of attention to this fact is one source of errors in diagnosis. As a rule, however, the first intimation of the disease is a severe and prolonged rigor, accompanied by intense headache, nausea, and vomiting, a severe pain in the side, embarrassment of the breathing, a rapid rise of tempera-

* J. W. Moore, 'Encyclopædia Medica,' article 'Pneumonia.'

ture, and marked prostration. The patient is at once gravely ill, and usually takes to his bed in a few hours. In this respect pneumonia differs from pleurisy, in which disease prostration is rarely profound and is never an early symptom. The mental condition in commencing pneumonia is usually abnormal. Some confusion of mind is usual; delirium may speedily develop, and is sometimes violent. A hard, dry cough makes its appearance, and owing to the pain which it causes, the cough is suppressed as much as possible by the patient. In a short time—often within twenty-four hours—the attack matures, and all the characteristic symptoms make their appearance. The patient lies like a log in bed, usually on his back. His eyes are bright, a flush often is present on the malar bones, and herpes often shows itself about the angles of the mouth. The expression is one of pain and anxiety. The breathing is rapid (40 to the minute, or more), shallow, painful, and ineffectual. The alæ nasi often work vigorously, a sign which is apt to be specially well marked in children. The skin has a pungent heat, which some observers regard as highly characteristic. The actual temperature may vary from 102° to 105°, the higher range being common in severe cases. There is a dry, hacking cough, either without expectoration or accompanied by a scanty, viscid, glue-like sputum, which soon takes on the well-known and pathognomonic rust-colour, owing to admixture of some of the colouring matters of the blood. The pulse is accelerated, but in a less degree than the respirations, and is full and bounding in character; later on it may intermit. Mental confusion or actual delirium, usually worse at night, may be present. Sleep is much impaired, and may be entirely wanting. The urine is febrile, loaded with urates, often albuminous, while chlorides are reduced or absent. The bowels are usually confined.

On examination we may either get the typical physical signs of pneumonia—viz., the so-called ‘ primary crepitus ’—

soon followed by the signs of consolidation—viz., dulness on percussion, bronchial or tubular breathing, and bronchophony—but in a considerable number of cases the physical signs are latent or quite absent, even when the symptoms are fully developed and pathognomonic. We shall return to this point in dealing with difficulties of diagnosis.

The above symptoms frequently last for from two or three to seven or eight days without material change. The prostration usually deepens; the pulse gets softer, more frequent, and occasionally intermittent; a tinge of jaundice is sometimes seen on the skin; the sputum becomes more abundant, less viscid, less definitely rust-coloured, more mucoid in type, or in bad cases becomes dark and fluid, or ceases altogether; the delirium is slight in favourable cases, and gets more pronounced in severe cases; the dyspnœa continues marked, and the *besoin de respirer* is pronounced. There is total anorexia. Thirst is often marked. Sleep continues much disturbed.

In a few days, not often before the fifth day, and rarely later than the ninth or tenth (most often about the seventh day), a definite crisis takes place. The temperature falls to normal either by a single drop in a few hours, or not uncommonly by two stages—viz., a drop from 104° or 105° to about 100°, followed by a slight rise to 101° or 102°, and then a second fall to normal, the entire crisis occupying from twenty-four to forty-eight hours. The distress of breathing is at once relieved, the patient breaks out into an abundant perspiration, and often falls into a quiet sleep, from which he wakes up much refreshed, and with the conviction that the worst is over. Improvement is now usually rapid. The temperature remains normal or shows trivial oscillations, unless in exceptional cases where a fresh invasion of lung takes place or empyema develops; the appetite begins to return; the albumin disappears from the urine; sleep is restored; and in a few days the patient feels almost well,

although incapable of exertion. Convalescence is usually uninterrupted, and in most cases is complete in from three to four weeks. Sequelæ of any kind are quite rare. The general health after an attack is usually in no way impaired, but second, and even third or fourth, attacks are by no means uncommon.

Symptoms of Special Diagnostic Import.—The symptom-complex of pneumonia is a highly characteristic one. In a typical case error is hardly possible. The points of most diagnostic importance are the following:

(*a*) The sudden onset, with rigor and rapidly developing prostration.

(*b*) The dyspnœa and altered pulse-respiration rate.

(*c*) The sputum.

(*d*) The course of the temperature.

(*e*) The prominence of nervous symptoms.

(*f*) The condition of the urine.

(*g*) The rapid and usually uninterrupted convalescence.

The onset in a typical case—*i.e.*, the combination of severe rigor, rapid rise of temperature, quickly developed and intense dyspnœa, and extreme prostration—is unlike that of any other disease. In typhus and relapsing fever the element of severe dyspnœa is wanting at the outset. In meningitis the onset is less sudden, the temperature usually lower, and the breathing is not specially involved. In pleurisy the temperature is lower, and there is much less prostration.

The character of the dyspnœa and the altered pulse-respiration rate of pneumonia are points of cardinal importance. The dyspnœa is usually intense, and the respirations increased to 35, 40, 45, or 50 in the minute. They may be as much as 80 or 90 in young children. The patient visibly struggles for breath, he hungers for air, and often seems as if he had no time and no strength to do anything but breathe. When he takes a draught of water or a mouthful of milk, he

pauses in the act in order to gasp for air. The pulse is relatively only moderately accelerated, and may be only 80, 90, or 100 per minute. In severe cases, however, it runs up to 110, 120, or higher, and this increase has usually a grave prognostic significance.

The sputum is highly characteristic, and often settles the diagnosis at a glance. It is scanty at the outset, very viscid owing to the presence of large quantities of nuclein, and more or less rust-coloured from the presence of colouring material from the blood. As the case progresses, the sputum usually becomes more abundant, less viscid, lighter in colour, and more mucoid in character. Sometimes it becomes abundant, watery, and dark in colour—the so-called 'prune-juice' sputum, which probably indicates the presence of a spreading congestion (not a true consolidation) of the lungs, and is of unfavourable import. In rare cases the sputum is yellowish or greenish in tint, even in the absence of jaundice. A few cases of pneumonia run their entire course without sputum, and this feature does not seem to be of unfavourable significance. The cessation of a previously abundant sputum often points to failing strength, and may presage the end.

The course of the temperature is one of the most characteristic features of pneumonia, and in typical cases the pneumonic chart cannot be mistaken. The sudden rise; the persistence for several days of a severe pyrexia, with slight daily oscillations; the well-marked crisis, followed frequently by a subnormal temperature for some days; and the usual absence of any secondary rise, are typical of the disease. But departures from the normal are by no means rare. The rise is sometimes by stages, a primary rise to 100° or 101° being followed in two or three days by a secondary rise to 104° or 105°. These cases are apt to mislead the unwary practitioner. It is probable that in such cases there is at first a slight or moderate implication of the lung, followed after a short interval by a further

invasion of the pulmonary parenchyma. In certain cases, especially in cases of privation or alcoholism, and in the aged, the thermometer may give little warning of the presence of pneumonia. In one case in my experience (not included in my series of forty-one) the temperature in a young alcoholic subject remained normal until within a few hours of death. The crisis may be delayed or even absent. In two out of my series of forty-one cases defervescence was by lysis. In another remarkable case in my experience (not included in the series) the crisis did not occur until the twentieth day in a young child, the explanation being that there were progressive invasions of different areas of the lungs before the crisis belonging to the first invasion had time to mature.

The rapid and uninterrupted convalescence is, on the whole, highly characteristic of pneumonia, but obviously is seldom helpful to us in diagnosis. In no disease, except perhaps typhus fever, do we feel less apprehension of complications arising after once the crisis has been fully surmounted. Tuberculosis is believed by some authorities to be an occasional sequela, but this must be a very rare event. We shall return to this question subsequently.

The character of the urine—scanty, high-coloured, acid, loaded with lithates, albuminous, and wanting in chlorides —possesses considerable diagnostic importance. Albumin was noted in fourteen out of my series of forty-one cases. Hæmaturia seems to be quite rare. There was no example of this symptom in my series.

I have not laid stress upon the presence of *herpes labialis*, because this condition is common in mere catarrhal affections, but in association with sharp pyrexia and marked dyspnœa herpes would certainly strengthen the suspicion of pneumonia. An icteric tinge of the skin and sclerotic is comparatively common in pneumonia, and should be allowed some weight in the diagnosis of a doubtful case.

Some affection of the sensorium is very frequent in pneumonia—much more frequent, for example, than in pleurisy—and may be so prominent that the question of cerebral involvement arises. I have seen a good many cases where pneumonia has been mistaken for meningitis, owing to the prominence of cerebral symptoms. It is a good clinical rule, when cerebral symptoms suddenly develop with high fever, to think of the possibility of pneumonia. A glance at the alæ nasi will sometimes give the clue to the correct diagnosis.

THE PHYSICAL SIGNS OF PNEUMONIA.—These are well known, and may be only briefly adverted to here. It is customary to describe them in relation to the three classic stages of the disease—viz., the stage of engorgement, the stage of red hepatization, the stage of grey hepatization.

The typical physical sign of the first stage is the so-called true pneumonic crepitus, generally regarded as due to the opening up of collapsed air cells, or of air cells of which the walls have been glued together by fibrinous exudate, but thought by some authorities to be of pleural origin. At this stage the percussion sound may be normal or unduly clear, or even tympanitic in places. The breath sounds are weak or harsh. The vocal fremitus is increased.

The typical physical signs of the second stage are dulness on percussion, increased vocal fremitus, bronchial or tubular breathing, and bronchophony. The dulness on percussion is less wooden than in pleural effusion, and gets less pronounced as we percuss towards the edges of the affected area. Increased vocal fremitus, bronchial breathing, and bronchophony are present *provided the main bronchi remain open*, but if these should be blocked by secretion we find that vocal fremitus is diminished, the breath sounds weak or absent, and vocal resonance feeble. When the consolidation is not very compact the breathing may be vaguely blowing instead of bronchial, while tubular breathing connotes a very dense form of con-

solidation. The heart sounds are intensified over the area of solidified lung for two reasons—first, the absence of the normal vesicular breathing throws them into relief; secondly, the solid lung acts as a conductor.*

The physical signs of the third stage are partly those of still-persisting consolidation, partly those pointing to the re-entry of air into the affected lobules—viz., a form of crepitation, coarser and 'moister' than the primary crepitus. Like the latter, this 'crepitus redux' is chiefly heard towards the end of inspiration. At this stage the percussion note sometimes takes on a tympanitic quality, probably the 'relaxed lung note' previously adverted to. During the third stage of pneumonia the physical signs of consolidation often disappear very rapidly. Tubular or bronchial breathing is replaced by vaguely blowing or faintly bronchial breathing; this in turn by weak breathing, perhaps with a slightly harsh quality, and ultimately true vesicular breathing is restored.

According to Leube, in those cases of deep-seated pneumonia when physical signs almost wholly fail us, it is possible to locate the disease by careful auscultation of the voice, a commencing bronchophony being audible over the affected area.† He adds that in such cases the first signs of pneumonia are frequently recognisable in the axilla.

Varieties in Types of Pneumonia.—It is perhaps well to be chary of erecting varieties of pneumonia which present one or more prominent symptoms into distinct 'types' of the disease. Such classifications possess little practical value. We may, however, recognise in a very provisional way such varieties as the 'gastric' type; the 'hepatic' type; the 'cerebral' type; the 'larval,' 'masked,' or 'ephemeral' type. In the gastric type the symptoms are predominantly gastro-intestinal, and these cases have now and again been most unhappily mistaken for appendicitis or perforating gastric

* J. W. Moore, *loc. cit.*

† Leube, 'Diagnose der Innern Krankheiten,' vol. i., p. 138.

ulcer. Such an error would hardly be possible if the thorax were carefully explored, the character of the breathing observed, and the temperature noted. In the hepatic form jaundice and pain over the liver are the prominent features, and a diagnosis of 'congestion of the liver' or of gall-stones has sometimes been given in such cases by observers who forgot that jaundice and pain over the liver are common in right-sided pneumonia, and who omitted to explore the chest thoroughly. Acute hæmorrhagic pancreatitis has been wrongly diagnosed for pneumonia.

The cerebral type is quite common, and is the cause of many unfortunate errors of diagnosis. Violent delirium, headache, and in children convulsions, are the prominent features of such cases. A diagnosis of meningitis has often been given in such cases, but meningitis does not set in with the startling suddenness of pneumonia; the temperature is lower, the respirations are only moderately increased in frequency, the pulse is sometimes irregular, there is no characteristic cough or sputum, and, of course, there are no signs in the chest.

The larval, masked, or ephemeral type of pneumonia is not uncommon. The patient presents the initial symptoms of pneumonia, but in moderate degree. Slight implication of the lung can be recognised, but the attack aborts in forty-eight hours or less, and the patient rapidly convalesces. Sir J. W. Moore relates an interesting case of this kind where there could be no doubt of the diagnosis.* I have often suspected this condition at the bedside, but from the nature of the case a positive diagnosis is rarely possible. Some of the cases of 'smart, feverish colds,' with pain in the chest, some degree of dyspnœa and labial herpes, probably belong to this category. If a diagnosis is not at first possible, we are not left long in doubt. Should the case be one of larval or ephemeral pneumonia a rapid subsidence of all the

* J. W. Moore, *loc. cit.*

symptoms in two or three days may be expected. A 'one-day pneumonia' has been described by Leube, Weil, Fischl, and Bernhard. The so-called 'febris ephemera' is probably often pneumonia. Our suspicions may be aroused by an undue frequency of the respirations, by 'stitch in the side,' slight bronchophony, or a few crepitant râles. The percussion sounds may be quite uncharacteristic, and bronchial breathing is rare. Steiner found 23 cases of ephemeral pneumonia out of 1,157 cases in the medical clinic at Leipzig.* V. Jürgensen found the crisis in the second day in 6 cases out of 933. In the Würzburg clinic 10 cases of one-day pneumonia were recognised out of a total of 1,057 cases. It is noteworthy that in these cases the initial stage is fairly characteristic in point of symptoms, which may be severe, but that the physical signs are somewhat indefinite and the sputum rarely possesses the typical characters.

The interpretation of these interesting cases is not altogether plain. Does the pneumonic process go through all its stages in an unusually brief space of time, or come to an arrest at a certain point? Is the short duration of the case due to a rapid development of antitoxin, or to the slight virulence of the infection? We can hardly answer these questions with any confidence in the present state of our knowledge.

A 'migratory' type of pneumonia is described in which the pneumonic process successively involves one lobe after another.

Diagnosis of Pneumonia.—Many of the problems of diagnosis have already incidentally engaged our attention, but the subject is worthy of some further amplification. Difficulties arise mainly in the following ways:

(*a*) Because the mode of onset is untypical.

(*b*) Because cerebral, gastric, or hepatic symptoms overshadow the proper pulmonary symptoms.

* *Deutsch Archiv für Klin. Med.*, 64, 1899.

(*c*) Because the physical signs are for a time latent.

(*d*) Because the physical signs leave us in doubt as between pleurisy and pneumonia.

(*e*) Because some condition—*e.g.*, tuberculosis—has preceded the attack of pneumonia.

(*f*) Because some co-existing condition—*e g.*, extreme privation, insanity, alcoholism, delirium tremens—modifies in some striking manner the usual symptom-complex.

Perhaps the most helpful way of regarding this subject will be to consider the actual errors of diagnosis, which experience shows to be somewhat frequent in connection with pneumonia. In my experience the diseases most often confused with pneumonia are pleurisy, typhoid fever, meningitis, and certain forms of phthisis; while less frequently typhus fever, delirium tremens, hepatic affections, appendicitis, and gastric ulcer are the sources of error. We shall consider these conditions in order.

Pleurisy.—The differential diagnosis between pleurisy and pneumonia has already been fully considered.* The most helpful points are the following:

1. The mode of onset.
2. The sputum.
3. The pulse-respiration rate.
4. The quality of the dulness and the outline of the dull area.
5. The state of the vocal fremitus.
6. The displacement of organs.
7. The results of puncture.

Typhoid Fever.—Mistakes between pneumonia and typhoid fever are frequent, as is proved by the presence in medical literature of the term 'typhoid pneumonia,' a condition which has no existence. We may have typhoid fever with pneumonia as a complication, or pneumonia with the so-called 'typhoid' symptoms, but never a hybrid of the two

* See p. 151 *et seq.*

diseases, although, of course, the two diseases may chance to co-exist. The confusion between pneumonia and typhoid fever arises in one of three ways—viz., by forgetfulness of the facts (1) that asthenic pneumonia has a gradual and more or less typhoid-like onset; (2) that typhoid fever may come on with symptoms which, at the outset, are predominantly pulmonary; (3) that pneumonia may be accompanied by symptoms which are somewhat unhappily denominated 'the typhoid state.' In any case of presumed typhoid fever or pneumonia, where the signs or symptoms afford room for doubt, the alternative diagnosis should be carefully inquired into in the following ways:

1. By a careful search for the characteristic physical signs (pulmonary signs, enlarged spleen, spots) of the two conditions.

2. By attention to the state of the breathing and the pulse-respiration rate—probably decidedly affected in pneumonia, less so in typhoid fever.

3. By attention to the state of the abdomen, probably more or less distended in typhoid fever, normal in pneumonia.

4. By attention to the state of the pulse; probably dicrotic in typhoid fever, not dicrotic in pneumonia.

5. By attention to the state of the tongue; probably thickly coated on dorsum, and red or raw-looking at tip and edges in typhoid fever; less characteristic in pneumonia.

6. By attention to the characters of the urine; albumin and absence of chlorides more probable in pneumonia.

7. By examination of the blood, a marked leucocytosis (15,000 to 50,000 white cells) being decidedly in favour of pneumonia, and as decidedly against typhoid fever.

8. By a careful analysis of the temperature chart, which will probably suggest some points of contrast.

Meningitis.—The confusion between meningitis and pneumonia, especially in the case of young children, is one of the commonest mistakes in practice. It arises from neglect of

the well-known fact that pneumonia—especially in young patients—may set in with violent delirium or convulsions. Error may be avoided by attention to the following points:

1. The onset in pneumonia is often absolutely sudden; in meningitis there are always some premonitory symptoms, usually extending over several days.

2. Pneumonia ushered in by severe cerebral symptoms will almost certainly exhibit a very high temperature—104° or 105°; in meningitis the temperature is lower.

3. A marked acceleration of the respirations is invariable in pneumonia; improbable in meningitis.

4. Retraction of the head and squint are frequent in meningitis; very rare, but not unknown, in pneumonia.

5. Vomiting may occur in either condition at the outset, but, if persistent, is suggestive of meningitis.

6. The abdomen is often retracted in meningitis; seldom or never in pneumonia.

7. Kernig's sign is frequent in meningitis; never present in pneumonia.

8. Early irregularity of the pulse is suggestive of meningitis.

9. Cough is usually irritating and persistent in pneumonia; often absent and rarely prominent in meningitis.

Certain Forms of Phthisis.—Confusion between pneumonia and phthisis is unusual, but may arise in the following ways:

1. Pulmonary tuberculosis may assume the form of acute pneumonic phthisis, characterized by sudden onset, rapid consolidation of an entire lobe, rusty sputum, and high temperature. For a time it may be quite impossible to differentiate these cases from croupous pneumonia, more especially as at the outset tubercle bacilli are often absent from the sputum. But in a few days our suspicions begin to be aroused. The attack becomes 'asthenic' in type. The patient is very prostrate, and perspires profusely. The pulse increases *pari passu* with the respirations, which is not the case

in croupous pneumonia. There is either no crisis, or an imperfect one, or there is a post-critical rise of temperature and a tendency for the chart to assume the hectic type. The patient soon begins to emaciate. After a time tubercle bacilli are found in the sputum.

2. A patient with slight quiescent phthisis may develop an ordinary attack of croupous pneumonia, which may run its usual course, and end either in death or complete recovery, or in re-awakened activity of the previously dormant tuberculosis. With care, such cases should not present any special difficulty in diagnosis.

3. Croupous pneumonia may be the starting-point of pulmonary tuberculosis in a patient not previously tubercular. This is probably a very rare event. In only one out of my series of forty-one were tubercle bacilli found in the sputum after the pneumonic attack, and it is doubtful whether in this case the patient was in normal health before the attack.

As to the conditions occasionally, but less frequently than the above, confused with pneumonia—viz., delirium tremens, typhus fever, hepatic affections, appendicitis, etc.—it is not necessary to enter into great detail. An error in such cases will, as a rule, be easily avoided if only the possibility of error is sufficiently weighed. In *delirium tremens* the state of the temperature and the character of the breathing present no analogy with pneumonia. In *typhus fever* the pulmonary symptoms and signs are slight or absent, and the rash soon makes error impossible. *Hepatic affections*—perihepatitis, gall-stones, etc.—will cause little difficulty if the possible hepatic complications of pneumonia are kept in view. *Appendicitis* will mislead only a very unwary or very careless diagnostician. It is important, however, that the bare possibility of such conditions giving rise to error should be allowed to pass through our minds in any case of presumed pneumonia where the signs or symptoms are obscure or ambiguous.

COMPLICATIONS AND SEQUELÆ OF PNEUMONIA.—Pleurisy is the most frequent complication of pneumonia, and is present in some degree in nearly all cases. Exceptionally, an effusion sets in, which is frequently purulent from the outset. General bronchitis sometimes develops, and, if pronounced, is of evil prognostic augury. Œdema of the lungs is frequent and sometimes grave. Pericarditis is an occasional complication, and one of the gravest. In my experience it has generally ended fatally. Endocarditis is also an occasional and very grave complication—above all, if it assumes the form of ulcerative endocarditis. Albuminuria is frequent, but has not much prognostic import. It very rarely leads to acute nephritis. Cerebral complications are common, especially in children, in debilitated persons, and in the alcoholic. Meningitis occasionally develops, and always ends fatally. Jaundice is frequent, and not of much prognostic importance. The motions continue to show the presence of bile pigment. Herpes labialis is a common complication, and seems to be more frequent in favourable cases.

The most important sequelæ of pneumonia are chronic interstitial pneumonia, gangrene of the lung, abscess of the lung, tuberculosis, and venous thrombosis. Gangrene and abscess will subsequently engage our attention. On the whole, pneumonia is remarkably free from sequelæ. Recrudescences of the disease in its course are not infrequent, but genuine relapses are very rare. Second attacks are frequent.

PROGNOSIS OF PNEUMONIA.—The average mortality in cases of pneumonia is difficult to determine. Hospital statistics are, for various reasons, of only limited value. The average mortality for England and Wales is reckoned at 14·5 per cent.* In America the rate would seem to be much higher—viz., 20 to 30 per cent.† Lenhartz states that in Germany the ordinary typical disease has an average mortality of only 3 to 5 per cent, while in the asthenic form the rate is

* J. W. Moore and others.

† Osler.

20 to 30 per cent., or even higher.* Of the forty-one cases in my series, death took place in four—*i.e.*, in 10 per cent. of cases. The mortality in cases seen in consultation practice is, for obvious reasons, very high. The mortality varies within very wide limits according to age, habits, and constitutional condition of the patient, complicating conditions—*e.g.*, influenza, type of the disease, and season. In the ordinary sthenic pneumonia of adults the mortality cannot be less than 20 or 25 per cent. In children the outlook is usually quite favourable. Children die of catarrhal, rather than of croupous, pneumonia.

The prognosis of pneumonia is chiefly conditioned by the following considerations—viz., the age of the patient, the constitutional condition and previous habits of the patient, the type of the disease, the extent of lung involved, the presence of complications, epidemic type. Age is the most important point of all. In infants the mortality is believed to be high, but from three to fifteen the prognosis is usually very favourable. The mortality between these ages varies from 5 to 10 per cent. After fifteen the mortality steadily rises with age, until at middle life it varies from 30 to 40 per cent. Over 70 the mortality is about 60 per cent. A history of privation, starvation, alcoholism, or other form of excess makes the prognosis much more grave than where these factors are absent.

The type of the disease is a point of much importance. The sthenic type is relatively favourable, the asthenic or 'typhoid' type decidedly unfavourable. The 'bilious' type does not seem to have much prognostic import. A low temperature with much dyspnœa and a frequent weak pulse is highly unfavourable.

The extent of lung involved has much influence, but this rule would seem to have some important exceptions. It must be remembered that our method of examination may

* Lenhartz, *op. cit.*, p. 246.

quite mislead us as to the amount of lung involved. Double pneumonia is notably more fatal than single. The most serious complications are chronic nephritis, diabetes, chronic endocarditis, phthisis, emphysema, pregnancy.

Epidemic type counts for something. Pneumonia is notably more fatal in some seasons than in others. The possible influence of influenza must be weighed.

Unfavourable symptoms are the following: Marked dyspnœa; orthopnœa; much cyanosis; pulse over 120 (except in young patients); early signs of nervous weakness; a 'prune-juice' or diffluent puriform expectoration; hæmoptysis; sudden cessation of sputum; hyperpyrexia.

Previous attacks have no weight in prognosis. Signs of cardiac weakness are always ominous in pneumonia. A persistent quickening of the pulse, irregularity or intermittency of the pulse, cyanosis, gradually spreading pulmonary œdema, are all significant of approaching heart failure. The character of the cardiac sounds in such cases is a point of much interest. The first sound does not necessarily become weak and indistinct, but often sharp, short, and high pitched. I had recently an opportunity of listening to the heart sounds in a lady aged seventy-four who was dying of pneumonia. The first sound as heard in the mitral area was louder than normal, very sharp, short, and high in pitch. The patient was fully conscious, and not cyanosed. The pulse was 140 and irregular, tension very low. Death took place within two hours of the time of my examination.

THE TREATMENT OF PNEUMONIA.—The treatment of pneumonia has been one of the great battle-grounds of medicine. One must speak with bated breath on a subject so controverted, so associated with erroneous practice in the past. There are those who look back regretfully to the days when venesection was in vogue, and who think we have surrendered a valuable therapeutic method. I do not share these regrets.

I have seen venesection employed in a few cases of pneumonia, and I do not desire to see it again recognised as a routine method of treatment. Its dangers are not few; its advantages are problematical; it is open to great abuse in rash or inexperienced hands. That venesection sometimes mitigates symptoms and relieves a labouring right ventricle is, of course, certain; but it is more open to question whether even in such cases it does permanent good. It may be a justifiable procedure in an emergency; it is not well to include it amongst our routine remedies. The pendulum has, however, swung far in the opposite direction. A crass method of over-feeding and indiscriminate stimulation has been too often adopted, without due regard to the patient's peculiarities, his powers of absorption, or the special indications of his case. If the 'Bloody Moloch' has been dethroned, let us beware of putting another idol in his place.

The therapeutic problem in pneumonia may be thus stated: The patient is the subject of an acute, specific, self-limited, febrile disease, which throws a special strain on the heart, but in which the morbid process tends irresistibly to subside, if only the patient survives for a limited time—seven to twelve days. Our aim is to keep the patient alive until the disease has run its stormy but evanescent course. We can do little to shorten the attack or abate its severity. We can do much to enable the patient to withstand it successfully.

The special danger in pneumonia is heart failure. 'Patients who die of pneumonia are killed by cardiac insufficiency,' says V. Jürgensen. Perhaps this view is too exclusive, but it is in the main sound. I have, however, seen a patient gradually sink from pneumonia, the pulse keeping about 80, regular, and of good tension until near the end; but this case is so far isolated in my experience. Œdema of the lungs, depending on weak cardiac action, is usually a prominent feature in fatal cases. Sudden death is comparatively

common. The patient does not, at least in the great majority of cases, die of dyspnœa or asphyxia; he dies in most cases because his heart succumbs to the strain upon it. The determining cause of heart failure in pneumonia is not quite certain. It is not myocarditis, which is not marked in fatal cases; nor is it the severity of the pyrexia, which in some of the worst cases is never high. Probably toxæmia is the usual explanation. Two facts stand out prominently: the patient is engaged in a struggle, usually sharp, always short; and his chief danger is heart failure. The therapeutic indications, then, are clear:

(*a*) To minimize the strain upon the heart.

(*b*) To sustain the strength of the heart.

(*c*) To avoid any method of treatment which, however palliative of other symptoms, might tend to wear out the strength of the heart.

Expanding the above indications somewhat, we may say that, pending the discovery of a specific remedy, the treatment of pneumonia is general and symptomatic, the aim being to promote the patient's comfort, to diminish his sufferings, to conserve nervous and circulatory energy, and to avert the tendency to heart failure.

The attempt to discover a specific serum for pneumonia has so far proved unsuccessful.

Absolute rest is indispensable. The patient should be kept perfectly quiet in a well-aired room at a temperature of about 60° F. He should not receive any visitors, or engage in any general conversation. He should not leave his bed for the purpose of relieving the bladder or rectum. The bedclothes should be light and the clothing loose. Freedom of the respiratory movements should be promoted as much as possible. For the relief of pain and the promotion of sleep I believe the moderate use of morphia in the early days of pneumonia to be both useful and safe. At a later stage its use requires to be very guarded. It should not be given

in the presence of cyanosis or of a weak and rapid pulse. A poultice—light, warm, and frequently renewed—seems generally welcome to the patient, and is apparently comforting. It should not be continued for more than two or three days. Ice to the chest has been recommended, but has not found general acceptance. It seems a question whether it might not in certain cases tend to depress the heart. A febrifuge mixture may be ordered. Aconite and tartar emetic are probably harmful.

The question of antipyresis in pneumonia raises some very important issues, which are not yet definitely settled. On the one hand, we have the facts that a smart pyrexia does not seem unfavourable, and that in some of the worst cases the pyrexia is moderate or low; while, on the other hand, we know that hyperpyrexia is a deadly complication, and general considerations would lead us to apprehend that a continued high temperature might tend towards heart failure. It cannot be said that experience has definitely decided this crucial difficulty. Liebermeister advises the regular use of baths at a temperature of 23° to 25° C., each bath to last about ten minutes, or of tepid baths of a temperature of about 30° C., continued for a longer period. Lenhartz endorses this line of treatment. Most authorities, however, are unconvinced of the special value of such treatment, while inclining to the view that hydrotherapy may be occasionally employed on general principles. I am in the habit of ordering regular tepid or cool sponging, with or without the addition of some fragrant spirit, and I have no doubt it has a favourable effect upon the nervous system, though its influence upon the pyrexia is usually slight. The use of antipyretic drugs seems to me in general contra-indicated. The cardiac depressants, such as antipyrin, antifebrin, and phenacetin, are much more likely to do harm than good. Quinine is lauded enthusiastically by many authorities, especially in Germany, and may have a

trial, though I am not convinced that it possesses any signal value. Aufrecht advises that it should be given in the form of the neutral tannate in 15 grain doses by the mouth, or the hydrochlorate in $7\frac{1}{2}$ grain doses hypodermically.

Of essential value are measures directed to sustain the strength of the heart. The diet should be adequate and sustaining, but not excessive. Much harm is, I believe, done by overfeeding in pneumonia. The patient's powers of digestion and absorption are almost in abeyance, and it is highly undesirable to fill his stomach with food which he cannot digest. Further, an overloaded stomach will press against the diaphragm and embarrass the labouring heart. The amount of nourishment should be moderate and the intervals of feeding not too brief. Milk fulfils the indications best, and is, in most cases, the only necessary food. Three pints in the twenty-four hours will usually be found sufficient. If it seems desirable to supplement this diet, small quantities of freshly-prepared beef-juice, egg flip, or albumin water may be allowed. A cup of tea is harmless and refreshing. All starchy foods should be withheld, as likely to create flatulence. Simple refrigerant drinks may be allowed, care being taken not to distend the stomach. The lavish and indiscriminate administration of excessive quantities of nourishment may lead to acute dilatation of the stomach, and precipitate a fatal issue.

The question of the value of alcohol in pneumonia is an important and much controverted one. We are still far from agreement as to the effects of alcohol upon the activity of the heart and upon the blood-pressure. Alcohol has sometimes been immoderately praised in pneumonia, sometimes condemned as useless or hurtful, sometimes recommended for occasional use, according to the special indications of the individual case. Amongst those who, upon the whole, favour the use of alcohol in pneumonia may be mentioned Germain Sée, V. Jürgensen, Lenhartz, Fagge, and Osler; amongst those

who, upon the whole, disapprove of its use may be mentioned Skoda, Hughes Bennett, Strümpsell, Aufrecht, and J. W. Moore. Most observers are agreed that young patients generally do well without alcohol, and that old patients and the intemperate require it. But the question still remains open, Is alcohol to be recommended in the majority of cases of pneumonia or not? Has it any valid claim to be regarded as a routine remedy? To this question I am disposed to give a negative answer, while inclining to the occasional and moderate use of alcohol, when it is indicated by the state of the pulse and the condition of the nervous system. It may be accepted that in proportion as the symptoms assume the 'adynamic' or 'typhoid' type, the indication for alcohol becomes more decided. With prostration, cyanosis, cold extremities, rapid feeble pulse, probably few would hesitate to give alcohol. My own practice is not to give alcohol if the patient is young, the symptoms mainly 'sthenic,' and the state of the pulse and of the nervous system fairly good. As regards the form and amount of alcohol, I think brandy is, in general, the best form, and that the amount should be from 3 to 4 ounces in twenty-four hours as a minimum, and from 7 to 8 ounces as a maximum. Even so judicious a practitioner as the late Hilton Fagge advised heroic doses of alcohol—as much as 20 ounces in the day—in certain cases of pneumonia. I believe this to be bad practice. It is melancholy to see a patient, as I have more than once seen, actually intoxicated by the medicinal administration of stimulants.

It is generally easily seen whether alcohol is doing good or not in cases of pneumonia. If under its use the patient seems refreshed and his sufferings diminished, if his skin acts more freely and his nervous and circulatory condition improves, we may feel sure that alcohol is doing good. But who has not seen the reverse picture? Who has not seen alcohol given *secundum artem*, although the patient protested

that he felt uncomfortable after its use, that his skin felt hotter than ever, that his breathing was more embarrassed than before, that sleep was not improved?

We must not forget that the effects of alcohol upon the organism in health vary widely. To one person it is an almost pure stimulant, to another an almost pure narcotic. I see no reason for believing that these idiosyncrasies disappear under the stress of acute disease. It is just as reasonable to suppose that they may be intensified under such conditions. Therefore, caution seems advisable in the use of alcohol. In pneumonia, apart from clear indications, it should not be given as a routine measure; its effects should be carefully watched, and we should be prepared to stay our hand if we have just cause for suspecting that it is doing harm.

As regards the value of drugs in obviating heart failure, we find considerable divergence of view. The remedies usually recommended are the following: Digitalis, strophanthus, strychnine, caffein, camphor, ammonia, ether, adrenalin, oxygen. I have made an extensive trial of digitalis in pneumonia, but never in the heroic doses advised by Traube, Fickl, Petrescu, and others. I am inclined to think it possesses a certain value, though its utility is far less certain and manifest than in cardiac disease. G. W. Balfour advises digitalis in drachm doses of the tincture in the pre-critical collapse of pneumonia. We must remember that digitalis is a slow-acting drug, and that if we are to rely upon it in pneumonia we must give it in adequate and quickly repeated doses. Strychnine is a remedy of great value, and is best given hypodermically in doses of 5 to 8 minims of the liquor. Its effect upon the pulse is sometimes at once manifest. Strophanthus, caffein, and camphor may be occasionally invoked to our aid. Ether and ammonia have a certain, but limited, utility. The effect of adrenalin is apparently too transient to be of real service. Much difference of opinion exists regard-

ing the utility of oxygen. After many trials of the remedy I have come to regard it as of doubtful utility, but patients often profess relief from its use, and call for the remedy. It may be given at short intervals for five minutes at a time, and the gas may be warmed with advantage. Expectorants are often given in pneumonia, probably on the basis of a false analogy with bronchitis. They are probably useless. Traube thought that acetate of lead had a good effect when prune-juice expectoration is present.

Special indications for treatment may be present. Œdema of the lungs, one of the gravest of complications, is a call for active stimulation and for leeching. Prostration and toxæmic symptoms may be relieved by saline injections into the rectum or hypodermically. Violent delirium may be combated by ice to the head, the cold pack, saline injections, alcohol, and perhaps in some cases morphia. Chloral should not be given. A spreading 'congestive' lesion is said to be favourably influenced by the tincture of the perchloride of iron. Insomnia is often most intractable. Paraldehyde occasionally acts well and is unobjectionable, but far oftener it fails entirely. Trional and sulphonal may have a trial. Morphia is often our only resource, but after the first three or four days it would appear to be a risky remedy.

Delayed resolution is best combated by counter-irritation and general tonic and hygienic measures. After a suitable interval mountain air is valuable in such cases. Pilocarpine, which has been recommended for this condition, does not seem a very promising remedy. I have not given it a trial.

Broncho-Pneumonia (Catarrhal Pneumonia, Lobular Pneumonia).—Broncho-pneumonia usually develops out of bronchitis, when the bronchial inflammation extends to the air cells, either through the supervention of collapse of the lung, or through aspiration of secretions containing bacteria, or as a complication of one of the acute infective diseases.

The difference between capillary bronchitis and broncho-pneumonia would appear to be simply one of degree; clinically, the distinction is often impossible. It has become more and more probable that in the causation of broncho-pneumonia micro-organisms play a predominating part. Fränkel's pneumococcus is present in at least one-half of the cases. The other micro-organisms which may be present are the *Streptococcus pyogenes*, the *Staphylococcus pyogenes aureus* and *albus*, Friedländer's pneumo-bacillus, and others. The relationship of broncho-pneumonia to the infectious fevers, especially measles, whooping-cough, and diphtheria, is close.

The disease is specially common in children and in the aged; in the former case, probably because the alveolar walls and their epithelium are peculiarly susceptible to irritative and septic influences in childhood; in the latter case, because secretions collect more readily in the bronchial tubes of the old, and are expelled with more difficulty. The lungs are usually both affected, but unequally. Their surface is studded with depressed areas of collapse, while small areas of consolidation project from the surface, and the surrounding lobules often show signs of over-distension. The pleura covering the consolidated areas is usually more or less inflamed.

The physical signs are partly those of catarrh of the finer bronchioles, partly of ill-defined patches of consolidation. The symptoms are those of sharp pyrexia; marked dyspnœa, with free movement of the alæ nasi; cyanosis; hard, painful, unsatisfying cough, with viscid expectoration, often streaked with blood, but never typically rusty; much restlessness; and a frequent weak pulse.

Diagnosis of Broncho-Pneumonia. — This problem usually confronts us in the following way: A child which has been suffering from measles, whooping-cough, or bronchitis, and has presented the ordinary symptoms and physical signs of

bronchitis, suddenly undergoes an exacerbation of symptoms. The question arises, Has broncho-pneumonia supervened? We endeavour to solve this question by attention to the following points:

(*a*) *The Character of the Breathing and Cough.*—A rapid increase of respirations to 50, 60, 70, or 80 per minute following upon bronchitis, measles, or whooping-cough is highly suggestive of broncho-pneumonia. An equal acceleration of the breathing might be present in croupous pneumonia, but in this case the onset will probably be more sudden, and not preceded by catarrhal symptoms. The cough becomes short, hard, dry, frequent, and painful—very different from the ordinary cough of bronchial catarrh. The sputum becomes scanty, tenacious, perhaps streaked with blood, never rusty. In young children the expectoration is absent, the alæ nasi work vigorously, the child is anxious and restless, the pulse is very rapid and weak.

(*b*) *The Course of the Temperature.*—A sudden rise of temperature in bronchitis, measles, or whooping-cough may have various explanations; but in association with signs of increasing respiratory distress it is suggestive of the supervention of broncho-pneumonia.

(*c*) *The Character of the Physical Signs.*—These may be ambiguous and cause much difficulty in diagnosis. In fairly typical cases the ordinary physical signs of bronchial catarrh are replaced by fine, crackling râles, not limited to inspiration, and present on both sides of the chest, but over variable areas. The breathing is usually harsh over the upper parts of the chest, and absent, feeble, or more or less bronchial at the bases or along the spine in the interscapular regions. Percussion exhibits many variations, and may be very misleading. Owing to the smallness of the areas of consolidation, and the over-distension of air cells in the vicinity of the consolidated areas, marked dulness is unusual, and the note may be even tympanitic in quality. Extensive collapse of

a lung, usually basic in site, may cause a considerable area of dulness, which is not usually very pronounced. The chest moves up and down, and there is little true expansion. Recession of the lower intercostal spaces and of the epigastrium is usually present. Vocal fremitus and vocal resonance are variable, increased in proportion to the amount of consolidation, but apt to be annulled by blocking of the bronchial tubes with secretion.

The chief conditions which may be confused with broncho-pneumonia are croupous pneumonia, acute disseminated tuberculosis of the lungs, typhoid fever. The chief differential rules are the following:

In croupous pneumonia the onset is more sudden; there will probably be no pre-existing bronchitis; the physical signs will probably be unilateral; dulness will be pronounced; the sputum, if present, will be characteristic; the course of the temperature will establish the diagnosis.

In acute disseminated tuberculosis the simulation of broncho-pneumonia may be so close that for a time a differential diagnosis may be impossible, but in the former condition the physical signs tend to become more marked at the apices, bacilli may be found in the sputum, and tubercles may be discovered in the choroid.

In typhoid fever the respiratory symptoms will probably be less intense, the temperature and tongue may show some characteristic changes, the abdomen may show some distension, and the spleen will probably be enlarged.

The prognosis in broncho-pneumonia is always grave, especially in very young or rickety children and after whooping-cough. Unfavourable signs are—much collapse, a rapid, thready pulse, much cyanosis, a very high temperature, much recession of the intercostal spaces, convulsions if occurring late in the disease.

TREATMENT OF BRONCHO-PNEUMONIA.—The treatment should be in general tonic and stimulating. All depressing

measures should be avoided, and opiates should be used as sparingly as possible, but cannot always be dispensed with. Expectorants are useful. Inhalations of steam are valuable, but the 'tent' should be avoided. A warm alkaline spray containing bicarbonate of soda and glycerine of carbolic acid may be tried. Emetics are not advisable.

Wet packs, tepid or cold baths, and cold affusion to the chest while the child sits in a warm bath are sometimes very useful. This last measure is somewhat severe and not to be ordered without discrimination, but it is a powerful remedy. The food should be as nourishing as possible. Stimulants are usually required.

PRIMARY CONGESTION OF THE LUNGS. — Whether a primary congestion of the lungs independent of pneumonia occurs is still an open question. French clinicians recognise the condition, and even subdivide it into numerous types, of which a recent writer, L. Rénon, describes no less than seven.* The 'Maladie de Woillez' has long had a place in French medical literature. The following are said to be the chief points in connection with this affection: It begins abruptly, with slight rise of temperature, rapid pulse, and friction sounds in the submammary region. There is expectoration of a yellowish froth, and a watery albuminous fluid resembling a thin solution of gum arabic. Vocal fremitus is normal or slightly diminished; slight dulness on percussion is present; soft blowing respiration and coarse moist crepitations are present. The crisis occurs on the fourth or fifth day, and by the tenth day the patient is well.

Other types described are: The catarrhal congestion of Dupré and Grasset, in which all the thoracic structures except the heart are more or less involved, pleurisy and muscular congestion being present; the pleuro-pulmonary congestion of Potain and Serraud, which has a more pro-

* L. Rénon, *Journal des Practiciens*, No. 24, 1905.

longed course than the foregoing; the spleno-pneumonia of Grancher and Queyrat; and the chronic pulmonary congestion of Bouchut, which may last for several months and be accompanied by persistent fever and extreme emaciation.

A study of the literature of the subject has failed to convince me that the above types represent real clinical or pathological entities.

LECTURE XI

BRONCHITIS, ACUTE AND CHRONIC—EMPHYSEMA

SUMMARY:

Symptoms of bronchitis.
Physical signs of bronchitis.
Diagnostic problems in connection with bronchitis.
Capillary bronchitis.
Prognosis in bronchitis.
Chronic bronchitis and emphysema.
Symptoms of chronic bronchitis.
Physical signs in chronic bronchitis and emphysema.
Course and event in cases of bronchitis.
Treatment of bronchitis:
Treatment of acute bronchitis of the large and middle-sized tubes.
Treatment of capillary bronchitis.
Treatment of chronic bronchitis and emphysema.

BRONCHITIS.

The problems which interest us most in connection with bronchitis are therapeutic rather than diagnostic. The diagnosis of the disease is in general obvious, but in many cases we have to ask the question whether the bronchial catarrh which reveals itself by easily recognised signs is a primary or substantive condition, or whether it is a mere symptom of some more important malady—*e.g.*, tuberculosis or typhoid fever. Bronchial catarrh is, perhaps, the most frequent of all secondary affections, and is an almost constant feature of heart disease, renal disease, tuberculosis, gout,

syphilis, many of the infectious fevers, especially measles and typhoid fever, and of most pulmonary affections. It may involve chiefly the larger and middle-sized bronchi or the fine bronchioles, and this difference in distribution is found to correspond to important variations in the severity and gravity of the malady. The disease is much under the influence of climate and weather, being particularly common in cold damp climates and in changeable weather, and is especially frequent amongst children and persons of feeble constitution. The most constant symptoms are cough, expectoration, and soreness or oppression of the chest; and the physical signs are chiefly rhonchi and râles. We shall first consider the symptoms of bronchitis; then deal with the physical signs a little more in detail; then consider the various diagnostic problems which arise; and finally pass on to questions of treatment.

THE SYMPTOMS OF BRONCHITIS—*Cough.*—This is a reflex act which begins in the acute stage, and is at first hard, 'dry,' and spasmodic; later it becomes looser and moister as secretion increases. The bifurcation of the trachea and the inter-arytænoid region seem to be especially sensitive portions of the air passages. When cough is severe and persistent it is not uncommonly followed by vomiting, especially in children. A severe spasmodic cough, followed by vomiting, should suggest to us the possibility of whooping-cough, but is not unknown in bronchitis.

Expectoration.—At first scanty, clear, viscid, and sometimes streaked with blood, the sputum tends to become more abundant, and goes through the stages of mucoid expectoration (*sputum crudum*), muco-purulent expectoration (*sputum coctum*), and in exceptional cases pure purulent expectoration. This last form is exceptional in ordinary bronchitis, and is rather suggestive of bronchiectasis, ruptured empyema, or phthisis.

Pain in the Chest.—This takes the form of soreness or

burning sensation under the sternum (substernal pain), or of pain along the attachments of the diaphragm or some of the other respiratory muscles. It is quite distinct, and easily differentiated from the 'stitch in the side,' which is characteristic of pleurisy. The chest pain of bronchitis is apparently dependent on two factors—viz., an inflamed bronchial membrane and strain of the diaphragm and other respiratory muscles in coughing.

Dyspnœa.—In the acute stage of bronchitis this takes the form of hurried and laboured breathing, affecting both inspiration and expiration, and is especially marked in capillary bronchitis; in the chronic stage the dyspnœa is decidedly expiratory.

Fever.—In many cases of bronchitis pyrexia plays a subordinate rôle, and the height of the temperature is not always a safe guide to the gravity of the case. In many cases of severe, and even fatal, bronchitis in the adult the temperature is at no time excessive. In the child, however, capillary bronchitis is pretty constantly accompanied by a sharp pyrexia, the thermometer usually rising to 103°, 104°, or higher. In debilitated subjects the temperature may be no index to the severity of the attack.

Pulse.—The pulse frequency is usually increased in proportion to the height of the temperature. Undue frequency, out of proportion to the pyrexia, is often an index of feeble constitution or the presence of some complication, such as rickets.

Urine.—In the acute stage of bronchitis the urine has usually the ordinary febrile characters; in chronic cases it may be influenced by weakness of the heart and passive congestion of the kidneys. Albumin and casts are occasionally present.

Dyspepsia, impaired nutrition, headache, cyanosis, may be mentioned as further symptoms of bronchitis, but do not call for detailed consideration.

PHYSICAL SIGNS OF BRONCHITIS—*Inspection.*—The breathing is usually hurried and more or less superficial; recession of the lower intercostal spaces and epigastrium is common in capillary bronchitis; delay in the expansion of one side is said to occur in those very rare cases of unilateral bronchitis; in chronic cases the movements of the chest are deficient in amplitude, not truly expansile, more or less of the 'up-and-down' type.

Palpation.—Any delay or inequality in the expansion of the chest may be further explored by palpation. Rhonchal fremitus is often present.

Percussion.—As a rule, in acute bronchitis the percussion note is normal. No amount of secretion in the bronchial tubes will produce dulness, if collapse or œdema is not present. Extensive œdema may be present without dulness. Acute or chronic emphysema will give rise to a tympanitic note.

Auscultation.—The breathing preserves its vesicular quality, but may be harsher or weaker than normal. It is never bronchial. Harsh breath sounds suggest either a difficulty in the entrance of air into the part, or compensatory hyperactivity of the part. Very weak or absent breath sounds point to complete bronchial obstruction or collapse. In chronic cases the expiration is much prolonged.

The character of the rhonchi and râles is distinctive and pathognomonic. In the 'dry' stage we get either sonorous or sibilant rhonchi, according as the large or the small tubes are involved. Sibilant rhonchi are not much influenced by coughing; sonorous rhonchi are much influenced. Rhonchi are often most marked during expiration. Bubbling or mucous râles are present when a certain amount of secretion is present in the tubes. They are coarse, medium, or fine according to the size of the tubes in which they are produced. They are often more abundant at the bases of the lungs than elsewhere. They are often much influenced by coughing. In the absence of complications—*e.g.*, œdema, hypostatic

congestion, broncho-pneumonia—these râles *never possess the crepitating or consonating quality*. The beginner should accustom himself as soon as possible to recognise the distinction between the 'bubbling,' 'mucous,' or 'non-consonating' râle, which indicates that the lung tissue in the neighbourhood of the tube where the râle is generated is spongy and air-containing, from the 'consonating' râle, which indicates that the adjacent lung tissue is in some way condensed or consolidated. This distinction is not always easy, but when appreciated it is of fundamental importance. The persistence of a fine râle after repeated coughing and deep inspiration points to the probability that it has its origin at the extreme end of the bronchioles or in the infundibula.

Diagnostic Problems in Connection with Bronchitis.—We shall put these problems in the form of hypothetical cases:

(*a*) A child (five to fifteen years of age) is suddenly seized with a sharp feverish attack, of which the most obvious local manifestation is general bronchial catarrh. What are the probable alternatives? The case may, of course, be one of simple primary bronchitis, but before accepting this diagnosis it is well to consider the possibility of measles, influenza, whooping-cough, or croupous pneumonia. Measles is usually ushered in by a sharp bronchial catarrh, and at first this condition may entirely dominate the clinical picture. But the child sickening for measles is commonly more sharply ill than the amount of bronchial catarrh present will readily account for; the temperature is higher and the prostration greater than the thoracic signs would suggest; conjunctival suffusion is usually present, and photophobia is not rare; by the second or third day Koplik's spots may be found in the mouth, and by the fourth day the characteristic measly rash will probably appear on the palate or face.

Influenza may set in with well-marked general bronchitis,

but this mode of onset is not, in my experience, common. In influenza, however, other characteristic symptoms will probably be present—viz., severe pains in head, back, and limbs; much physical and mental prostration, quite out of proportion to the thoracic signs; general hyperæsthesia. The presence of an epidemic or the existence of other sporadic cases in the house or neighbourhood will, of course, often much assist the diagnosis.

Whooping-cough often sets in with smart, general bronchial catarrh, and the practitioner should have it in his mind, especially in presence of an epidemic. The characteristic whoop may be for a time absent, but the attacks of coughing will usually be severe, paroxysmal, and frequently end in vomiting; while the physical signs in the chest are often slight in comparison with the severity of the symptoms.

Croupous pneumonia is in certain rare cases ushered in by smart, general bronchial catarrh. The chief helps to diagnosis here will be the greater suddenness of the onset, the higher range of the temperature, and the character of the sputum, if any be present. Physical signs may for a time be inconclusive, but at any moment we may find unequivocal signs of consolidation. Physical examination in these cases should be frequent and thorough.

(*b*) A child or young adolescent develops within a few days to a week or two a serious pyrexia, of which the chief local manifestation is general bronchial catarrh. What are the probable alternatives? Once more, the case may be simply one of bronchitis, but before accepting this diagnosis it is well to consider the possibility of typhoid fever or general miliary tuberculosis.

Typhoid fever is always accompanied by some bronchial catarrh, and this catarrh may be so marked that the practitioner is thrown off his guard, and accepts it as the substantive diagnosis. If this source of difficulty is carefully borne in mind, error ought to be rare. Typhoid fever is usually

accompanied by well-marked frontal headache, which abates in a few days, and rarely lasts far into the second week; the state of the tongue and the abdomen, the enlarged spleen, in some cases the presence of spots, and the peculiar features of the temperature chart, ought to make diagnosis easy. These cases are commonly misinterpreted (and the error is by no means a rare one) because the possibility of typhoid fever is never considered.

General miliary tuberculosis may closely simulate bronchitis, but may in general be differentiated by the higher range of the temperature, the more persistent pyrexia, the more marked prostration, and the tendency to delirium. The adventitious sounds in acute miliary tuberculosis tend to assume the form of fine crackling râles, and to be more or less localized. Bacilli may be present in the sputum, but their absence at the outset must not lull to rest the suspicion of tubercle, if on other grounds that suspicion seems to rest upon a reasonable foundation.

(*c*) A patient of any age has been ailing for some time —weeks or months—and the only obvious physical signs are those of general bronchial catarrh. What are the probable alternatives?

Most of these cases are simply bronchitis, but it is well to inquire for the possibility of heart disease, renal disease, gout, and chronic pulmonary phthisis. As a rule these conditions, if carefully inquired for, may be recognised without serious difficulty. The differential diagnosis of bronchitis and chronic phthisis will be hereafter considered.

(*d*) A patient presents a localized bronchial catarrh, involving either one entire lung or one lobe of one lung. What are the probable alternatives?

There is no doubt that bronchitis is occasionally limited to one lung, or even to a single lobe, yet this is one of the rarest phenomena of disease. I have, however, seen several cases where no other view seemed tenable. Cases

are much less rare where the signs of catarrh are marked on one side and slight on the other side. Localized bronchial catarrh must always be looked upon with great suspicion, and if confined to one apex the probability of tubercle amounts to little short of a certainty. Yet nearly every observer reports cases of simple apical catarrh. The existence of such cases is not to be denied; but such a diagnosis should only be formulated with extreme caution, after repeated examination of the sputum, and after the symptoms and history have been fully elicited and scrupulously weighed. A simple apical catarrh will probably exhibit less constant physical signs from day to day than if tubercle is present; the breathing will not depart much from the usual vesicular type; and afternoon fever and other phthisical symptoms will be absent. Localized cog-wheel breathing may possess some significance in such cases as pointing to tubercle, but the value of this sign is much disputed. A localized bronchial catarrh confined to one lower lobe is very rare, but sometimes in the course of convalescence one lower lobe clears up before the other, and if such cases are first seen at this stage doubts regarding the diagnosis may arise. Generally in such cases a patch of pneumonic consolidation is present, although it may not necessarily yield recognisable physical signs.

(*e*) A patient presents some of the usual bronchial symptoms—cough, expectoration, dyspnœa, or pain in the chest—but there are no physical signs. What are the probable alternatives?

I suppose most practitioners have met such cases, and have had a good deal of difficulty and anxiety in dealing with them. The most usual explanations are the following:

1. *Incipient Phthisis which does not yet yield any Physical Signs.*—Our safety in such cases will lie in keeping the patient under observation; repeated examinations of the sputum, if any can be obtained; observations of the after-

noon temperature; and the prescription of reasonable prophylactic measures.

2. *Hysterical Phthisiophobia.*—These cases are not at all rare. Nervous young women whose nervous tone has been lowered by constant attendance upon phthisical relatives and by bereavement often fancy that 'they are falling into consumption,' and their unconscious mimetic faculty soon fills out the symptomatic picture. Many of these cases have come under my observation, and they require very careful handling. To exclude the possibility of phthisis is obviously difficult, often impossible, while to label a real case of incipient phthisis as simply one of neurosis is to commit one of the most unfortunate of errors. Our first duty is clearly to make sure that there are no signs of phthisis in the chest, and no afternoon fever or loss of flesh not otherwise capable of explanation. If any sputum can be obtained (and these patients usually manage to hawk up phlegm and not uncommonly produce a quite respectable hæmoptysis), it should, of course, be carefully examined. The throat should be examined, and not uncommonly a relaxed and congested pharynx will afford a probable source for some of the patient's symptoms. The patient's eagerness to affirm the worst view of her case is usually in striking contrast with the commonly hopeful attitude of the consumptive. We can generally recognise whether the patient is of neurotic temperament or not.

3. *Foreign Bodies in the Air Passages.*—The cough in such conditions is usually hard, paroxysmal, unsatisfying, and either unaccompanied by expectoration or there may be a little clear, perhaps blood-stained, mucus. Local soreness in the trachea or over a main bronchus is often present. The history may, of course, afford decisive assistance, but is sometimes misleading.

4. *Enlargement of the Bronchial Glands.*—This is the explanation of some cases of persistent, hard, paroxysmal

cough. The diagnosis is very difficult, and is sometimes conjectural. Dulness on percussion may sometimes be made out about the upper end of the sternum. Stridor is not uncommon. Dysphagia is rare.

5. *Aneurysm.*—In this condition the cough is often hard, 'brassy,' and clanging, the expectoration either absent or blood-stained; local pain and pressure signs may be present; aphonia is common; tracheal tugging should be enquired for; age, sex, history of syphilis, alcoholism and hard living are most important; evidence of vascular degeneration will probably be present.

Capillary Bronchitis.—This condition may be either primary or may supervene upon a bronchitis of the large and middle-sized tubes. In some cases the involvement of the finer tubes is much more pronounced upon one side than the other, or may even be limited to one side. As stated in dealing with broncho-pneumonia, the distinction between this latter condition and capillary bronchitis is difficult and often impossible. Capillary bronchitis is common amongst young children and the aged. Its gravity in the former case is probably due in large measure to the small calibre of the fine tubes at this age, and in the latter case to the defective elasticity of the tubes. When capillary bronchitis supervenes upon a bronchitis of the large or middle-sized tubes there is a marked aggravation of the patient's symptoms. The pyrexia increases; the breathing becomes more rapid, painful, and accompanied by an incessant, hard, irritating cough; there is recession of the lower part of the chest; the expectoration is scanty, viscid, and perhaps blood-stained; cyanosis develops; the pulse becomes very frequent and weak; the general condition indicates a severe illness and a precarious outlook. The essential physical sign is a small crackling râle audible over the affected tubes.

The diagnosis of such cases presents little difficulty.

Henoch has a useful axiom: 'Children which can cry for some time without coughing are not suffering from bronchitis.' This rule will be found trustworthy.

THE PROGNOSIS IN BRONCHITIS.—In bronchitis limited to the large and middle-sized tubes the outlook at any age is favourable as regards danger to life, provided there are no complications which involve exceptional risk. In cachectic children and old people, however, the forecast requires to be guarded. The chief danger of bronchitis of the large and middle-sized tubes is that the condition may recur and become chronic. This danger is by no means confined to the elderly, but is present in childhood also.

In capillary bronchitis, on the other hand, the danger is always serious and often extremely grave. In infants the disease may be fatal within twenty-four hours of the onset. The prognosis depends chiefly upon the constitutional condition of the patient, the nature of the antecedent disease from which capillary bronchitis has developed, and the severity of the symptoms. The presence of cachexia, as in rickets or hereditary syphilis; the co-existence of whooping-cough; marked dyspnœa and recession of the ribs; much cyanosis; and a rapid 'running' pulse, are of evil augury. The response to treatment has a good deal of prognostic import. Signs of grave involvement of the nervous system are ominous. There is an old clinical rule that when once the child falls back in the bed in these cases and ceases to cough the end is not far off. This rule will usually be found trustworthy.

CHRONIC BRONCHITIS AND EMPHYSEMA.—Bronchitis is very prone to recur and become chronic, and in chronic cases more or less emphysematous change is practically constant. Chronic bronchitis may also develop gradually, without any preceding acute stage, as in persons engaged in dusty occupations, the intemperate, the subjects of heart

disease, gout, rickets, renal disease, and certain forms of pulmonary disease, such as chronic phthisis. Affections of the nose and naso-pharynx, giving rise to mouth-breathing, are among the causes of chronic bronchitis.

SYMPTOMS OF CHRONIC BRONCHITIS—*Cough.*—This is the most important symptom, and presents many variations as regards character and intensity. It is usually worst in the mornings; in winter, or in cold, damp, changeable weather; or after exertion. Some patients suffer most on going to bed, or wake up to cough after a short sleep. The cough is closely connected with the accumulation of secretion and the necessity for its expulsion.

Sputum.—The sputum is usually abundant, and more or less muco-purulent in character, but varieties of many kinds may be observed. It is sometimes tough and scanty, oftener loose and abundant. A dry catarrh was supposed by Laennec to be specially characteristic of gouty cases.

Dyspnœa.—Some degree of dyspnœa is usually present, slight in early uncomplicated cases, marked in advanced cases, where there is much emphysema and important changes in the right heart. The dyspnœa is much affected by all conditions which tend to aggravate the general state, and by exertion. There is often much wheezing and a prolongation of the expiration.

Pain in the Chest.—This is not usually marked, but there may be some substernal soreness or pain along the attachments of the diaphragm and other muscles of respiration.

Aspect of Patient.—In early or slight cases nothing peculiar is observed, but in advanced cases, where circulatory changes are marked, the patient tends to have a characteristic aspect. The face is swollen; the cheeks, nose, lips, ears, and finger-tips assume a bluish tinge, which deepens on exertion; the auxiliary muscles of inspiration stand out prominently; the patient speaks in broken sentences, and often pauses for a word.

Urine.—In early cases the urine presents no characteristic changes; in advanced cases it is often scanty, high-coloured, loaded with urates, and may present a trace of albumin. These changes depend mainly, or perhaps entirely, upon circulatory conditions.

Physical Signs of Chronic Bronchitis and Emphysema.—The physical signs vary much, according to the severity of the case, its duration, and the extent to which secondary changes are developed. At an early stage the chief physical signs are sonorous and sibilant rhonchi, with more or less moist râles, the former generally distributed throughout the chest, the latter more numerous at the bases. The breath-sounds are either fairly normal, or somewhat weak, or, in some cases, harsh. The rhythm of the breathing is not much affected, but even at this stage there is a tendency to prolongation of the expiration. The percussion note is not perceptibly altered.

As the case progresses, and as emphysema develops, we get characteristic signs. The chest tends to become rounded, and to assume the 'permanent inspiratory' form. The percussion note becomes hyper-resonant, and falls in pitch. The area of pulmonary resonance is increased in all directions, and the superficial area of cardiac dulness is diminished, or may even disappear. The breath-sounds are weakened, and the prolongation of the expiratory sound becomes pronounced. Rhonchi and moist râles may or may not be present, but are rarely wanting for long. Epigastric pulsation is often visible.

Course and Event in Cases of Bronchitis.—Acute cases may end in death, complete recovery, permanently impaired health, or recurrence of the disease. In the capillary bronchitis of children recovery is often tedious. The tendency to recurrence is marked in all varieties of bronchitis.

In chronic cases we can distinguish certain types:

(*a*) The patient who is subject to recurring attacks of

bronchitis, but who is quite well during the intervals between his attacks.

(*b*) The patient who has an attack of bronchitis every winter or spring, but who is well during the warm season.

(*c*) The patient who is never free from bronchitis, but has exacerbations and ameliorations, the former being usually associated with season, weather, and exposure.

(*d*) The patient who is a chronic invalid, and is, to a large extent, a prisoner in his house.

These types represent progressive degrees of gravity, and are important from the point of view of prognosis and treatment. To determine to which of these classes any individual patient belongs gives us, to a certain extent, the measure of the case, and assists us in forecasting what may be fairly expected from treatment, and in the choice of therapeutic measures.

THE TREATMENT OF BRONCHITIS.—The treatment of bronchitis involves many important problems, and should be undertaken in the conviction that in the large majority of cases much may be done, and that no case is so intractable as to be outside the range of palliative measures. It will be convenient to consider the subject under three heads: treatment of acute bronchitis of the larger tubes, treatment of capillary bronchitis, treatment of chronic bronchitis and emphysema.

Treatment of Acute Bronchitis of the Larger Tubes.—In the absence of grave symptoms, such as much dyspnœa, cyanosis, or feeble pulse, active treatment is unnecessary, as these cases tend naturally to a favourable issue. The patient should be kept in bed for a few days in a comfortable room of a temperature from about 65° F., and the air may be with advantage moistened by means of a steam-kettle. Continuous high temperatures up to 70° F. or over and saturation of the air with steam are decidedly to be avoided as too depressing. A warm poultice to the chest is usually agreeable

to the patient, or in certain cases we may see cause to prefer iodine or the liniment of turpentine. Too frequent or too long-continued poulticing is undesirable. After the poultices have been withdrawn, the chest may be wrapped in cotton-wool. The ventilation of the room should be carefully attended to. The hot, stuffy, over-humid air which is often found in rooms where these cases are under treatment is opposed to all sound views of treatment. As regards the use of drugs in these cases. a diaphoretic mixture, with the addition of a little hippo, antimonial wine, and in certain cases compound tincture of camphor, will be found beneficial. Antimony is a powerful remedy, at present much out of fashion. In young, vigorous patients and in 'sthenic' attacks it should not be overlooked. Aconite in minute doses sometimes acts well. The use of sedatives requires much care and discrimination. If they can be dispensed with, so much the better; but in many cases some relief to a teasing and irritable cough is required. In very young children, and in the elderly, sedatives must be used with great parsimony, but small doses of compound tincture of camphor will be usually safe and effectual. Dilute hydrocyanic acid is also useful. Saline aperients are valuable at this stage. A little later, when expectoration has begun to come freely, our chief remedies are carbonate of ammonia, alkalies, strychnine, squill, iodide of potash, senega. When the sputum is tough, apomorphine is a valuable drug. During convalescence iron, quinine, and arsenic are our chief resource. The food may be liquid at first, but in a short time a more generous dietary is both safe and valuable. Stimulants are sometimes required. Change of air when the case is convalescing is often the most potent therapeutic measure of all. Various health resorts have been found useful in bronchitic cases. The selection amongst such resorts is somewhat difficult, and must have regard to the patient's general condition, especially from the point of view of the digestive and nervous systems,

as well as to the bronchial malady. In general, fairly warm and sheltered inland resorts are best. We shall return to this subject in discussing the treatment of chronic bronchitis.

A few points in the above sketch require a little fuller consideration. Some good authorities doubt the value of the ammonia preparations as expectorants.* It would seem to me that carbonate of ammonia, which on the whole I am inclined to regard as a valuable drug, sometimes dries the expectoration up more than is desirable, and that with the checking of the expectoration the patient is in certain cases worse, instead of better. Senega is, I think, a much overrated drug, which I employ but seldom. The nauseating drugs—antimony, hippo, apomorphine, and pilocarpine—are undoubtedly amongst our best remedies in bronchitis if used with discrimination. The tendency is to use them in too large doses. We can get the required expectorant action without producing active nausea. Apomorphine should be given in doses of about $\frac{1}{10}$ to $\frac{1}{5}$ grain for an adult, and proportionately less for a child. Pilocarpine may be given in doses of $\frac{1}{120}$ to $\frac{1}{60}$ grain three to five times daily.†

Amongst sedative drugs we have belladonna, hydrocyanic acid, and the various opium preparations to select from. By far the most trustworthy are morphia, heroin, and compound tincture of camphor. Belladonna has been recommended in the form of a spray, and cocaine, especially in cases where there is irritation of the upper part of the air passages. When using sedatives we ought to watch carefully their effect upon the amount of expectoration. If this is diminished while at the same time the breathing and general condition are not improved, these remedies are doing harm. A simple inhalation which I have used extensively and with much satisfaction is composed of equal parts of compound tincture

* Hoffmann, 'Nothnagel's Encyclopædia of Practical Medicine,' English edition, article 'Bronchitis.'

† *Ibid.*

of camphor and compound tincture of benzoin. A drachm or two of this is added to a pint of hot water in a long-necked jug, and inhaled frequently. Inhalations of hippo and of hot saline solutions are also good.

Iodide of potash is a good drug in bronchitis, but rather, I think, in chronic cases. When asthmatic symptoms begin to show themselves it is invaluable. The balsams—copaiba, turpentine, eucalyptus, and terebene—are, I think, sometimes valuable, but rather in chronic cases. I have seen some very good results from terebene. There is an old tradition that tar is of value in bronchial conditions, and I am inclined to share this view. The reputation of many expectorant drugs seems to rest on no sure basis.

Treatment of Capillary Bronchitis.—In cases of moderate severity the treatment does not differ essentially from the treatment of bronchitis of the larger tubes, but in severe cases, which constitute a large percentage, the practitioner should not adhere too closely to a routine treatment on bronchial lines. He should recognise that the patient is, or may soon be, in imminent peril, that prompt and thorough treatment is required, and that all debilitating measures must be avoided. The usual expectorants are of very little avail in these cases. Active stimulation, the promotion of free respiratory movements, and the warding off of pulmonary collapse and œdema, are the principal objects of treatment. The child should be warmly but lightly clothed; poultices should be avoided; ether, ammonia, strychnine, oxygen, digitalis, and brandy may be employed. Electricity has been found useful. A single emetic dose of hippo is sometimes decidedly beneficial. Leeches or venesection may be resorted to if there be extreme dyspnœa or cyanosis.

A measure about which there is some difference of opinion is the following: The child is placed in a hot bath, and cold water is freely poured over the chest and back. In some cases the child gives a few powerful gasps, which probably

serve to open up a number of collapsed air vesicles, and is obviously relieved. But this measure is somewhat severe, and is certainly capable in some cases of doing harm. The practitioner must use his own judgment whether in any individual case the patient is likely to respond to this procedure. It is unsuitable in cases of feeble patients.

The Treatment of Chronic Bronchitis.—This requires to be varied, according to whether the patient is simply the subject of chronic winter cough, or is gouty or tubercular, or has some chronic cardiac or renal affection. It is evident that every case requires to be considered on its merits, and to be treated largely on general therapeutic lines. Warm clothing; protection from the weather; suitable change of climate, when possible; the due regulation of nutrition, are points which apply to most cases. The patient's occupation may require serious consideration, above all if it is carried on in a dust-laden atmosphere. His place of residence, both as regards locality and the type of house, may call for investigation. A sunny, fairly sheltered locality with a sandy or gravelly soil, and an airy, well-ventilated house, with a southerly, south-easterly, or south-westerly aspect, will in general be found most suitable. If the patient is a boy or a girl at school, the situation of the school, its general régime, the questions arising out of amusements, may all have some bearing on the management of the case. Nasal affections, such as adenoids, chronic naso-pharyngeal catarrh, nasal obstruction, may call for vigorous treatment, as they are a fruitful source of mouth-breathing, a habit which contributes its share to keeping up bronchial irritation.

The general constitutional treatment is highly important, and a broad distinction may be drawn between young patients, with whom the keynote of such treatment should be a bracing and hardening regimen, and elderly patients, where the cardinal rule should rather be protective and soothing management. For the former some form of hydro-

therapy is highly desirable, but must be adapted to the gravity of the case, the constitutional condition of the patient, and in particular his circulatory vigour. Cold or tepid spongings, with vigorous friction afterwards, will in general be found suitable, while prolonged immersion in cold water is to be avoided. In the case of elderly patients, dry friction will often be found best.

Diet and habits must be studied. A mixed diet, nourishing but not luxurious, containing a fair proportion of fatty elements, and only a small relative proportion of carbohydrates, in order to obviate flatulent distension of the stomach and bowels, is desirable. Alcohol should be used sparingly or altogether withheld. Smoking should, if possible, be forbidden. The diet may, of course, have to take special account of such conditions as gout, heart disease, obesity, or other complication. General dietetic rules will call for numerous exceptions. A pure milk diet has sometimes been found effectual. The medicinal treatment requires to be varied according to constitutional condition, the nature of the complications (if any are present), the character of the sputum, the state of the digestion.

In general, some form of counter-irritation is desirable, and I do not know any better counter-irritant than the ordinary turpentine liniment, fortified, if necessary, by the addition of some glacial acetic acid. Iodine is less convenient in cases where the frequent use of a counter-irritant is required. The same objection applies to another very potent counter-irritant—viz., croton-oil liniment. This last is apt to leave ugly scars behind it.

When there is little secretion with much hacking cough, hippo, apomorphia, or pilocarpine may be tried. A pill of nux vomica and hippo is sometimes valuable in such cases. If a sedative is absolutely required morphia is best. It has the advantage of combining easily with apomorphine. When the right side of the heart is involved in these cases,

digitalis is indicated. When secretion is abundant, we may have recourse to the balsams—turpentine, myrtol, copaiba; or to chloride of ammonium; or to creosote, tar, carbolic acid, or terebene. Perchloride of iron is one of the best remedies in these cases.

Inhalations are of great value. We have to choose between inhalations of simple steam, or of hot salines, or vapour arising from hot water containing creosote, tincture of benzoin, turpentine, compound tincture of camphor, or two or more of these remedies in combination. Inhalations from steam atomizers of tar and hippo wine are highly recommended.

Some form of respiratory gymnastics may be employed with advantage in these cases. 'The beginning of respiratory gymnastics,' says Hoffmann, 'consisted in simple compression of the thorax.' This may be done by an assistant during expiration. A breathing-chair has been devised by Rossbach, in which the patient sits, and by means of two levers, to which broad straps are attached, compresses the lower portion of the chest at each expiration. Wolf recommends an elastic shirt or corset to oppose inspiration while helping expiration. He claims that by this instrument the chest is kept in the expiratory position in which the muscles of inspiration act at the best advantage, and that the obstruction to breathing in has no drawbacks.* While the use of any special apparatus must be considered of doubtful expediency, the patient should be exercised in systematized deep breathing. 'The fundamental rule,' says Hoffmann, 'is that movements in which the arms are moved away from the chest assist inspiration, while movements in which the arms are brought near the chest assist expiration, whether the movements be active or passive. In ordinary cases the patient may hold a light dumb-bell, weighing about a pound, in each hand, and raise the arms from the ordinary position

* *Wiener Klinik*, April, 1905.

at the sides to the horizontal position; the hands are then brought into supination, palms upward, and during this movement an inspiration should be taken. The same movements are then repeated in the reverse order until the arms are brought to the sides of the body, and the patient then bends the knees until the dumb-bells touch the floor. During this movement an expiration is performed. In the beginning of the course the patient must rest even after a single movement, and I have come to regard six inspiratory movements of this kind as a very respectable performance that should not be exceeded by most emphysematous patients. . . . The number of movements to be performed is to be accurately prescribed by the physician.'* Such exercises are well conceived, but must obviously be carried out under strict medical supervision, and with due regard to the state of the heart and bloodvessels. They are capable of having an injurious effect if employed excessively or in unsuitable cases.

Waldenburg's apparatus for breathing compressed air has fallen into merited neglect, and all forms of respiratory cabinets are to be regarded rather as curiosities of therapeutics than as devices worthy of a recognised place in practice.

The treatment of chronic bronchitis and emphysema by recourse to spas or bathing stations and by climatic change is, when practicable, the most successful of all methods. The subject is a large one, and can only be dealt with here in outline. Any hard-and-fast rules, such as the rule that patients with scanty expectoration should be sent to moist resorts—*e.g.*, Madeira, Torquay, Falmouth, Lisbon—and patients with abundant expectoration to dry resorts such as Egypt, the Western Riviera, Malaga, or Biskra, is not really to be depended upon. Nor, while warm climates have undoubtedly a much larger range of applicability in bron-

* Hoffmann, 'Nothnagel's Encyclopædia of Practical Medicine,' English edition, article 'Emphysema.'

chitic conditions than cold climates, are we therefore justified in quite excluding the latter. Some bronchitic cases in young persons with vigorous circulations, steady nervous systems, and little emphysema, do excellently well at some of the Alpine stations, preferably of moderate elevation (3,000 to 4,000 feet), but even higher elevations may sometimes be advised. We cannot generalize so easily upon a difficult subject. The patient's nervous and circulatory condition, and even his personal preferences, have to be taken into consideration. Warmth and shelter are usually desirable points, especially in serious cases and in elderly patients. Egypt has great advantages, as the climate can be depended upon, warmth and dryness are assured, and the accommodation is of the first order. Mentone, San Remo, Bordighera, Rapallo, Costebelle, and other Rivieran stations, have much to recommend them. Arcachon is a good resort. Montreux and Bex are good autumn stations. In spring Lugano, Bellagio, or Gardone-Riviera may be tried. In summer there is a wide choice of resorts at home and abroad. Folkestone, Eastbourne, Wiesbaden, Baden-Baden, the Harz Mountains, the Alpine stations up to an elevation of 2,000 feet, may be recommended. I agree with West that sea air is often unsuitable.* The opposite view is held by many writers.

There is not complete agreement as to the type of spa to be advised in bronchitic cases. As a general rule, those stations which possess brine baths and inhalations of salt water may be thought of, such as Reichenhall, Ems, Soden, Salzbrunn, Kissingen, Karlsbad, and Marienbad. Others would give a higher place to the sulphur waters, such as those of Weilbach, Eaux Bonnes, Barèges, Cauterets, Bagnères-de-Luchon. It is probable that in most of these cases the really determining factor is climate rather than the character of the mineral waters.

* S. West, 'Diseases of the Organs of Respiration,' vol. i., p. 144.

LECTURE XII

PLASTIC BRONCHITIS—BRONCHIECTASIS—DISEASE OF THE BRONCHIAL GLANDS—ASTHMA

SUMMARY :

- Plastic bronchitis.
  - Structure of the casts.
  - Diagnosis of plastic bronchitis.
  - Prognosis and treatment of plastic bronchitis.
- Bronchiectasis.
  - Varieties of bronchiectasis.
  - Causes of bronchiectasis.
  - Diagnosis of bronchiectasis.
    - Physical signs.
    - Sputum.
  - Differential diagnosis.
  - Prognosis.
  - Treatment.
- Diseases of the bronchial glands.
  - Causes, symptoms, prognosis, and treatment.
- Asthma.
  - Theories regarding the causation of asthma.
  - The asthmatic paroxysm.
  - Hay asthma.
  - The sputum in asthma.
  - Diagnosis of asthma.
  - Prognosis in asthma.
  - Treatment of asthma.

Plastic Bronchitis.

This is an extremely rare disease, characterized by the exudation of plastic fibrinous material, which forms either solid casts or hollow cylinders within the bronchi. It is a very rare affection, if we confine the term to cases which occur independently of any other obvious morbid condition —*e.g.*, diphtheria, measles, pneumonia, typhoid fever, heart disease, inhalations of irritating gases. Only two cases have come under my notice. The secondary cases are much more numerous; but the bronchial condition, although giving rise to serious symptoms, is apt to be overshadowed by the primary disease.

The bacteriology of the disease is still unsettled. In cases regarded as primary the following micro-organisms have been found: The pneumococcus, *Staphylococcus pyogenes aureus* and *albus*, Löffler's bacillus, and *Streptococcus pyogenes*.

The casts vary in length from a few lines to several inches, a length of from 1½ to 2 inches being common. They have been known to measure as much as 6 or 7 inches. They have a diameter corresponding to that of the tubes in which they originate. They are white or pearly grey in colour, the larger of firm consistence, the smaller softer. They have a laminated arrangement, and most observers are agreed that they are composed of fibrin. In the meshes of the fibrin are inclosed leucocytes, mucus, and epithelial cells. Charcot's crystals, pigmented cells, and fat globules may also be included, and blood corpuscles may be present in the outer layers. Some observers find that the expectorated material is composed of mucin. The condition of the bronchial mucous membrane in this disease has not been determined with certainty.

Diagnosis of Fibrinous Bronchitis.—This is only possible by the discovery of the casts in the expectoration,

and the disease being of great rarity the practitioner is hardly likely to suspect its existence until his attention is drawn to the sputum, when the case may become at once clear. The symptoms may be much like those of asthma—viz., severe paroxysmal cough and expiratory dyspnœa, with mucoid sputum, in which after from five to ten days the characteristic casts make their appearance. Pyrexia usually attends the paroxysms and hæmoptysis is present in about one-third of the cases. The patients are often cyanotic. The physical signs depend upon the distribution and behaviour of the casts. Ordinary mucous râles may be heard, weak breathing is common, and a palpable thrill may be present.

In acute cases death occurs in about 50 per cent. In chronic cases death is rare, except as the result of complications.

The treatment consists in the use of iodide of potash, inhalations of warm vapour containing creosote or lime-water, or change to a warm, dry climate. One of my cases got quite well after some years' residence on the Orange River.

Bronchiectasis.

This disease is rarely primary, but usually follows upon chronic bronchitis, broncho-pneumonia, pulmonary tuberculosis, cirrhosis of the lung, or pleurisy. A majority of cases occur in the male sex and in middle life.

The dilatation of the bronchi may take the following forms: cylindrical, saccular, fusiform, moniliform. The cavities in the saccular form may be as small as a hazel-nut or as large as a hen's egg, or even as the human fist.

The precise physical condition which determines the dilatation of the bronchi is doubtful, whether it be accumulation of secretion, nutritive changes in the bronchial walls, constant coughing, weakness of the bronchial musculature, or cirrhotic change in the lung.

A large number of tubes are usually affected, and both lungs are commonly involved, but the process is sometimes limited to one lobe. The distribution of the affection has a relation to its cause. In tuberculosis the affection is often limited to one upper lobe, and its presence may give rise to puzzling physical signs. When bronchiectasis is a sequel of acute or chronic bronchitis or of broncho-pneumonia many tubes are affected. When bronchiectasis follows upon pleurisy, impaction of a foreign body, or stenosis of a large bronchus, it may for a time be limited to the area of lung affected by the primary lesion. The walls of the bronchi may be thickened or thinned, the mucous membrane is often swollen, reddened, and velvety. The surrounding lung tissue may be either healthy, emphysematous, condensed or fibroid. Extensive areas of broncho-pneumonia and of gangrene may be found around dilated tubes. General bronchitis is usually present, and pleural adhesions are frequent. Gangrenous changes in the lung may be present. Changes in the right heart, lardaceous degenerations of organs, and cerebral abscess are amongst the complications which may be present.

Diagnosis of Bronchiectasis.—This turns upon the gradual development of certain physical signs in the chest and of a characteristic sputum as sequelæ of some chronic chest malady. An area is discovered over which at one time complete dulness is present, and at another time hyper-resonance or cracked-pot sound; at one time silence or weak breathing, at another cavernous or hollow blowing breathing. The sputum takes the form of the expulsion at considerable intervals of large quantities of abundant, offensive expectoration. It is the combination of these signs which goes far to establish the diagnosis of bronchiectasis. We shall now consider these points more in detail.

Inspection.—This may yield no characteristic sign, or in certain cases flattening and delayed expansion may be present.

Palpation.—If the bronchiectatic cavity communicates freely with a bronchus, the tactile fremitus will be increased; if it be filled with fluid, the fremitus will be diminished or entirely lost.

Percussion.—The percussion note depends upon the position and size of the bronchiectatic cavity, the condition of its walls, the character of its contents, and the presence or absence of pleural adhesions. Dulness, usually pronounced, is common, but a normal, hyper-resonant, tympanitic, or amphoric note may be present. A typical cracked-pot sound is, in my experience, rare.

Auscultation.—The auscultatory signs, like those of percussion, will vary much according to the conditions above enumerated. The breathing may be absent, or hollow and blowing, or truly cavernous in character. A saccular bronchiectasis may give rise to signs not distinguishable from those of a tubercular vomica. Râles are often present, and may be bubbling or crackling in character. Coarse gurgling râles of more or less metallic quality are frequent. Bronchophony or pectoriloquy may be present.

Character of the Sputum.—This is often highly characteristic. The expectoration is usually profuse, purulent, fœtid, and voided in large quantities at considerable intervals. The fœtor has been compared to that of rotten cabbage. The expectoration is usually diffluent, not nummulated. On standing for twenty-four hours, it separates into three layers—an upper thin, brownish, frothy layer; a middle layer, greenish in colour, and either clear or containing hanging threads; a lower layer of thick purulent material, containing pus cells undergoing fatty degeneration, epithelium, putrefactive bacteria, and crystals of margarine. Elastic fibres are seldom, or never, present. Some blood is often present; the quantity is often very large, and may amount to 25 or 30 ounces in the day. The accumulation of sputum is chiefly in cases of basic bronchiectasis. Its expulsion often takes

place in the morning, the patient enjoying comparative relief from coughing during the rest of the day. Posture has some influence upon the ease and completeness of expectoration, the patient usually discovering for himself the posture which helps him most.

The symptoms of bronchiectasis vary according to the cause, extent, duration, and progress of the condition. Dyspnœa may be slight or marked. It has relation to the presence of cardiac complications or emphysema. Hæmoptysis is frequent, and seems often to depend on ulceration of the bronchial walls. Anæmia or cyanosis in varying degree is usually to be observed. Pyrexia is frequent, and seems to depend upon retained secretion. The best treatment for it is to promote the expulsion of the sputum. Pyrexia may also depend upon inflammatory affection of the adjacent lung or upon the formation of abscess. Pain, if present, is probably due to co-existing pleurisy. Diarrhœa may occur as a consequence of septic infection or of lardaceous degeneration of the intestines. Albuminuria may be present, and is usually due to lardaceous kidney. Clubbing of the fingers and toes is a frequent symptom, and has considerable diagnostic importance.

The differential diagnosis is sometimes difficult. The following conditions require to be differentiated from bronchiectasis:

1. *Chronic Bronchitis and Emphysema.*—The chief points to be relied upon in this connection are the varying character of the physical signs in bronchiectasis and the character and mode of evacuation of the sputum. Careful percussion is specially valuable.

2. *Ruptured Empyema.*—The history is here the chief guide. It must be borne in mind that empyema may be accompanied by, or be a cause of, bronchiectasis. If the signs of pneumothorax are present the diagnosis is easy.

3. *Tuberculosis.*—The diagnosis in this case rests mainly

upon the examination of the sputum, but it must be remembered—

(*a*) That the failure to discover tubercle bacilli does not disprove the presence of tuberculosis.

(*b*) That the presence of tubercle bacilli does not exclude the possibility of bronchiectasis.

The situation of the physical signs and the progress of the case are important.

Kingston Fowler lays down the following useful axiom: 'A case of pulmonary disease, marked by long-continued copious fœtid expectoration, from which tubercle bacilli and elastic fibres are absent; by diffuse bronchial rather than by limited cavernous or amphoric breathing; by coarse bubbling or metallic rather than gurgling râles; and by extreme clubbing of the fingers, toes, and nose, is probably one of bronchiectasis, in which many tubes are involved.'

Prognosis of Bronchiectasis.—Favourable as regards life and the prospect of the mitigation of symptoms, unfavourable as regards complete recovery. Some cases in children consequent on measles or whooping-cough get quite well. Generally speaking, the disease is a chronic condition which causes much annoyance, but does not necessarily interfere decidedly with nutrition or directly threaten life. Serious complications—such as general sepsis, abscess of the brain or spinal cord, inflammation of the lungs, pyopneumothorax—though not to be forgotten in taking account of the risks attendant upon bronchiectasis, are, upon the whole, rare. Hæmoptysis is occasionally profuse, and may precipitate a fatal issue. Cardiac and renal complications are the usual precursors of death.

Treatment of Bronchiectasis.—The aim of treatment is to remove the cause of the disease, to promote the evacuation of the sputum, to prevent putrefactive changes in it, to combat complications and to maintain the general nutrition. For the evacuation of the sputum the most effectual methods

are respiratory gymnastics, cold affusion to the chest, and general exercise. For the prevention of putrefactive changes in the sputum we may try inhalations of turpentine, creosote, or carbolic acid, or the creosote bath. Intra-tracheal injections of menthol or guaiacol after the method of Rosenberg are, in my judgment, decidedly objectionable. Gangrene of the lung has been known to follow this line of treatment. The internal use of such drugs as Peruvian balsam, copaiba, turpentine, eucalyptus, and myrtol is much praised by some authorities, but the results are seldom brilliant. Subcutaneous injections of guaiacol or creosote have been recommended. Tonics and a general tonic regimen are important. A warm, dry climate, a well-regulated life of outdoor activity and systematized breathing will do more than any other form of treatment.

Surgical interference has been advocated, but the results are not encouraging. Tuffier reports ten deaths out of forty-six cases operated on, and in most of the non-fatal cases a permanent fistula was left behind. It is evident that a condition like bronchiectasis, which usually affects many tubes, and, where a precise local diagnosis is often impracticable, does not offer a very promising field to the surgeon.

Diseases of the Bronchial Glands.

A diagnosis of disease of the bronchial glands is only occasionally possible during life, but these affections are common, and should be before our minds in investigating obscure pulmonary conditions. These glands are situated chiefly in the space formed by the bifurcation of the trachea and around the main bronchi, and are from twelve to fifteen in number. They are often found after death of a deep black colour from absorption of carbon in persons who have lived in towns and who have attained adult age. They are found enlarged in a great variety of morbid conditions, including tuberculosis, syphilis, lymphadenoma, measles,

scarlet fever, whooping-cough, broncho-pneumonia, gangrene of the lung, pneumoconiosis. In a large majority of cases this enlargement gives rise to no symptoms, and subsides with the disappearance of the primary disease. When symptoms arise they are mainly, or entirely, due to mechanical pressure of the enlarged glands upon neighbouring parts—*e.g.*, the trachea, a main bronchus, the vagus nerve or one of its recurrent laryngeal branches, the superior cava, the innominate vein, the thoracic parietes. Dulness may be present between the shoulder-blades.

In the few cases where I have ventured upon a diagnosis of disease of the bronchial glands the leading symptoms were paroxysmal cough, expectoration of various characters, dyspnœa, and some degree of stridor. Most of the cases were probably tubercular.

When these symptoms are at all pronounced, *i.e.*, where there is evidence of pressure on important parts, the outlook is grave, and the prognosis should be very guarded. I have known sudden death to occur with very slight warning. On the other hand, it is probable that many cases of enlargement of the bronchial glands get well without giving rise to marked symptoms. The possibility that pulmonary tuberculosis may either co-exist or develop as a sequel to the involvement of the glands should always be before our minds. The glands may suppurate, and cerebral abscess has been an occasional sequela. Perforation of a bronchus may lead to a septic broncho-pneumonia.

The treatment must be mainly constitutional and symptomatic. When marked stridor is present, the possibility of surgical interference might be considered, but, from the nature of the case, can seldom be recommended.

Asthma.

Asthma is a disease of great interest, around which many controversies, ætiological and therapeutical, have

raged, and with regard to which many questions still remain open. The use of the term 'asthma' as applying to the dyspnœa of heart disease or renal disease is objectionable, as it suggests a connection between conditions which have nothing in common except a superficial resemblance of symptoms. 'Hay asthma' is a separate and clearly differentiated condition which need cause no confusion.

Asthma is best understood in a clinical sense, and might be defined as a condition 'characterized by intermittent attacks of severe paroxysmal dyspnœa of the expiratory type, often accompanied by a characteristic sputum, not directly threatening life, and unaccompanied by any known anatomical changes.' It is probable that the proximate cause of the asthmatic paroxysm is spasm of the muscular fibres of the bronchioles, with or without a concomitant *bronchiolitis exudativa;* but other authorities regard it as depending on a fluxionary hyperæmia of the bronchial mucous membrane. It cannot be doubted that the nervous factor is the predominating influence in asthma, but it is not clear whether it acts through the motor branches of the vagus to the muscular fibres of the bronchi, or through the vaso-motor branches of the sympathetic to the vessels of the bronchial mucous membrane. The suggestions that the dyspnœa may be due to spasm of the diaphragm, spasm of the inspiratory muscles, or paralysis of the bronchial muscles, do not seem very probable. How far affections of the upper air passages, such as adenoids, nasal polypi, hypertrophy of the tonsils, chronic naso-pharyngeal catarrh, nasal obstruction, and the like, are valid causes of genuine asthma must be considered doubtful. The experience of rhinologists on this subject and of physicians seems somewhat conflicting. Many cases are on record where the removal of nasal polypi is said to have cured asthmatic attacks, and it is affirmed that in certain cases the growth of a fresh polypus has been followed by a return of the

asthmatic paroxysms. I have never seen a case of this kind. It must be borne in mind in estimating the value of such statements that there is evidence to show that the application of the galvano-cautery to the nasal passages has been followed by improvement in certain cases of asthma, although the nasal passages were to all appearance healthy. The influence of suggestion and of counter-irritation in a condition where the nervous factor is clearly predominant must not be left out of account. The rhinologist often sees the temporary benefit of his local medication, but does not see the relapses so often as the physician, to whom these cases so frequently drift back. The evidence that nasal causes ever produce, or nasal medications cure, asthma is not to my mind entirely conclusive, but due weight must be assigned to the contrary opinion, which has the support of so many good observers. I have no experience of genuine asthma as the result of reflex irritation from the digestive organs, the ear, or the uterus; but such cases are reported on good authority. We must assume that in such cases there was some predisposition to asthma in the form of undue excitability of the bronchial mucous membrane, or of the vagus nerve. All my experience tends to support the view of A. Fränkel and Curschmann that in asthma we have both a neurotic and an inflammatory element in the ætiology. Fränkel believes that there is always some change in the bronchial mucous membrane, and that the process originates in the bronchioles, in which an abundant epithelial desquamation takes place. The suddenness of the attacks, and their occasional dependence on peripheral irritation, require the postulate of a nervous factor. As a rule the catarrh, the broncho-spasm, and the influence of the nervous system, work hand in hand.*

The distinction so often drawn between 'bronchial asthma'

* 'Nothnagel's Encyclopædia of Practical Medicine,' article 'Asthma.'

and pure 'neurotic asthma' is illusory. The two elements are usually present conjointly, and it is impossible to disentangle them.

THE ASTHMATIC PAROXYSM—*Premonitory Stage.*—This is present in about half the cases, and consists of alteration in the spirits, headache and desire to sleep, frequent sneezing, itching of the skin, flatulence, polyuria.

Stage of Invasion.—This is usually quite sudden, and frequently occurs during the night, often about 2 a.m. The patient awakes after one or several hours of quiet sleep, and either immediately passes into a paroxysm or has a brief period of warning, characterized by tightness in the chest, short dry cough, and wheezing. The patient feels a sense of anxiety and oppression; the breathing is difficult and characterized by a long whistling expiration. The patient jumps up and assumes the sitting posture, seizes hold of the top or side of the bed in order to support himself and give the respiratory muscles assistance. The breathing is slow and difficult, inspiration is short, expiration much prolonged and accompanied by loud sibilant and sonorous rhonchi. The chest is fixed in the position of extreme inspiratory distension, the sterno-mastoids stand out prominently, the supraclavicular fossæ recede during inspiration, the diaphragm is lowered to the lowest possible extent, and there is marked pulsation in the epigastrium. The patient's panting and gasping are audible at some distance. His face becomes cyanotic and swollen, tears flow from his eyes, a profuse sweat breaks out on his skin, and his whole aspect is one of anxiety and distress. During the attack the percussion note is hyper-resonant, cardiac and hepatic dulness are obliterated, and posteriorly pulmonary resonance may extend as low as the twelfth rib. Oxygen may be quite replaced by CO_2 in the expired air. The vesicular murmur is feeble or absent. The pulse is small and feeble, and may intermit with inspiration (*pulsus paradoxus*). The patient

has a sense of intense air-hunger, and sometimes, even in winter, rushes to the window and flings it open.

The duration of an attack varies from one to five or six hours. The attack subsides gradually. The whistling and gasping sounds subside, and are succeeded by moist râles. Cough occurs; secretion increases; pellets of semi-transparent mucus, compared to boiled tapioca and containing Curschmann's spirals and Charcot-Leyden crystals, are expectorated; the sense of anxiety and oppression gradually disappears; the pulse improves; and the patient passes into a stage of comparative comfort, but the breathing may remain laboured for some time. Often he is well enough on the day after his attack to resume his occupation. In less favourable cases the attacks succeed each other with only short intervals, and the patient may remain for days or weeks in a condition of almost continuous distress. As regards the frequency of the attacks, in the ordinary type of case no rule can be laid down. They may occur almost nightly, or only once or twice in the year. A 'larval' form of asthmatic attack has been described, characterized by the symptoms of a sudden 'cold in the head' and persistent sneezing and abundant flow of mucus. That these attacks are really abortive asthmatic paroxysms seems probable from the fact that they sometimes replace the regular attack in asthmatic patients. In certain cases inspiration is as laboured as expiration. Sometimes grave nervous phenomena attend the asthmatic paroxysm; the patient may even lose consciousness, fall down, or have a regular epileptic convulsion. Spasm of the glottis is sometimes observed. The amount of catarrh is variable: sometimes very profuse, at other times moderate or scanty. In children the asthmatic attack sometimes sets in with symptoms resembling those of broncho-pneumonia, but the attack passes off in a day or two. In other cases the simulation of whooping-cough is for a time close.

HAY ASTHMA.—This is a special form of asthma, and its relationship to the ordinary type is doubtful. It is characterized by attacks of paroxysmal dyspnœa not distinguishable from those of ordinary asthma, but the exciting cause is known to be invariably the pollen of certain grasses. In England it is the hay, in Germany the blossom of the rye, which brings on attacks. In America the *Ambrosia artemisiæfolia* is specially feared. In some cases of hay asthma catarrhal symptoms—coryza, conjunctivitis, and bronchitis—predominate; in others the symptoms are more typically asthmatic—viz., expiratory dyspnœa, sibilant râles, and distension of the lungs. Attacks of hay asthma are usually limited to the summer months, and are often much under the influence of locality. Hay asthma is in many cases a nasal asthma, and it is common to find in these patients various abnormalities of the naso-pharynx, such as polypi, hypertrophy of the mucous membrane, and other allied conditions.

THE SPUTUM IN ASTHMA.—Expectoration usually appears only towards the end of the asthmatic paroxysm. It varies much in amount, the variations having relation to the frequency and duration of the attacks. It is greyish-white in colour, and consists of thick tenacious mucus, bearing on the surface a white frothy stratum, resembling white of egg. Curschmann's spirals and Charcot-Leyden crystals are usually present. The sputum has also been found to contain calcium oxalate, pigment cells, eosinophile cells, and traces of blood.

DIAGNOSIS OF ASTHMA.—This is usually easy. The paroxysmal character of the attacks, the expiratory type of dyspnœa present, the wheezing râles and rhonchi, the severity and brief duration of the attacks, the characters of the sputum, the comparative absence of symptoms between the attacks, the slight nature of the physical signs, are, in general, distinctive. The following conditions, however, may require to be considered before making a diagnosis:

(*a*) Laryngeal obstruction, depending on such causes as spasm of the glottis, œdema of the glottis, paralysis of the posterior crico-arytænoid muscles, stenosis of the larynx, foreign body in the larynx.

(*b*) Tracheal obstruction.

(*c*) Chronic bronchitis and emphysema.

(*d*) Cardiac dyspnœa.

(*e*) Uræmic dyspnœa.

(*f*) Aortic aneurysm.

(*g*) Hysterical spasm of the diaphragm.

(*h*) Enlargement of the bronchial glands.

(*i*) Tumours of the medulla oblongata and upper part of the spinal cord.

All forms of obstruction of the upper air passages, larynx, and trachea can be readily distinguished from asthma by attention to the type of dyspnœa present. In the former it is inspiratory, in the latter expiratory. In the former we have stridor, in the latter wheezing. Spasm of the glottis is the only one of the above conditions which commonly gives rise to any real difficulty, and in this case the paroxysm is very brief.

Chronic bronchitis and emphysema are often accompanied by attacks of spasmodic dyspnœa, which may simulate or even merge in genuine asthma. The distinction rests chiefly on the history, the paroxysmal character or otherwise of the attacks, and the condition of the patient between the attacks. The spirometer will probably show that the capacity of the lungs is reduced in emphysema, normal in asthma between the attacks. Lastly, the characters of the sputum may decide the diagnosis.

Cardiac dyspnœa does not usually cause any difficulty. It is not markedly paroxysmal, the breathing is sighing or panting in character, and the physical signs of cardiac lesion are present.

Uræmic dyspnœa may be either inspiratory or expiratory

in type, or the breathing may be simply hurried, sometimes noisy and stridulous, sometimes of the Cheyne-Stokes type. The history, the aspect of the patient, and the presence of albumin in the urine assist the diagnosis.

Aortic aneurysm may, by pressure on the vagi, trachea, or left bronchus, cause attacks of an asthmatic type. The paroxysms in these cases are apt to be severe and prolonged, not much under the influence of remedies, and there will be the history and physical signs of aneurysm.

Hysterical spasm of the diaphragm is a very rare condition, of which I have no personal experience. It is said to be characterized by a short 'snapping' inspiration with a brief bulging of the epigastrium, followed by an expiration unaccompanied by râles.

Enlargement of the bronchial glands causes a dry, irritative cough, sometimes stridor; the dyspnœa is not decidedly paroxysmal; dulness may be present between the shoulder-blades; and the patients are often obviously tubercular.

Tumours of the medulla oblongata and upper part of the spinal cord cause a variety of dyspnœa which is definitely inspiratory in type.

PROGNOSIS IN ASTHMA.—The danger in the paroxysmal stage is very small. Death during a paroxysm is practically unknown. In young patients, where there is no serious involvement of the lung, emphysematous or otherwise, the outlook is fairly favourable, a certain proportion getting quite well (this is, however, exceptional), and others improving decidedly under treatment. Fully developed asthma in adults is rarely cured, but can often be held in check by treatment. The disease has not much tendency to shorten life, but asthmatics do not on the average attain the full term of life. The patient's mode of life, occupation, and prospects of careful treatment are important. Much emphysema, involvement of the right heart, or decided nervous debility are very unfavourable.

Treatment of Asthma.—The treatment of asthma involves many difficult problems, and has been one of the favourite fields of the quack. It is desirable that, before we undertake the management of a case, we should make up our minds what may be fairly expected from treatment, and what principles are to guide our therapeutic measures. Nothing can be more unscientific than the reckless prescription of powerful narcotic remedies, which give temporary relief at the cost, too often, of the ruin of the patient's nervous and digestive systems, and of evils as great as, or greater than, the asthmatic paroxysms. To order in any routine fashion the regular use of such remedies as stramonium, chloral, morphine, amyl nitrite, lobelia, belladonna, cannabis indica, hyoscine, bromides, or chloroform, and stop short with such medication, is to misunderstand the case completely. Some of these remedies are most valuable, and most of them will generally find an occasional place in our therapeutic armamentarium, but their employment simply to control symptoms is usually illusory, and all these remedies are capable of doing harm. We have first to ask the fundamental question of all therapeutics, Can we discover and remove the cause of the asthmatic paroxysms in any individual case? If any cause of reflex irritation—nasal, digestive, uterine, or urinary—can be discovered, it should, of course, be removed. But the number of cases which admit of this kind of radical treatment is, in my experience, a small one. It is wise and reasonable to have any obvious abnormalities in the naso-pharynx corrected, but the physician learns by experience not to promise too brilliant results from such treatment. The discovery of the so-called 'asthma points' in the nasal cavities and their treatment by cauterization or by operative procedure may be approved, but the active treatment of a normal naso-pharynx in these cases, followed though it has been in some cases by benefit, seems to border on quackery. I agree with Hoffmann that 'the

results of the direct nasal treatment are often very slight, or while apparently successful for a time, they are often of very slight duration.'* A marked febrile reaction following the cauterization of 'asthma points' has been reported by Brügelmann.†

If we are right in assuming that by far the commonest cause of asthma is a bronchiolitis acting upon a hypersensitive respiratory centre—and this I take to be at least a highly probable view—two indications are clear: (*a*) to obviate causes which tend to produce bronchial catarrh, and (*b*) to brace up the general nervous system. I believe excellent results may be obtained by attention to these two cardinal indications. As regards the former point the patient should, if possible, reside in a fairly sheltered locality, protected from harsh winds, and preferably not at the seaside. It has long been remarked that asthmatics often do well in the centre of large smoke-laden cities, and the assumption has usually been that a smoky atmosphere has some mysterious virtue. It seems not improbable that the element of shelter has a good deal to do with the good effect of such localities. Many asthmatics do well in regions where there is a good deal of wood, and where the soil is dry and sandy—for example, Ascot, Aldershot, Bournemouth. Care about undue exposure, warm clothing, and attention to nasal breathing are important.

Not less vital is the second indication, viz., to brace up the general nervous system, and we must be on our guard, lest in safeguarding the first indication we sacrifice the second. The patient, especially if young, should be subjected to a regular course of hydrotherapy, respiratory gymnastics, calisthenics, and outdoor exercises and games. It would be impossible to lay down rules to suit all cases. The prac-

* 'Nothnagel's Encyclopædia of Practical Medicine,' English edition, article 'Asthma.'

† *Ibid.*

titioner must take the measure of his case, and be careful not to err either by slackness or over-activity. The daily sponging of the body with tepid or nearly cold water, followed by vigorous friction with a rough towel, will nearly always be practicable, and in some cases cold affusion may be safely tried. Respiratory gymnastics can always be practised, the patient being taught to take long deep inspirations with the mouth closed, and then expire as fully as possible, with or without dumb-bells in the hands. The object of this manœuvre is not only to change the residual air as much as possible, but also to discipline the respiratory centre in the medulla. The ordinary exercises of the gymnasium, the Swedish manual exercises, and various games might be utilized to attain the ends in view. It need hardly be said that if much emphysema or any dilatation of the right heart is present, all forms of active exercise will require either to be strictly limited or altogether forbidden. In general these methods are suitable only for young patients, and for uncomplicated asthma. Much will depend on the discretion with which such methods are applied to cases really likely to benefit by them.

How far some form of hypnotic suggestion or psychotherapy can be made available in cases of asthma, it is difficult to say. There seems little doubt that suggestion plays a part in precipitating attacks, and might be used to prevent them. In special institutions such modes of treatment, in careful hands, would probably produce considerable results.

The diet requires to be regulated on two principles, first, a sufficient amount of nourishing material to promote a maximum of nervous and general energy; and, secondly, the avoidance of indigestible articles and of late suppers, which might cause reflex irritation and precipitate a nocturnal attack. Alcohol and coffee are to be used very sparingly. Diet cures have been advocated from time to time, and

remarkable results have been claimed for them. A very sparing diet of the ordinary type has been said to prove efficacious in some cases; in others, an exclusively milk diet or vegetarianism or a sugar-free diet has been found useful. I have not seen anything to incline me to put the dietetic treatment of asthma in the first rank of therapeutic measures, but it is not to be entirely overlooked, and if regulated with due regard to the patient's weight, digestive capacity, general nutrition, and habits may strengthen the general therapeutic programme. A radical change of diet has much effect upon the nervous system as well as on the whole organism, and its influence upon certain cases of asthma would not be surprising.

Climatic change may often be thought of in asthma, but there are few more embarrassing problems than to be asked to select a health-resort for an asthmatic. The disease is so wayward and erratic, so little under the influence of meteorological conditions, that the choice has to be determined largely by the known peculiarities of the individual case. Nevertheless, we are not entirely without guidance in this difficult matter. Where there is much bronchial catarrh present a warm, dry, fairly sheltered resort will usually be found best. The presence of much emphysema or of cardiac complications would suggest similar indications. In young patients with little emphysema and sound hearts mountain air up to 4,000 feet or higher has been found advantageous. Mont Dore has a special repute for asthma. Some cases have even been found to do well at so elevated a resort as St. Moritz. The sea air is, on the whole, unsuitable, but Curschmann reports good results on the Riviera.

The medicinal treatment of asthma is important. By far the most important drug is iodide of potash, from the use of which I have repeatedly seen brilliant results. It must be given with a free hand—5 to 20 grains thrice daily. Iodism has been in my experience quite rare. The best additions to

the iodide mixture are, I think, moderate doses of Fowler's solution or ethereal tincture of lobelia. These latter drugs are not very effectual when given without the iodide of potash, but I have little doubt that they are valuable adjuvants. During the last few years I have used lobelia more frequently than formerly, and I think my results are better. I cannot agree with those writers who regard the influence of lobelia as simply psychical. Nux vomica has been found very useful in cases where attacks of asthma are apt to come on when the patient is run down and debilitated. Stramonium, which has supreme value in combating the actual asthmatic paroxysm, has also virtue in warding off attacks, but the regular use of this drug is to be deprecated. Its effects upon the nervous system are, in the long run, disastrous. Grindelia robusta in doses of 10 to 30 minims of the liquid extract is worthy of an occasional trial, but is not to be relied upon. I have seen one case where caffeine seemed to exercise specific virtues. Leyden and Curschmann advise inhalations of common salt and carbonate of soda.

For cutting short a paroxysm the chief remedies are stramonium, either in the form of cigarettes or of fumes from burning powder; the fumes of nitrate paper; chloral; morphia by hypodermic injection; heroin in doses of $\frac{1}{12}$ to $\frac{1}{6}$ grain of the hydrochloride hypodermically. The inhalation of ammonia relieves some patients. Amyl nitrite is less useful than the above, and chloroform inhalations should seldom be necessary.

It is imperative to remember that the powerful nervine remedies, especially stramonium, which have so much influence upon asthma, are disastrous to the nervous and digestive systems if long persevered with. I have seen more than one patient whose asthma was 'cured' at the cost of the ruin of his general health.

LECTURE XIII

THE CAUSES OF PULMONARY TUBERCULOSIS

SUMMARY:

General pathology of tuberculosis.

Modes of entry of the bacillus of tubercle into the human body.

Question of infection in pulmonary tuberculosis:

Hereditary influences.

Transmission by inoculation.

Transmission by inhalation of particles of sputum.

Transmission through the agency of food—milk, meat, bread.

Conditions which favour infection by the bacillus of tubercle:

Hereditary predisposition.

Influence of unhealthy occupations.

Influence of insanitary dwellings, schools, etc.

Influence of the food-supply.

Influence of frequent child-bearing.

Influence of alcoholism.

Influence of antecedent disease.

Influence of malformations of the thorax.

Influence of care, anxiety, prolonged nursing.

Influence of traumatism.

Influence of exposure.

The discovery in 1882 by Robert Koch of the bacillus of tuberculosis is one of the great landmarks of medicine. It not merely rendered at once obsolete all the former contro-

versies regarding the unity or diversity of the various forms of pulmonary tuberculosis, but gave us invaluable aid in diagnosis and a fresh standpoint for prevention and treatment. The remarkable diminution in the tuberculosis rate which has taken place in nearly all civilized countries in the last two decades must be attributed not only to a general improvement in the standard of living, but to the more enlightened view of the disease and of its prevention and treatment, which is the direct fruit of Koch's great discovery.

Henceforth all questions regarding the causation of tuberculosis may be considered under three heads—viz. :

1. The natural history of the *Bacillus tuberculosis.*
2. Its modes of entry into the human organism.
3. The conditions which increase or diminish susceptibility to infection.

The Bacillus Tuberculosis (Koch).—This organism, which belongs to the class of the schizomycetes, is a thin rod-shaped organism, often slightly bent, measuring from 2 to 4 μ in length and about 0·4 μ in breadth. About 300 to 400 of these organisms placed in a row would give the length of 1 millimetre. When stained, it often presents a beaded appearance, which has been attributed to the presence of spores, but which rather seems to be due to the presence of vacuoles depending on degeneration. The range of temperature suited to the bacillus is between 30° and 40° C., the most active changes taking place at the temperature of the blood, 37° C. The rarity of the disease in cold-blooded animals is thus easily understood. The disease is common in bovines; rare in horses, dogs, and cats; rare in rabbits and guinea-pigs in the natural state, but these animals are very susceptible to inoculation; unknown amongst the monkey tribe in the wild state, but common and destructive to them when in captivity. The disease, though very widespread amongst the human family, does not seem to be ubiquitous. It is especially the disease of civilized com-

munities and of settled domesticated life. It would seem to have been very rare, if not unknown, amongst some of the negro tribes of Central Africa, the North American Indians, and the inhabitants of the Polynesian Islands, before these peoples came into close contact with civilized nations. Nomadic peoples, like the Bedawîn or the inhabitants of the Central Asian steppes, are still comparatively exempt. When such peoples relinquish their nomadic, open-air life and become city dwellers, they are at once attacked in large numbers. These facts, and the before-mentioned effects of domestication upon certain animals such as the monkey tribe, have important inferences as regards prevention and treatment. The bacilli are present in all recent and rapidly-growing tuberculous lesions, the number as a rule bearing a relation to the activity of the process. In obsolete or caseated lesions and in fibrosed lung tissue it may be impossible to demonstrate their presence, except by inoculation, and even this test may fail. They are present in the blood in many cases. Septic organisms are usually present in association with the tubercle bacillus, and are responsible for many of the most characteristic symptoms of pulmonary tuberculosis. The opinion has more and more gained ground that the disease in its ordinary forms is essentially a mixed infection. It is even probable that the *Bacillus tuberculosis*, when not accompanied by septic organisms, is in a large proportion of cases comparatively innocuous. The bacilli are usually present in large numbers in the sputum of patients suffering from pulmonary tubercle, and in general, when the number of bacilli is large, the process is probably active.

The bacilli give rise to tubercles, which consist of epithelioid cells, giant cells, lymphocytes, and a fibrillary network. The further progress of the case depends in large measure upon the evolution of the tubercles—*i.e.*, whether they caseate and soften, or undergo fibroid or calcareous change, and whether they cause much or little local in-

flammatory reaction. For further pathological details, which lie outside the scope of these lectures, you are referred to the special manuals of bacteriology.

Modes of Entry of the Bacillus of Tubercle into the Human Body.

We have to consider the possibility of the following modes of infection:

(1) By hereditary transmission.

(2) By inoculation.

(3) By inhalation of fine divided particles of sputum or of dust containing bacilli.

(4) By infection through milk.

(5) By infection through meat.

It is impossible for me to do justice to the enormous literature of the subject. I must content myself with outlining the various views held, and indicating the direction in which the weight of evidence seems to me to tend. There is no doubt that much remains to be done before the subject is finally removed from the region of controversy. Let us first of all consider the broad question of infection before entering into a discussion of its modes.

From time immemorial an impression has prevailed in some countries that pulmonary tuberculosis is infectious, but, on the other hand, direct proof of infection is usually wanting, and many practitioners go through a long professional life without seeing any conclusive case, while nothing is more familiar than the fact that the closest domestic relations may exist between the tuberculous and healthy relatives without the latter becoming affected. I believe that scepticism regarding the infectiousness of tuberculosis largely arises from the fact that the practitioner has before his mind the misleading analogy of the infective fevers, and that he fails to observe cases of infection, because these cases do not

conform to his preconceived standard of what infectivity may be expected to involve. If we assume, hypothetically, that tuberculosis is a disease of a low grade of infectivity and probably of a long incubation stage; that its infectivity is much under the influence of predisposing conditions, such as overcrowding, bad ventilation, dirt, poverty, improper dietary; that individual proclivity to infection has considerable weight—I think we shall have no difficulty in reconciling the facts of clinical experience with the theory of infection. The following passage from Lenhartz puts this position clearly and well: 'For the careful observer the cases of the transmission of the disease from one individual to another are not at all rare. Sometimes we see that in a family free from tuberculosis the disease is introduced by the return home of a tuberculous member of the family circle. Especially amongst peoples who live in close domestic relationship are such cases observed, and the result is sometimes that in the long run the whole family perishes from tuberculosis. It also frequently happens that when a husband or wife dies of tuberculosis the survivor becomes subsequently affected, perhaps after the lapse of years, although nothing in the hereditary or constitutional conditions made such a development probable. Experience shows that the wife is oftener infected from the husband than the husband from the wife, probably because the wife on the average oftener devotes herself to the care of the sick husband than *vice versâ*. In localities where a tubercular individual has dwelt infection long abides. Many cases show that families formerly free from tuberculosis often become infected when they take possession of a house where tuberculous patients have lived. Also, it not seldom happens that a second wife introduced into a household where the former wife has died of tuberculosis herself falls a victim to the disease. Finally, cases are known where tradesmen of various kinds, especially tailors, have gone to health resorts

for tuberculosis for reasons of trade, and have returned infected with the disease.'*

The cases which have come under my own observation of families becoming the subjects of tuberculosis after taking up their residence in infected houses, and the not less striking cases where families after losing several of their members from tuberculosis in one house have remained free from the disease on removing to another, seem to me to admit of only one explanation. Family practitioners who have special opportunities of observing facts such as these would do good service to the cause of medicine by recording and publishing them. I am strongly of opinion that what may be fairly termed 'house infection' is one of the fundamental facts, perhaps *the* fundamental fact, in connection with the spread of pulmonary tuberculosis. Nor is this view shaken by the testimony of many heads of consumptive hospitals and sanatoria that tuberculosis is not more frequent amongst the servants, attendants, and nurses in such institutions than in institutions concerned with other diseases. Dr. Turban, of Davos, for example, affirmed in 1899 that of 207 healthy individuals introduced into his sanatorium not one contracted tuberculosis. Dr. Dettweiler had a similar experience at Falkenstein, and the reports from the Brompton Hospital point in the same direction. Such evidence is open to two criticisms—first, that in many cases the ultimate history of the nurses and attendants in consumptive sanatoria is not known; and, secondly, that even if infection is rare in well-managed, scrupulously clean, and well-ventilated institutions, it by no means follows that infection is equally rare in dirty, ill-ventilated houses, and amongst careless people. Important evidence can be quoted in opposition to the views given above. Thus, Cornet reports that an investigation made into the causes of death in the Prussian

* Lenhartz in Ebstein and Schwalbe's 'Handbuch der Praktischen Medizin,' Bd. i. 1, pp. 279, 280.

Catholic Orders, extending over twenty-five years and embracing 74,306 persons, brought out the fact that tuberculosis was the cause of death in more than two-thirds of the cases—a proportion far in excess of the general average.* It has often been asserted that at health resorts which have become frequented by consumptives no increase in the tuberculosis rate amongst the inhabitants can be observed, but Cornet has shown that this is a very doubtful assertion.†

As regards the mode of infection, we have to consider—

1. HEREDITARY TRANSMISSION.—Influenced by a one-sided view of the infective doctrine, many physicians have ceased to regard pulmonary tuberculosis as in any true sense hereditary. Now, there is no proof of the transmission of tuberculosis by the sperm; very little proof of its transmission by the ovum; and conflicting evidence as regards its transmission through the channel of the placental blood. Baumgarten and his followers hold that tubercle bacilli are frequently transmitted by direct inheritance, and that they lie latent until a later period of life, owing to the greater resisting power of the tissues of children. This view has not met with general acceptance, and on many grounds it seems to me improbable. That the young tissues of the infant should be specially resistant to the influence of tubercle bacilli seems to me a most improbable hypothesis. But, if we cannot accept Baumgarten's doctrine, and if the direct proof from the pathological standpoint of the transmission of tuberculosis from parent to offspring is weak, we must not, therefore, ignore the clinical fact that *pulmonary tuberculosis runs strongly in families*. To deny this proposition is to run counter to one of the most familiar facts of experience. But to accept the fact is one thing, to

* G. Cornet, 'Nothnagel's Encyclopædia of Practical Medicine,' English edition, 'Tuberculosis,' p. 272.

† *Ibid.*, pp. 274, 275.

interpret it correctly is another. How far direct inheritance of the germ of tubercle is the fundamental fact in these cases, how far an inherited proclivity to infection, how far simply contiguity in association with house infection, it is very difficult to say. We certainly need further evidence on these points. But to assert that pulmonary tuberculosis is simply an infectious disease, and that inheritance has no influence, is to go beyond the evidence. One of the most recent inquirers into this matter is A. Riffel, who has subjected the question to an exhaustive examination. His plan was to take certain groups of families and follow their health history through several generations. His conclusion is that phthisis and other tubercular affections arise especially, and almost exclusively, in certain families; that tuberculosis is propagated from generation to generation, sometimes missing a generation, and that specially affected families tend to die out.* It is quite open to argument that the two facts underlying these phenomena are (1) an inherited proclivity to infection; and (2) contiguity in infected houses. I am inclined to attribute great weight to both these factors. Facts have come under my notice which lend strong support to the view that families living under the same roof, but with different inherited tendencies, respond in the most opposite ways to tubercular infection, the virus being deadly to one family, and almost, or quite, innocuous to the other. These facts have to be reckoned with before we are justified in saying that pulmonary tuberculosis is not a hereditary disease. The assertion might be, in strictness, correct; the inferences from it might be misleading.

2. Transmission by Inoculation.—Such cases undoubtedly occur, but they are rare and of little practical importance. They have been known to arise in connection with dissection wounds, circumcision, and vaccination. With

* Ebstein and Schwalbe's 'Handbuch der Praktischen Medizin,' Bd. i. 1., p. 281.

the most ordinary precautions, transmission by this mode ought to be almost impossible.

3. TRANSMISSION BY INHALATION OF FINELY-DIVIDED PARTICLES OF SPUTUM OR OF DUST CONTAINING BACILLI.—In the opinion of a large majority of authorities, this is the usual mode in which pulmonary tuberculosis is contracted. It is true the expired air of tuberculous patients contains no tubercle bacilli, but on coughing or sneezing the finely-divided sputum may contain these organisms. More important still is the fact that the dried sputum, whether on handkerchiefs, floors, lips, or beard, contains bacilli. The proofs that dried sputum from tubercular patients is the chief source of the dissemination of the disease have been fully set forth by Cornet, and seem to me very strong. They have not, however, won universal acceptance. Flügge thinks the finely-divided sputum is the real source of danger, and Von Behring, whose views will be considered more fully presently, regards milk as the main agent of infection. Volland thinks that the lungs are invaded from the bronchial glands and not *per viam respirationis*. Others have supposed that the bacilli gain access from the mouth, nose, and other parts directly to the blood, and are conveyed by it to the lungs. It is not my purpose to attempt to summarize all the pathological arguments which can be adduced on behalf of the inspiration theory of the transmission of pulmonary tuberculosis; it must suffice to say that the theory would explain the phenomena, and that there are no clinical facts known to me which contravene it. Cases where the occupation of infected apartments has led to the development of the disease—and such cases are, in my judgment, of frequent occurrence—are easily explained on the dust-borne theory, and the good effects of cleanliness upon the prevention of the disease are thus also easily understood. The fact, too, cannot be controverted that bacilli are frequently found in the dust of the walls of apartments which have been occupied by tubercular patients.

There are grounds for thinking it probable that in young children the bacilli in a large proportion of cases make their way through the mucous membrane of the pulmonary alveoli and are carried by the lymph-stream to the bronchial glands. It is certain that in these patients the bronchial glands are often primarily attacked, and that in other cases the distribution of the lesion in the lung would suggest that invasion had taken place by way of the bronchi. In adults, on the other hand, owing to the firmer tissue of the alveolar mucous membrane, the smaller calibre of the lymph channels, and the blocking of many lymph channels with particles of coal, stone, or dust, the bacilli cannot make their way through the alveolar mucous membrane and along the lymph channels, and hence develop *in situ*. As proliferation of the bacilli in the alveoli goes on, a certain number of the micro-organisms make their way into the lymph channels and secondarily involve the bronchial glands.* The view that inhalation infection plays a relatively small part in the tuberculosis of childhood does not seem well founded. 'Rauchfass observed that nine-tenths of the tuberculous children in the Foundlings Asylum of St. Petersburg suffered from disease of the lungs and bronchi, and Klein found a similar proportion in Moscow. Flesh found that the bronchial glands were intensely affected in a third of all cases of tuberculosis in childhood. The lungs also invariably presented either isolated or confluent tubercles, although the changes here were of much more recent date than in the bronchial glands.'†

Von Behring controverts the foregoing views at almost every point, although not denying the possibility of infection by inhalation of particles of dust or moisture containing tubercle bacilli. He is of opinion that infection takes place almost invariably in infancy, and mainly through the agency of milk, and that persons who develop phthisis in after life have had a quiescent tubercular focus in the lungs dating

* Cornet, *loc. cit.* † *Ibid.*

from infancy. In his view the chronicity of ordinary phthisis is explicable on the hypothesis that the individual thus affected is partially immunized by a tubercular attack in infancy. 'Were we to inject into the tissue juices of a person not yet partially immunized against tuberculosis an amount of tubercle bacilli equal to that usually found in the lungs of consumptives, the person would die of an acute miliary tuberculosis, but he would never develop pulmonary consumption.'* Von Behring further maintains that while tubercle bacilli may be inhaled, there is no positive proof that they ever reach the lungs. We shall resume the consideration of Von Behring's views in the next section.

The inhalation view of the origin of pulmonary tuberculosis has passed into ordinary medical theory. There can be little doubt that it is adequate to explain the facts, and I am not convinced that the objections which have been urged against it possess any real validity.

4. Transmission by Infection through Milk.—According to Von Behring, this is the usual method—'The milk fed to infants is the chief cause of consumption.' He founds his view largely upon experiments of Römer and others, which go to prove that the intestinal mucous membrane of the young of certain animals is permeable to true albumins and to bacteria. This was found to be true of anthrax bacilli and of tubercle bacilli. 'In the results of all these investigations,' he says, 'I see experimental support for the view I have for some time maintained—namely, that the origin of the epidemiological pulmonary tuberculosis in man, and that of the epizoötic pulmonary tuberculosis in cattle, is a primary intestinal infection occurring in very early infancy. . . .† It may be months, years, or decades before the infection leads to manifest disease. This depends on the

* E. Von Behring, 'Suppression of Tuberculosis,' translated by C. Bolduan, p. 28.

† *Ibid.*, p. 35.

virulence of the virus, which is generally much greater in the virus of bovine tuberculosis than in that of human tuberculosis. It also depends on the number of bacilli introduced per stomach, and whether such introduction is single or oft repeated. In the human being months and years may elapse before the infection is followed by any sensitiveness to tuberculin injections in the usual dose. If, then, at the time of puberty, or after an exhausting puerperium; after too great a demand on the milk secretion (especially with insufficient food); after so-called colds and other unfavourable meteorological conditions; after muscular over-exertion, under conditions unfavourable to life, such as improper nourishment, confinement in insufficiently or badly-ventilated rooms, etc.—if after any of these pulmonary disease develops whose tubercular nature we cannot doubt, then we are dealing with *the beginning of consumption; the beginning of tubercular lesions* is much farther back; and the first introduction of the disease germs—in other words, *the beginning of the infection*—is far back in earliest infancy.'* The same authority holds that tubercular infection in adolescence or adult life is quite rare, and that the preventive treatment of tuberculosis has to concern itself chiefly with the quality of the milk-supply of infants.

When we recollect that in 1901 Koch informed the British Congress of Tuberculosis that bovine and human tuberculosis were specifically distinct, that the human subject was never infected from milk or meat, and that precautions regarding the use of these articles was unnecessary, we see how far we are from agreement on the pathological side—how diametrically opposed, in fact, are the views of the leading experimental pathologists on this vital question. Since Koch's pronouncement in 1901 a host of observers, including Delépine, Ravenal, Arloing, De Jong, Prettner, Thomassen, Wolff, Hamilton, and Young, have claimed to have succeeded in infecting cattle with bacilli from human sources. The discussion of patho-

* E. Von Behring, *op. cit*, pp. 40, 41.

logical controversies lies outside the scope of these lectures, so I must content myself with a mere statement of the conflicting views. But we may fairly ask, when pathological opinion is in a state of such acute antagonism, whether clinical experience can throw any light on this all-important matter. In discussing this matter, we must not forget that milk might easily infect the tonsils, and that the entrance of the disease through this portal is believed by many observers to be common. Against the view that milk is the chief source of tubercular infection we have the following facts:

(*a*) The rarity of primary tuberculosis of the bowel. In 3,104 autopsies in cases of tuberculosis in children at the Charité Hospital, Berlin, there were only 16 cases of primary bowel affection.

(*b*) The strong evidence in favour of 'house infection' in tuberculosis.

(*c*) The generally accepted belief that tuberculosis of the lungs is decidedly more frequent during adolescence than in infancy and early childhood.

(*d*) The absence of proof that infantile tuberculosis is rarer during infancy in countries—*e.g.*, Japan—where milk forms a less indispensable element in the nutrition of infants than it does in European countries.

(*e*) The absence of proof that boiling of milk, which is the rule in many countries, such as France and Germany, diminishes the amount of infantile tuberculosis as compared with countries—*e.g.*, England—where boiling of milk is unusual. *Ex hypothesi*, a contrast would be expected.

It would be wrong to dogmatize on some of these points, regarding which our knowledge is by no means complete; but it will be evident that clinical experience, on the whole, fails to support Von Behring's view, and raises many difficulties in the way of its acceptance.

5. Transmission by Means of Tuberculous Meat.—Much difference of opinion exists on this subject. It is, of

course, certain that the meat of tuberculous cattle is constantly offered for sale; but comparative safety is probably secured by the facts that meat inspection is now practised in all civilized countries, that the parts most used for food are little affected by tuberculosis, and that the ordinary methods of cooking go far to obviate danger.

6. Transmission by Means of Bread.—This is a possible source of infection. Many bakers are tubercular.

Conditions which Favour Infection by the Bacillus of Tubercle.

The following are the chief of these conditions:

(*a*) Hereditary predisposition.

(*b*) Unhealthy occupations.

(*c*) Small, ill-ventilated, insanitary houses.

(*d*) Insanitary schools.

(*e*) Improper nourishment.

(*f*) Frequent child-bearing.

(*g*) Intemperance.

(*h*) Co-existing or antecedent diseases—measles, whooping-cough, influenza, broncho-pneumonia, bronchitis, pleurisy, cancer, diabetes, typhoid fever.

(*i*) Malformations of the thorax.

(*j*) Congenital small heart and narrowing of the pulmonary arteries.

(*k*) Care, anxiety, prolonged nursing.

(*l*) Trauma.

(*m*) Exposure.

Our limits will only permit a somewhat summary discussion of the above causes, which will again engage our attention when we come to deal with the prevention of tuberculosis.

On the question of hereditary predisposition much differ-

ence of opinion exists. The revolt from the old view of the essentially hereditary character of tuberculosis has, I think, been too complete. That the disease *runs strongly in families* is, as I have already insisted, absolutely certain, however we may explain the fact My own view is that there are probably two factors at work—viz., increased proclivity to infection and increased exposure to contagion. It is a familiar fact of medical experience that as regards the various infectious fevers the most varying degrees of tendency to be infected, or to escape, are found in different families. In one family most of the members, as the phrase goes, 'take everything that is going.' In other families infection is the rare exception, in spite of repeated exposure. Why should not similar differences be found as regards tuberculosis? This view is much strengthened when we find that a tendency to become easily infected with tuberculosis is often allied with certain inherited physical characteristics —viz., a certain shape of chest, peculiarities of hair, skin, etc.

The influence of unhealthy occupations in predisposing to tubercular infection is one of the most outstanding facts of the subject. The mortality from pulmonary tuberculosis, which is 108 per 1,000 amongst fishermen, and 118 per 1,000 amongst agricultural labourers, rises to 371 per 1,000 amongst cutlers, 433 per 1,000 amongst file-makers, 461 per 1,000 amongst printers, 473 per 1,000 amongst earthenware manufacturers, and 690 per 1,000 amongst Cornish miners.*

The unhealthiness of a trade seems to be essentially associated with the factors of impure, dust-laden air, overcrowding, bad ventilation, exposure to sudden alternations of temperature, and physical strain. Obviously, also, much will depend upon whether a trade is practised by persons on other grounds susceptible to infection. Thus the spinners in the Belfast flax-mills yield a high tubercular rate, partly from the nature of the trade, partly because it is so largely carried

* Newsholme, 'Vital Statistics,' p. 163.

on by young women, who are likely to become easily infected.

The influence of small, ill-ventilated, insanitary houses, with the concomitant element of overcrowding, is without doubt one of the most potent factors in predisposing to infection. Hirsch has shown that the rate of pulmonary tuberculosis rises with each increase in the density of the population. In Denmark, for example, the mortality from pulmonary tuberculosis is given as 2·1 per 1,000 inhabitants; in the twenty-four towns of medium size as 2·2 per 1,000; while in the five largest towns it is stated to be 2·6 per 1,000, and in the capital, Copenhagen, 3·0 per 1,000. In Holland the death-rate from pulmonary tuberculosis in the towns is to that in the open country as 21 to 16. In Switzerland, in the agricultural cantons, where the population is scanty, the death-rate from pulmonary tuberculosis is only 1·1 per 1,000, while in the mixed cantons (partly agricultural, partly industrial) it is 1·7, and in the purely industrial cantons 2·5 per 1,000.* Ransome has shown that in Manchester and Salford pulmonary tuberculosis prevails, especially in close courts and alleys, and in houses built back to back. In London, the investigations of Beevor and Shirley Murphy have proved that there is a direct relation between overcrowding and the tubercular rate. This law has become universally recognised, and is the basis of much of the best preventive work. It is probable that overcrowding acts partly by lowering 'resisting power,' and partly by multiplying opportunities for infection. Overcrowding is, of course, not an isolated fact; it is a visible sign of straitened circumstances, of poverty, struggle, and privation. We find that where the standard of living is low—*i.e.*, where there is a large percentage of people living in unhealthy houses, eating insufficient or unwholesome food, and following unwholesome

* Hirsch. These figures are not recent, and the present day condition is probably much better than they would suggest.

trades—the tubercular rate is high; and that where these conditions are reversed, it is relatively low. The tubercular rate is, in fact, a rough index of the well-being of a civilized community, and it cannot be too much borne in mind, a fact to which we shall return in dealing with the prevention of the disease, that no measures for stamping out tuberculosis are likely to be successful unless they are devised with the object of raising the general social and moral level of the community.

This consideration lends weight and interest to the fight against tuberculosis. It is a fight for the amelioration of social evils in general, and the betterment of society in some of its most essential relationships. It would be a matter of profound interest if a sanitary register of houses were kept in this country as has been attempted in Paris, as such a register would bring out in glaring light how pulmonary tubercle clings to houses and localities. Dr. Niven, of Oldham, has shown how in that city more than one death from the disease occurred in 10 per cent. of houses; that the chances of one house being twice affected accidentally were only 68, whereas 274 were thus affected; that the chances of one house being affected three times were only 7·6, whereas 24 were so affected. We shall return to this vital question when we come to deal with the prevention of tuberculosis.

The influence of insanitary schools in the spread of tubercular infection has not received the attention which the subject deserves, but public opinion is being gradually aroused on this question. There can be little doubt that crowded and ill-ventilated schools are a source of serious danger, the more so as cases of pulmonary tuberculosis so often escape detection for a considerable period. The condition of the primary schools in Ireland is in many cases deplorable, the buildings being unsuitable in point of construction, ill-ventilated, dirty, and imperfectly provided with

sanitary requisites and playgrounds. It is pleasant to be able to affirm that improvement in these respects is in progress, and that many of the new schools are in all respects excellent. Medical inspection of the children and the segregation of the tuberculous are not yet attempted, but the necessity for such measures is obvious.

The relation of the food-supply of the people to tubercular infection is important from two points of view, first, because ill-nourished people are likely to be easy subjects for infection; and, secondly, because tuberculous meat may be a source of danger. The former element is probably the more important of the two, but the latter is not to be ignored. There can be little doubt that the heavy incidence of tuberculosis amongst the agricultural population of Ireland finds some part of its explanation in the low standard of living, from the dietetic point of view, which prevails in that country. The rôle of dietetic causes in the ætiology of tuberculosis is, however, by no means clear. Ransome points out that in India the rice-eating Hindoo and the meat-eating Mahometan and Jew suffer from the disease in equal measure.* On the other hand, Dr. Lorand of Carlsbad has observed that with monkeys kept in captivity the tubercular rate is much lower amongst those which were able to take a meat diet than amongst those which were unable to do so,† while quite a number of observers have testified to the beneficial effects of a raw-meat dietary in phthisis. We must not forget that amongst the lower animals the herbivora suffer much, the carnivora little, from tuberculosis. I am not aware of any statistics showing the incidence of pulmonary tuberculosis amongst vegetarians. The disease is certainly much more prevalent amongst the classes which consume little, than amongst those who consume much, meat; but, then, it has to be borne in mind that ability to obtain

* A. Ransome, *op. cit.*, p. 90.

† *Lancet*, December 14, 1905.

meat freely is a sign of a relatively well-to-do class, and the question becomes complicated with problems of housing, habits and occupation.

The influence of too frequent child-bearing on liability to tubercular infection has been noted by all observers. As a general rule, pregnancy has some retarding influence on the progress of the disease, which tends to become more rapid after parturition. Tubercular women are, unfortunately, somewhat fertile.

The idea that alcoholism is in some way antagonistic to tubercular infection, which at one time prevailed, has been shown to be erroneous. Such a doctrine would be in sharp conflict with well-known and undisputed facts, *e.g.*, the tendency of malnutrition, exposure, irregular hours, poverty, to increase the liability to tuberculosis. The alcoholic suffer in large proportion from the disease, but apparently not more than any other class of ill-nourished and careless-living people. A history of alcoholism is not exceptionally frequent in cases of pulmonary tuberculosis.

The influence of antecedent or co-existing diseases in the causation of tuberculosis is potent. Amongst the diseases of children, measles and whooping-cough are the most important. To these may be added a disease often associated with the two foregoing maladies, viz., broncho-pneumonia. It is a question whether when, as so often happens, tuberculosis in children develops from broncho-pneumonia, what seemed a non-specific broncho-pneumonia may not have been really tubercular. I by no means affirm that this is always, or even usually, the case; only that the possible tubercular origin of broncho-pneumonia in children is not always sufficiently kept in view. We are somewhat apt to ask the question 'Is this child in danger of becoming tubercular?' without fully considering the prior question—'Is this child already the subject of tuberculosis?' Influenza also appears to be, in many cases, the starting-point of tuber-

culosis. We must, of course, discount many of the histories of this connection which are proffered to us, on the ground that what the patient terms 'an attack of influenza' is really the first premonition of tuberculosis. But, after making all deductions, the connection of influenza and tuberculosis would seem to be a real one. It does not appear, however, that the epidemic prevalence of influenza in recent years has tended to raise the tuberculosis rate, which has been steadily falling in most civilized countries. The relation of pleurisy to tuberculosis is a peculiar one, and has already been fully considered. Whatever be the correct explanation, the relation is certainly close. Bronchitis is usually regarded as a frequent predisposing cause of pulmonary tuberculosis. Its influence cannot be denied, but has probably been much over-rated. Broadly speaking, the bronchitic patient remains bronchitic, and does not become tubercular. Croupous pneumonia very rarely leads to tuberculosis of the lungs, but it would appear that the assertion that it never does so is somewhat too sweeping. Typhoid fever sometimes leads to pulmonary tuberculosis, a fact which is in no way surprising when we recollect that the disease is often prolonged and accompanied by much debility. Typhoid fever patients and tubercular cases should not be treated in the same hospital ward. Amongst chronic diseases, diabetes and cancer are notable for the frequency with which they are complicated by tuberculosis.

Malformations of the thorax are believed to predispose to tubercular invasion, and I see no reason to abandon this doctrine, which may, however, have been sometimes over-accentuated in the past. The general belief is that persons with flat, ill-developed, and imperfectly expansile chests are prone to pulmonary tuberculosis. West writes as follows on this subject: 'Whether this old belief be true is difficult to prove, but it may be partly true on the general principle that ill-formed organs are specially liable to disease. We

certainly know that even in phthisical families the chest is often perfectly formed; that, where it is ill-formed, many escape phthisis; and, lastly, that phthisis is common enough in persons whose chests are perfect in form, and even remarkably well developed. All that can be safely inferred, therefore, in respect both of constitution and malformation of thorax, is that frail and delicate persons with ill-formed chests are somewhat more liable than robust, well-developed persons to develop phthisis.'*

Beneke has brought forward facts to prove that persons with a congenitally small heart and proportionately narrow arteries are peculiarly liable to become tubercular. It should be borne in mind that Virchow has described similar congenital peculiarities in chlorotic girls, who do not tend to become tubercular in any preponderating number of cases.

Care, anxiety, prolonged nursing, and other such conditions, linked as they so often are with the factors of malnutrition and disregard of personal health, predispose to tubercular infection, as might be expected. The important effect of depressing mental conditions upon bodily function is noteworthy, and has hardly received sufficient attention.

Traumatism may predispose to tubercular invasion in various ways. It may cause abrasions of the skin, injury to bones and joints, injury to glands, or it may stir up a latent tubercular focus in, *e.g.*, the bronchial glands or the lungs. While many such cases are, undoubtedly, on record, it hardly appears that the influence of traumatism upon tubercular invasion is a considerable one.

Exposure is included amongst the predisposing causes of tuberculosis. Obviously, it may act in various ways: by giving rise to pleurisy, broncho-pneumonia, bronchitis, or croupous pneumonia; by lowering nutrition; by the influence of ground damp, and perhaps in other ways. We must beware of overrating this factor of exposure. Ransome

* S. West, 'Diseases of the Organs of Respiration,' vol. ii., p. 453.

thinks it rather antagonistic in its operation, pointing out how the poor fishermen of Iceland, the hunters and trappers of North America, the nomad tribes of Asia and Africa, even the wretched aborigines of Australia, suffer little from tuberculosis, while the relatively comfortable classes in the towns of these countries suffer much.*

* A Ransome, 'Causes and Prevention of Phthisis,' p. 50.

LECTURE XIV

THE CLINICAL HISTORY OF PULMONARY TUBERCULOSIS

SUMMARY:

Difficulty of forming any satisfactory classification of cases of phthisis.

Provisional classification:

A. Acute miliary tuberculosis of the lungs.

B. Acute caseous tuberculosis of the lungs (pneumonic phthisis).

C. Fibroid tuberculosis of the lungs (fibroid phthisis).

D. Fibro-caseous tuberculosis of the lungs (chronic pulmonary phthisis).

Acute miliary tuberculosis of the lungs:

Symptoms of the disease.

Physical signs.

Diagnosis.

Course and event.

Acute caseous tuberculosis of the lungs:

Broncho-pneumonic (disseminated) and lobar types.

Symptoms, physical signs, course and event.

Fibroid tuberculosis of the lungs:

General characters of the affection.

Fibro-caseous tuberculosis of the lungs:

Nature and progress of the lesions.

Modes of onset of the disease.

Symptoms: Cough, sputum, dyspnœa, hæmoptysis, hoarseness, pain in the chest, dysphagia, pyrexia, sweating, emaciation, diarrhœa, dyspepsia, fistula in ano, pityriasis versicolor, purpura.

The clinical history of pulmonary tuberculosis presents a most diversified picture. Any attempt, however, to divide cases of the disease into distinct classes must be regarded as, to a large extent, obsolete. The cause is always the same; the general tenor of the symptoms shows a variation in intensity rather than in character; the results in the lungs, amongst far-reaching differences, present a certain identity. Tubercular lungs become infiltrated with tubercle; the infiltration is usually local, and remains local, but in exceptional cases rapidly pervades large areas of the lungs. The inflammatory reaction of the pulmonary tissue may be slight, moderate, or marked, but is never absent. The tendency to caseate is always present. Fibrosis takes place in all cases which run a chronic course. Where such processes are present, either actually or potentially, in all tubercular lungs, it is evident that a classification of cases of pulmonary tuberculosis on a pathological basis would be quite illusory. As little can we suggest a satisfactory clinical basis for a good classification. Cases present every possible diversity as regards the severity of the symptoms and the rapidity of the course of the disease. But the attack which begins acutely does not always run an acute course; it is liable at any time to slacken in its development, and become subacute or even chronic. The chronic case may, after years of chronicity, assume the fulminating type, and the patient may die with all the symptoms of an acute malady; or a case may go through stages, now acute, and again chronic.

Nevertheless, for the purposes of study and description, and more particularly to facilitate the labours of the learner, the following classification will be found to possess a certain practical utility, however small may be its claim to scientific validity: viz.—

A. Acute miliary tuberculosis of the lungs.
B. Acute caseous tuberculosis (acute pneumonic phthisis).
C. Fibroid tuberculosis of the lungs.

D. Fibro-caseous tuberculosis of the lungs (chronic pulmonary phthisis).

The above terms are almost self-explanatory. By acute miliary tuberculosis of the lungs we understand that variety of the disease in which there is a rapid dissemination of miliary tubercles throughout a large portion of the pulmonary area. By acute caseous tuberculosis (or acute pneumonic phthisis) we understand that variety of the disease where a considerable portion of lung tissue is rapidly infiltrated by tubercle and quickly undergoes caseation, the case simulating in many of its physical signs and symptoms croupous pneumonia. By fibroid tuberculosis of the lungs we understand that variety of the disease where the chief anatomical change in the lungs is the supplanting of the normal pulmonary parenchyma by fibroid tissue. By fibro-caseous tuberculosis of the lungs (or chronic pulmonary phthisis) we understand that variety of the disease where there is a chronic course and varying degrees of fibroid and caseous change in the lungs. The student or practitioner will do well, while using this classification as a *memoria technica*, to study the disease by preference as a whole, to recollect at all times that one type may supervene upon another, that the essential forces at work are always the same. It will be convenient if we consider A, B, and C in a somewhat summary fashion, and deal at length with D.

A. Acute Miliary Tuberculosis of the Lungs.

This may be a primary condition, which is probably very rare; or it may be the terminal event in cases where there has previously been a quiescent seat of disease, an encapsuled focus breaking down and the bacilli finding entry into the blood-stream. Infection through the blood is the rule in acute miliary tuberculosis, and in many of these cases tuberculosis of the bloodvessels is present. In some cases the

meninges of the brain also become involved, and the symptoms of tubercular meningitis become added to those of the primary malady. Buhl was no doubt right in contending that in most of these cases a softening caseous mass can be detected in some organ or tissue of the body.

Acute miliary tuberculosis is relatively common in young children, and is rare after the age of fifty. It is commoner in the male sex than in the female. The miliary tubercles are widely disseminated throughout the lungs, and may, or may not, show traces of caseation. The former is the rule in the adult, the latter in the child.

Symptoms of Acute Miliary Tuberculosis. — The onset may be gradual or sudden. The usual symptoms are malaise, anorexia, headache, dyspnœa, cyanosis, pyrexia. Hæmoptysis may mark the onset of the disease in cases where there is an old tubercular lesion. Cough is usually present, but expectoration may be absent, or, if present, not characteristic in quality, and it is most important to remember that bacilli are frequently absent. The dyspnœa is a highly characteristic feature of the disease, the respirations being often 50 or 60 per minute in the adult, or as much as 80 per minute in the child. Cyanosis is a prominent feature, and is most marked on the lips and the tips of the fingers. Pyrexia is present, but has no definitely fixed type. It is often irregular, sometimes continuously high, sometimes intermittent, occasionally for a few days it may assume the inverse type—*i.e.*, a morning exacerbation with an evening remission. Abrupt rises to 104° F. or over, with rapid falls to a subnormal point and symptoms of collapse, may occur. As compared with the temperature of typhoid fever, the pyrexia of acute miliary tuberculosis is on the average less continuously high, more irregular, oftener descends to or below the normal point, is oftener of the inverse type. According to Cornet, the temperature may never exceed a moderate height—100·4° to 101·3° F.—or may

remain normal even in fatal cases.* When tubercular meningitis complicates the pulmonary affection, the temperature is usually moderate. The pulse is very frequent, often as much as 120 to 150, even at the outset, and is more frequent than the temperature would lead us to expect. It is usually small and weak, and occasionally dicrotic. In old persons, or in the presence of meningitis, the pulse may be slow. The prominence of the nervous symptoms—headache, torpor, restlessness, tinnitus aurium, somnolence, insomnia, vertigo, coma, delirium, apathy—is an important feature of the disease. The spleen is often enlarged. Albuminuria has been observed. The skin is usually cyanotic and drenched with perspiration; herpes on the lips, a roseolar or petechial eruption, have been occasionally observed, but are, on the whole, rare. Towards the end of the disease œdema of the extremities makes its appearance.

The Physical Signs of Acute Miliary Tuberculosis.—If there be old tubercular foci, these will yield characteristic signs. In the absence of such foci the physical signs are chiefly those of diffuse catarrh. As the case progresses, the percussion sound may have the tympanitic quality of relaxed lung. The vesicular breathing is harsh, expiration is prolonged, sibilant and sonorous rhonchi are present, and fine crepitant râles are heard. A soft friction murmur, due to tubercles in the pleura, may be heard. There is a notable disproportion between the urgency of the dyspnœa and the comparatively ill-marked physical signs. As the case advances, the signs of pulmonary œdema make their appearance. Tubercles are often found in the choroid.

Diagnosis of Acute Miliary Tuberculosis.—There may be some difficulty in distinguishing the disease from capillary bronchitis (broncho-pneumonia) and from typhoid fever. As regards the former alternative, the chief points which assist the diagnosis are the history of previous

* Cornet, *op. cit.*, p. 659.

pulmonary disease, the severity of the symptoms in proportion to the physical signs, the prominence of nervous symptoms, the occasional history of hæmoptysis, the rapid emaciation, the more irregular temperature. Typhoid fever can usually be excluded by attention to the urgency of the dyspnœa, the degree of cyanosis, the early prostration, the great frequency of the pulse in proportion to the temperature, the less regular temperature, the uniform progress downwards, the absence of any of the distinctive signs of typhoid fever. In some cases the simulation is close.

Course and Event.—The disease usually ends fatally in from ten days to three weeks, but life may be prolonged for three months or longer. The outlook is much worse than in acute caseous tuberculosis of the lungs.

B. Acute Caseous Tuberculosis of the Lungs (Acute Pneumonic Phthisis).

In this variety of the disease the lesions may assume either the broncho-pneumonic (disseminated) or the lobar type, the former being by far the more common. The disseminated form is most common in children and young persons. Children are often infected during convalescence from measles or whooping-cough. The disease is more often a primary pulmonary affection than acute miliary tuberculosis. The latter is the usual type when an acute pulmonary attack supervenes upon a chronic attack. In the disseminated form the process begins in the bronchioles, and rapidly extends to the alveoli. A cellular exudation takes place into both bronchi and alveoli, and rapidly undergoes caseation. The lesions may be irregularly scattered throughout one or both lungs in a large portion of their extent, softening and excavation are usually in progress, and are in most cases more advanced at the apices than elsewhere. In the lobar form a single lobe may be involved, or even a whole lung. The consolidation is complete, caseation

rapidly ensues, and a cavity may form. The pleura is usually inflamed, and necrosis of the pleura, followed by perforation and pneumothorax, is an occasional sequel. The bronchial glands are enlarged and often caseous. Secondary tubercular lesions may be present in the larynx, trachea, or intestines.

Symptoms, Physical Signs, Course and Event in Acute Caseous Tuberculosis.—I doubt if any useful distinction can be drawn between the symptoms of this variety and those of acute miliary tuberculosis. Nor can the physical signs be sharply differentiated. Baumgarten says truly that the difference is quantitative rather than qualitative. I am under the impression, however, based on actual experience, that a certain distinction may be drawn from the point of view of prognosis. I have never seen a case definitely diagnosed as acute miliary tuberculosis take a favourable course, although some slackening in the acuteness of the tubercular process is not unknown. But I have seen cases of acute caseous tuberculosis make the most decided, and sometimes wholly unhoped-for, rallies, and attain a condition of more or less complete arrest with or without the formation of a cavity in the caseous lung. It seems reasonable to suppose that in such cases the consolidated area or areas of lung may have been largely composed simply of inflammatory products. Most observers seem to be agreed that bacilli are more uniformly present in the sputum in acute caseous tuberculosis than in acute miliary tuberculosis.

The lobar form of this variety may be confused with croupous pneumonia, but the absence of crisis, the very rapid pulse, the marked sweating and emaciation, and tubercle bacilli in the sputum, make the distinction easy.

C. Fibroid Tuberculosis of the Lungs.

In all probability this variety is simply a very chronic form of ordinary phthisis occurring in persons of good resisting power. The conception of a 'fibroid phthisis' independent of tuberculosis is obsolete.

Fibroid tuberculosis occurs at a later age than the other varieties; in men more often than in women; often in persons of muscular build and good digestive power; frequently in persons engaged in dusty occupations. The disease begins in the finer bronchi, but its course is more towards the peribronchial tissue than towards the alveoli. The tubercles, instead of caseating and softening, become transformed into firm, fibrous, and pigmented tissue. The lung substance shows a strong tendency to contract. Some emphysema usually develops around the fibrosed areas.

The symptoms are chronic in course and of slight intensity. Hæmoptysis may be an early symptom. There is not much expectoration, and bacilli may be absent for long periods. There is little or no pyrexia, and no marked emaciation.

The physical signs include retraction of the chest on the affected side; some impairment of resonance, unless much emphysema be present; weak breathing with prolonged expiration and fine crackling râles.

The prognosis is temporarily favourable, but ultimately in most cases the disease becomes more active, and pursues the ordinary course of chronic phthisis, the fibroid tuberculosis becoming fibro-caseous in type.

D. Fibro-Caseous Tuberculosis of the Lungs (Chronic Pulmonary Phthisis).

This is the ordinary type of the disease. Each case presents, according to its stage and rate of progress, one or more or all of the following fundamental pathological processes—viz., infiltration, caseation, softening, excavation.

Some degree is also present of the following conditions—viz., fibrosis, pigmentation, inflammatory exudation and consolidation, and bronchial catarrh. These are the elements which make up the pathological condition, and combine to give rise to a most diversified symptom-complex, which, nevertheless, preserves a certain unity in diversity. A tendency to bronchial catarrh, with cough and expectoration; to fever and wasting, which are closely associated; to hyper-secretion from the skin; to low vascular tone; to some disorder of digestion—is nearly invariable, although all these symptoms may, in favourable cases, have their periods of complete latency. The disease is also characterized as a rule by periods of improvement, which may last long or even amount to complete arrest, but which in most cases are succeeded by relapses and downward progress. A mental condition of undue hopefulness, at variance with the obvious facts of the case, is sufficiently often present to possess diagnostic import, while the patient's sufferings, except in presence of some of the more painful complications, often seem surprisingly slight. The patient often wastes away with little actual suffering, and may cherish hopes of recovery up to the brink of the grave.

Nature and Progress of the Lesion in Chronic Fibro-Caseous Tuberculosis.—The primary infiltration of the lungs is in a very large majority of cases in one apex, from 1 inch to 1½ inches below the summit of the lung. The marked preference of tubercle for the apex has not been finally explained, but there is much to be said for the usual view that it is due to the lesser functional activity of this part, together, perhaps, with the fact that the bronchus leading to the apices is at a more acute angle with the main air passages than the other bronchi, and hence secretions may find a firmer lodgment there. It seems probable that coughing may in some cases tend to drive secretions towards, rather than away from, the apices. In view of the fact that

the primary invasion of tubercle is often by way of the bronchial glands, it seems probable that anatomical reasons may explain the transference of the bacilli from the glands to the apices. The primary focus of infiltration corresponds in front either to the supraclavicular fossa or to a spot immediately below the centre of the clavicle; behind, to a spot in the supraspinous fossa near the middle of the fossa, and about 1 inch below the summit of the lung. From the original focus the lesion tends to spread, either irregularly in the more rapid cases, or in the more chronic cases with some approach to uniformity—viz., first downwards and backwards in the primarily affected apex; then to the apex of the opposite lung; then to the upper portion—a little below its highest point—of the lower lobe on the side first affected; then to the upper portion of the lower lobe on the side secondarily affected; then throughout the pulmonary substance more generally, the bases being usually last and least affected by an actual tubercular deposit, though they are frequently the seats of congestion and œdema. The lesions are nearly always more advanced at the apices, and in chronic cases are nearly always bilateral. Many observers believe that the lower lobe on the side first affected is generally involved before the apex of the opposite lung. There is certainly no hard-and-fast rule as regards this matter. The lesions tend to go through the ordinary changes from infiltration to softening and excavation, fibrosis being usually a prominent feature in slowly progressive cases and in subjects of good resisting power. Cavities ultimately form in most cases, usually by the coalescence of groups of tubercular foci, which caseate and soften, the contents being expectorated after the opening up of a bronchus of sufficient size, or less frequently by the breaking down *en masse* of caseous areas of considerable extent. Recent cavities are usually characterized by irregular outline, ill-defined limits, ragged interior, and the absence of a distinct wall. Trabeculæ

are usually present, and the cavity is often crossed by blood-vessels. Chronic cavities have a greyish lining membrane and a fibrous capsule, the latter produced by the irritant effect of the tuberculizing process upon the neighbouring tissues. Most cavities contain secretion, but some are quite dry. The cavity may or may not communicate freely with a bronchus. Basic tuberculosis is rare; but the practitioner would do well, in a case where the signs seem exclusively basic, but where the probability of tuberculosis is strong, to scrutinize carefully both the apical regions and the inter-scapular regions about the level of the fifth dorsal spine. He will often succeed in detecting slight but significant signs in one or other of these regions in cases which seem at first to be exclusively basic. A useful clinical rule is the following: 'If the whole lower lobe be continuously affected, the lesion is probably tubercular; if the base is affected, but the apex free, the lesion is either non-tubercular—viz., œdema, collapse, catarrhal pneumonia, pleurisy—or, if tubercular, the resisting power of the base has been diminished by some previous affection.'*

Bronchiectasis is commonly present in chronic tuberculosis accompanied by fibrous induration; emphysema is usually found in the neighbourhood of contracting tubercular lesions; collapse and œdema are not infrequent; some pleural involvement is always present; gangrene is rare.

MODES OF ONSET IN FIBRO-CASEOUS TUBERCULOSIS OF THE LUNGS.—Some information of value, especially from the point of view of prognosis, may be obtained by studying the modes of onset of the disease, but the more closely we scrutinize our cases the more we shall find much essential identity under a somewhat misleading guise of diversity. As examples, take the following:

(*a*) A case sets in with a smart hæmoptysis in the midst of apparently perfect health, and we may feel inclined to talk

* Fowler and Godlee, 'Diseases of the Lungs,' p. 354.

of a 'hæmorrhagic onset.' But in the overwhelming majority of these cases, hæmoptysis was not the first symptom, but only the first symptom which attracted attention. I have enquired into many of these cases, and I have hardly ever failed to elicit a history that prior to the occurrence of hæmoptysis the patient had been for some time coughing, losing weight, or otherwise out of health. Nor does an early hæmorrhage warrant the belief that repeated hæmorrhages will characterize the further course of the case. This may or may not be the case, but an early hæmorrhage may be followed by a non-hæmorrhagic course or *vice versâ*.

(*b*) A patient has a succession of 'bronchial colds,' is ultimately found to be tubercular, and we feel disposed to speak of a 'catarrhal onset.' If this term is construed simply to indicate that bronchial catarrh was a prominent feature of the early stage, there can be no objection to the term, which then simply states a fact; but if the expression be employed to signify that 'bronchial catarrh' is the cause of the subsequent phthisis, then, in all probability, the term is, in a large majority of cases, misleading. The bronchial catarrh in such cases is far oftener the effect of a tubercular deposit already present than the cause of such a deposit. Experience amply demonstrates that the tendency to bronchitis and the tendency to tuberculosis have, in general, little in common.

(*c*) A patient is attacked by pleurisy, and, later on, becomes tubercular, and we are disposed to speak of a 'pleuritic' mode of onset. Before using this term, which I am inclined to think is often legitimate, we require some assurance that the attack of pleurisy was not preceded by a tubercular focus at one apex.

(*d*) A patient is attacked with laryngeal symptoms, which are found to depend on tubercular laryngitis, and we are disposed to speak of a 'laryngeal onset.' But in the vast majority of these cases—probably not less than 95 per cent. —there is pre-existing tubercular disease of the lungs. Some

authorities have gone so far as to deny the existence of a primary tuberculosis of the larynx. This is probably an exaggeration, but the condition is certainly very rare.

If we could definitely trace the mode of origin of pulmonary tuberculosis, and had an accurate register of all the early phenomena, I have little doubt that the 'insidious onset,' which everyone recognises as much the most frequent, would account for the great majority of cases. The acute cases for the most part represent an acute exacerbation of a pre-existing lesion. The 'hæmorrhagic' and 'catarrhal' cases simply represent types in which a certain symptom, or group of symptoms, happens to predominate. The 'laryngeal' cases represent the type in which laryngeal complications are earlier and more outstanding than usual, but we must not forget that tubercular involvement of the larynx is found in nearly half of all fatal cases of ordinary phthisis. The significance of the 'pleuritic' mode of onset cannot be fully determined until we know fully the inter-relation of pleurisy and tuberculosis, a subject which still awaits complete elucidation.*

The prognostic inferences which may be drawn in a very tentative fashion from the modes of onset of pulmonary tuberculosis will be considered in connection with the general prognosis of the disease.†

THE SYMPTOMS OF FIBRO-CASEOUS TUBERCULOSIS OF THE LUNGS.—These present the very diversified picture with which we are familiar. They would appear to depend on the following causal factors:

(*a*) The local consequences of tubercular infiltration, caseation, softening, and excavation.

(*b*) The constitutional effects of the tubercular virus.

(*c*) The individual reaction of the patient.

(*d*) The effects of mixed infection, especially by the pyogenetic bacteria.

* See Lecture VIII.

† See Lecture XVII.

(*e*) The consequences of complications—laryngeal, pulmonary, gastric, hepatic, intestinal, renal, splenic, nervous.

In studying the symptoms of the disease, we should do well to keep these factors distinct in our minds, although in many cases it is impossible to disentangle them. This analysis will help us to understand how variable the symptoms are; the prominence of pulmonary symptoms in one case, their comparative latency in another; the nervous erethism of one patient, the torpor of another patient; the pronounced evidence of sepsis, or the absence of such evidence; the widespread disorder of many functions, or the comparative good health and freedom from symptoms which the patient may exhibit. We are apt to underrate the importance of the element of individual reaction to the disease in these cases, and we do not always distinguish sufficiently the effects of the tubercular virus from those of the mixed infections. We shall proceed to consider the chief symptoms in order.

Cough, Expectoration, Dyspnœa, Hæmoptysis.—Cough is rarely absent in phthisis, except during periods of complete latency of the disease. It is generally an early, often the earliest, symptom. A few patients who presented unequivocal evidence of phthisis have assured me that their condition had developed without cough, but I have always doubted the accuracy of the statement. The cough is at first short and 'dry,' perhaps hacking, in character; worst in the mornings or after meals, or on lying down at night. The expectoration is often wanting at the outset, especially in miliary tuberculosis, caseous consolidation without softening, and fibroid tuberculosis; but it soon appears, and consists of clear, viscid, glairy mucus, which may, or may not, be streaked with blood. As the case proceeds the cough becomes looser, and the expectoration contains small grey or greenish purulent masses, which often contain numerous bacilli. At a still later stage the expectoration is often profuse, muco-purulent, yellow, or greenish in character, and in some cases 'nummulated.' The

sputum has often a faintly sweet odour; sometimes it is fœtid, but extreme degrees of fœtor are decidedly rare. In some cases the cough assumes a severe, paroxysmal character, and frequently continues until vomiting ensues. This is especially common when there are cavities present which contain secretion difficult of expulsion. The amount of coughing is much dependent on the state of the patient's nervous system, and has no constant relation to the amount of disease present or its rate of progress. Old persons cough, as a rule, less than the young, and the insane often do not cough at all. Cough may depend upon, or be aggravated by, pharyngeal catarrh, which is often present; laryngitis, simple or tubercular; pleurisy; or by enlarged bronchial glands pressing on the vagus.

Dyspnœa is often slight, even in advanced cases, the respiratory needs of the organism apparently getting less as the breathing capacity of the individual declines. In the incipient stage of the disease shortness of breath on moderate exertion is often present. The degree of dyspnœa has very little relation to the amount of lung involved, but much relation to the rapidity with which the disease progresses. Thus, in chronic and slowly progressive cases, it is usually quite slight, while it is a pronounced feature in acute miliary tuberculosis. The cause of dyspnœa is partly a limitation of the breathing surface, partly pyrexia, partly emphysema, partly psychic influences. It is increased by much bronchial catarrh, broncho-pneumonia, pulmonary œdema, pleurisy, or emphysema. Some cases of phthisis have 'asthmatic' attacks, due probably to emphysematous distension of the air vesicles in the vicinity of areas of fibrosed pulmonary substance. If the case be seen for the first time during one of these attacks, its true nature may escape observation. Hæmoptysis will form the subject of a special lecture.*

* See Lecture XXI.

Hoarseness.—This is a common symptom in phthisis, especially when the disease has advanced a certain stage, and has various causes. It may, of course, depend on tubercular invasion of the larynx, but this is not the most usual explanation. Often it depends upon simple laryngeal catarrh, to which these patients are prone; on nervous debility and feebleness of expiration; on pressure by enlarged glands on the recurrent laryngeal nerve; on syphilis. The practitioner is apt to attach a grave prognostic import to the development of hoarseness in the course of a case of chronic phthisis, and in a large proportion of cases he will be right in so doing. But he must recollect that in numerous cases this symptom does not mark the opening up of the final stage of the disease, but is dependent on remediable causes, or on co-existing conditions which at least admit of palliation. A thorough laryngoscopic examination is, of course, our main reliance in differentiating these conditions, but even this may yield indecisive evidence. In my experience, the involvement of the larynx by tubercular deposit usually coincides with marked constitutional symptoms and an obvious development for the worse in the general condition; also, it is usually gradual. But these rules are hardly to be absolutely relied upon.

Pain in the Chest.—This is a frequent symptom, and has some diagnostic value. It may be present either over the seat of the lesion, in which case it usually points to the existence of pleuritic adhesions, or along the attachments of the muscles, in which case it probably depends on dragging upon these muscles during coughing. In my experience, the most frequent seats of pain are the infraclavicular and mammary regions, and in these regions it is often accompanied by superficial tenderness. In an incipient case, where signs and symptoms were mainly negative, I have frequently obtained some assistance by attention to the fact of pain at one apex, and by a re-examination of the painful

area. Cornet relates the case of a patient who could always predict the occurrence of an attack of hæmoptysis by pain, probably depending on congestion. At later stages of the disease pain may have many explanations—*e.g.*, pneumothorax—and becomes a symptom of relatively small importance. Some cases of phthisis run their entire course without pain.

Dysphagia.—This is usually a late and very distressing symptom, depending on tubercular invasion of the pharynx and larynx. Ulceration of the epiglottis or epiglottic folds is often present in these cases. Sometimes the cause of the dysphagia seems to be paralysis of the depressors of the larynx.

Pyrexia.—This is one of the most constant symptoms of phthisis, and depends, no doubt, upon the absorption of toxins by the blood. It cannot, however, be too strongly affirmed that phthisis may develop, progress, and even proceed to a fatal issue (this last is, however, extremely rare) without fever. The idea which sometimes finds favour that if in a suspicious case we can exclude the presence of afternoon pyrexia for some time, phthisis may be put out of account, is quite untenable. I have seen many exceptions to the law that afternoon pyrexia is the rule in phthisis, exceptions by no means limited to convalescent or arrested cases. Pyrexia, especially of a certain type, often decidedly strengthens the suspicion of phthisis, but its absence should not be accorded any decisive diagnostic weight. It seems highly probable that the pyrexia of phthisis depends in large measure upon the presence of mixed infections, especially the pyogenetic cocci. We must be further on our guard against expecting the pyrexia of phthisis always to correspond to a certain standard. Certain types of pyrexia are, no doubt, very common in the disease, and have much significance, but the variations from type are innumerable, and present the most diverse characters. Irregular variations

and changes of type, according to the amount, stage, and rapidity of progress of the disease, are always to be expected; but it is an error, in my judgment, to set up a series of clearly-defined varieties of the phthisical pyrexia. Experience does not support the view that the pyrexia of acute miliary tuberculosis can be clearly differentiated from that of acute caseous tuberculosis, or that of fibroid tuberculosis from that of fibro-caseous tuberculosis. We can affirm that certain types of pyrexia are relatively common in each of these conditions, but that is all. There is no sharp line of differentiation. I agree entirely with West when he says: 'Nor do I think that any special form of temperature curve can be associated with the different forms of phthisis, as some authors maintain—at any rate, in more than a perfectly general way—viz., that where the process is active or acute the pyrexia is high; where it is not very active, moderate; and where it is stationary, absent altogether.'* The most frequent type of temperature in characteristic cases is the hectic type—viz., an irregular and unstable temperature, with sharp oscillations and a marked daily curve, usually characterized by a decided afternoon or evening rise, and a fall to or below normal in the early hours of the morning. The degree and character of the pyrexia probably depend partly on the amount or activity of the tubercular virus, partly on the constitutional peculiarities of the patient. The maximum temperature is usually reached between the hours of 2 and 8 p.m., but may be later. The minimum usually occurs in the early morning between the hours of 2 and 4 a.m. If a two-hour chart be kept it will sometimes be found that there are two periods of alternate rise and fall of pyrexia—one in the forenoon, and the more usual one in the afternoon. The reverse type of pyrexia—viz., a morning maximum and an evening minimum—has been occasionally observed, but is very rare. The phthisical pyrexia is, as West observes, characterized by

* S. West, 'Diseases of the Organs of Respiration,' vol. ii., p. 457.

great instability, rarely remaining long at the same level, but always either rising or falling. The maximum varies with the intensity of the morbid process. An afternoon temperature of from 101° to 103° is common, but higher temperatures may be observed. Hyperpyrexia is, in my experience, very rare. The minimum is often much below normal, and this is a point of considerable practical weight. A high afternoon rise with a low morning minimum represents one of the most unfavourable clinical types. The early pyrexia of phthisis is sometimes very like that of malaria, but periodicity is more marked in this latter condition. Complications may, of course, cause the most diverse varieties of pyrexia—a pneumonia or a pleurisy, for example, causing a rise, pneumothorax or profuse hæmoptysis causing a fall. A continuously high temperature is sometimes observed in cases of very active tuberculization.

It is an interesting and important question, which has not yet been fully elucidated, how far the pyrexia of phthisis is due directly to the tubercular virus, and how far to mixed infections, especially of the pyogenetic cocci. I have already expressed the view that the influence of micro-organisms of the latter class is potent, but the effects of tuberculin injections show us that the tubercular virus itself is an adequate cause of pyrexia. The most ordinary observation, however, serves to show that the pyrexia of phthisis and that of suppurative fever have much in common—viz., marked rises of temperature inaugurated by a chill, and sharp remissions with profuse sweating. The importance of endeavouring to prevent mixed infections should always be present to our minds in the management of phthisis.

Sweating.—The association of sweating with pyrexia is close, but not invariable. The most out-standing characteristic of phthisical sweating is its proneness to occur during sleep, whether by day or night. In the latter case it may occur shortly after the patient falls asleep, but more

frequently, in my experience, towards the early hours of the morning. In the early stage of the disease, when there is little or no pyrexia, the patient may exhibit a tendency to sweat on slight exertion, or his hands and feet may be constantly moist. The sweating is, as a rule, proportionate to the severity of the disease, and the dread with which it is viewed by the laity is well founded. Louis found sweating absent in one-tenth of his cases of phthisis. According to West, it is most often absent in acute cases which present more the characters of acute broncho-pneumonia. I have generally found it most marked in cases where there was much debility, with a high afternoon and low morning temperature. Sweating sometimes alternates with diarrhœa.

It is important to recognise that night-sweating is not confined to phthisis, but may occur in cases of debility, hyper-lactation, or overwork. The symptom may thus sometimes give rise to groundless fears.

Emaciation.—This is usually an essential feature of the disease, as the term 'consumption' conveys. It is often an early symptom, and may be the first to attract attention. It is usually closely related to the pyrexia of the disease. It is, on the whole, the best gauge of the activity of the disease, and the weighing-machine, which should be in constant use in dealing with phthisical patients, gives us information hardly second in importance to that yielded by the stethoscope and the thermometer. The loss in weight is not in general dependent on obvious dyspepsia, but seems to be a direct result of the tubercular toxin. A gain in weight is usually coincident with favourable progress, but under hyper-alimentation and sanatorium methods we now not infrequently see patients who gain weight in spite of active inroads of the disease. This represents one of the least favourable types of phthisis. The gain in weight is, as might be expected, more rapid in patients who enter a hospital or a sanatorium after a life of privation than in those who have had an easy life

and every luxury. Rest in bed is a potent assistance in putting on weight.

Disorders of the Digestive Organs.—Dyspepsia in some form is common in phthisis, but it is not invariable, and, in my experience, does not conform to any definite types. Early dyspepsia, sometimes in the form of anorexia, may occur, but not more frequently, I think, than in other forms of chronic disease. The ordinary dyspepsia of the more advanced stages of the disease generally takes the form of gastric catarrh, and is closely related to the dietetic usages of the patient. The tongue is often furred and the papillæ prominent; but in other cases we may have the large, flabby tongue of atonic dyspepsia; or the red, raw, 'beefy,' or dry and fissured tongue characteristic of ulceration of the bowels. Vomiting is common, and is often the termination of a violent fit of coughing.

Diarrhœa is frequent and is a sign of great importance. It is needful to distinguish three types of diarrhœa in phthisis —viz., (*a*) the diarrhœa due to errors in diet and fermentative changes in the food; (*b*) the diarrhœa depending on ulceration of the bowels; (*c*) the diarrhœa depending on lardaceous disease of the intestines. In general, these varieties of diarrhœa can be readily distinguished. The diarrhœa of dietetic origin will be accompanied by furred tongue, foul breath, uneasiness in the epigastrium, irregular bowels, in some cases by nausea and vomiting. The diarrhœa dependent on ulceration of the bowels is painful, some blood may be present in the motions, but is often absent, and there is usually pain on deep pressure in various parts of the abdomen. The diarrhœa of lardaceous disease of the intestines is a persistent, painless, watery flux; the liver and spleen will probably be enlarged, and albumin may be present in the urine. Melæna is rare in phthisis. Fistula in ano is somewhat common, especially in the male sex. Sir Douglas Powell puts its frequency at 5 per cent. of cases.

It must be remembered that fistula occurring in a phthisical patient is not necessarily of tubercular origin. Ulceration of the rectum is found in about 13 per cent. of fatal cases. Thrush is not uncommon in bad cases and towards the close. It is always a very ominous sign.

The Urinary System.—The urinary symptoms are unimportant and uncharacteristic.

The Cutaneous System.—Pityriasis versicolor is frequent. Purpura has been occasionally observed.

In reviewing the symptoms of chronic phthisis we see how variable is the clinical picture—how much we have to do with a process which presents every degree of intensity, but which at bottom is always essentially the same. The key to each case is the intensity of the tubercular virus and the resisting power of the individual; and when we come to consider the prognosis of the disease we shall find that the same considerations govern our forecasts of its probable course. Of the intensity of the virus, whether that be a question of quantity or quality of the poison, we have no definite gauge, but it is probable that the virulence or mildness of an attack in any individual case finds in this circumstance a large portion of its explanation. The resisting power of the individual is more capable of estimate, and would appear to depend on many factors, of which hereditary tendency, occupation and mode of life, and state of nutrition are probably the chief.

We shall reserve questions of diagnosis for the next lecture.

LECTURE XV

THE EARLY DIAGNOSIS OF PULMONARY TUBERCULOSIS

SUMMARY:

The meaning of 'early diagnosis.'

Assistance in early diagnosis may be obtained from—

A. Family history.

B. Personal history.

C. Symptoms and mode of onset of the disease.

D. Sputum.

E. Physical signs.

Family history.

Personal history. Influence of occupation, constitutional state, and previous diseases.

Symptoms and modes of onset of the disease:

Importance of chronic cough, hæmoptysis, pyrexia, wasting, pain in the chest, persistent frequency of the pulse.

The sputum:

Rules regarding the presence of tubercle bacilli.

Physical signs.

It is not necessary to dwell at length upon the importance of the early diagnosis of pulmonary tuberculosis. The disease is, in a certain proportion of cases, comparatively tractable in its early manifestations, whereas the fully-developed malady is usually incurable. The popular impression that everything depends upon 'getting the case in time' is, upon the whole, well founded.

It may be well to enquire what we mean by 'early diagnosis.'

The picture drawn of the incipient stage of the disease is often misleading. When some degree of flattening and dulness is present at one apex, with, perhaps, a few dry, crackling râles, and the symptoms of cough, wasting, and afternoon pyrexia are well established, the case is no longer incipient, although the lesion may yet be comparatively local. By this time, indeed, phthisis has made serious, perhaps irreparable, headway. The problem of the early diagnosis of phthisis should rather be stated as follows: What facts or combination of facts as regards history, symptoms, and physical signs afford presumptive evidence that the process of tuberculization has begun in the lungs? Are we justified in sounding the alarm before we find unequivocal signs in the lungs or bacilli in the sputum? How are we to appraise, on the one hand, suspicious symptoms in the absence of physical signs, or suspicious signs in the absence of symptoms? Is a patient who has had several attacks of hæmoptysis, and who is coughing and losing flesh, to be pronounced tubercular, although we can detect neither definite physical signs in the lungs nor bacilli in the sputum? What are we to say regarding a case where we find slight dulness and prolonged expiration under one clavicle, while symptoms are practically wanting? These problems might be multiplied indefinitely. They are not fanciful problems. Every physician has felt their weight, and has sometimes wavered in his judgment. The important point to bear in mind is this: the early diagnosis of pulmonary tuberculosis is a complex problem, not to be settled by a reference to any set of physical signs or any definite assemblage of symptoms, but to be viewed as a whole, its difficulty to be always fully borne in mind. I exclude from present consideration all cases in which the diagnosis is, with reasonable care and skill, obvious.

It would be easy to say, 'Do not give a positive diagnosis until either bacilli are found in the sputum or definite physical signs can be detected in the lungs. In other cases

take refuge in such euphemisms, which the lay mind is only too ready to accept, as "a delicate chest," "weak lungs," or "tendency to consumption."' It must be insisted that this is an absolutely pernicious policy. It means in many cases the sacrifice of the patient's best, perhaps his only, chance of recovery. While we are waiting for evidence to relieve us of the risk of diagnostic error, tubercle is too often making disastrous progress. Every case in which the history, symptoms, or physical signs fairly suggest the possibility of incipient phthisis should be promptly faced alike by physician, patient, and patient's friends. If a positive opinion can be arrived at, then the therapeutic problem arises. If a positive opinion is impossible, it is at least well to have considered the graver alternative. The one thing which the physician must not do is to use agreeable euphemisms and quiet alarms which may ultimately prove to have been only too well founded.

Assistance in Early Diagnosis.

We may seek for assistance in the early diagnosis of pulmonary phthisis by attention to the following points:

A. Family history.
B. Personal history.
C. Symptoms and mode of onset of the disease.
D. Sputum.
E. Physical signs, including the results of radioscopy.

A. Family History.—The tendency at the present day is to minimize the importance of this factor. No doubt its significance has been much exaggerated in the past. It is, however, an error to regard it as of no consequence. The suggestion put forward by Dr. Arthur Latham, that 'the children of tuberculous parents possess some natural immunity to the disease,'* is, I think, opposed to general

* Arthur Latham, 'Pulmonary Consumption,' second edition, p. 211.

experience. The deadly virulence of the disease in certain stocks from generation to generation can hardly be denied. Most authorities put the proportion of cases of phthisis where the disease is found in the direct family line of the patient at about 28 to 30 per cent. If the average risk be taken at about 15 per cent., we get 13 to 15 per cent. as the value of family predisposition, as such. As I have pointed out in an earlier lecture, the disentanglement of family proclivity from the element of special exposure to infection is practically impossible. The certain fact that pulmonary tuberculosis affects some stocks more than others, and in some stocks is particularly deadly, may be allowed a certain weight in the diagnosis of a doubtful case. We must be careful, however, not to allow such a history to prejudice our opinion in favour of a diagnosis of tubercle, nor to permit the absence of such a history to make us unduly sceptical about the probability of tubercle. Members of phthisical families who escape infection are often up to the full normal standard of health.

B. Personal History.—This is much more important than family history. Even if we admit, as I am disposed to do, the influence of an inherited tendency to phthisis, this factor is, without doubt, of much less importance than those influences which operate in the individual subsequent to birth. We may consider the following points—viz.:

1. The patient's 'constitution,' development, weight, and general health history.
2. His occupation.
3. His previous diseases.
4. The presence of anæmia, dyspepsia, glandular enlargements or scars, disease of bones or joints.

Phthisis rarely arises in persons of healthy aspect, sound digestion, adequate weight, and good blood-making power. Yet even this rule has its exceptions, and the wider our outlook the more numerous shall we find those exceptions to

be. I can recall a number of cases where tubercle developed in individuals of apparently faultless physique and quite exceptional vigour and development. But such cases are decidedly rare. Some impairment of the general health is a fairly constant forerunner or concomitant of the onset of phthisis.

The shape of the chest will receive attention. The flat chest and the alar or winged chest are common in phthisis. Let it be remembered, however, that the disease not rarely develops in chests of normal shape and dimensions.

The erethic constitution (*eretische constitution*) is common in connection with phthisis. This condition seems to be one of deficient nervous and vaso-motor stability. The patient is often intellectually alert, responsive to all kinds of stimuli, capable of considerable effort, but soon fatigued; his pulse is too fast, his eye too bright, his skin too moist.

The weight is a point of great importance. Phthisical patients in the great majority of cases show a decided loss of weight either before, or in the early stages of, tubercular invasion of the lungs. Yet it has fallen to my experience in a considerable number of cases to find bacilli in the sputum where there was no material loss of weight.

A proneness to catarrhs—'colds on the chest'—is common in the subjects of tuberculosis, but is, of course, often simply an evidence of a tendency to bronchitis. If a series of catarrhs is the precursor of tuberculosis, we may expect that the tendency will be to flatness and deficient resonance of the chest; if such catarrhs are merely of bronchitic origin, the tendency will be to over-distension and hyper-resonance of the chest.

Occupations involving continuous confinement in dusty and vitiated air carry with them the heaviest risk of tuberculosis, and the fact of a patient who presents ambiguous signs or symptoms of the disease being engaged in such

occupations adds weight to suspicions of infection. The fact of an indoor occupation in impure air should make us scrutinize with special care any signs or symptoms of commencing phthisis.

The diseases most often followed by phthisis are the following: Pleurisy, broncho-pneumonia, measles, influenza, whooping-cough, typhoid fever, insanity, diabetes, chronic syphilis, cancer, severe dyspepsia, anæmia. The relation of pleurisy to phthisis has already been fully considered. Although some exaggeration has characterized the statements of certain writers who have emphasized the closeness of the relation, yet it would be easy to under-estimate the importance of the connection between the two diseases. Most physicians will endorse Osler's declaration: 'I confess that the more carefully I have studied the question, the larger does the proportion appear to be of primary pleurisies of tubercular causation.'* In one respect pleurisy appears to differ from certain other diseases often followed by phthisis—*e.g.*, influenza, measles, and whooping-cough—viz., in the fact that even a pleurisy apparently completely recovered from, and leaving neither physical signs nor impairment of the general health, seems to involve a legacy of increased proclivity to phthisis. I doubt if this is true of the other diseases mentioned, but it seems to be true of pleurisy, however we may interpret the fact. Broncho-pneumonia is followed by phthisis in an important proportion of cases; croupous pneumonia exceedingly seldom, but we must bear in mind that an attack of pneumonia may bring to light a pre-existing tuberculosis of the lungs. Measles and whooping-cough are often followed by phthisis, especially in ill-nourished and badly-cared-for children. Influenza seems to be a frequent precursor of phthisis. No doubt the history of antecedent influenza so often proffered by phthisical patients is in many cases misleading, the patient confusing influenza

* Osler, 'Practice of Medicine,' p. 666.

with the incipient stage of tuberculosis. It is important to remember, also, that the great pandemics of influenza of the last two decades, which raised the pneumonia rate so decidedly, were coincident with a general fall in the tuberculosis rate in most civilized countries. It is probable that influenza frequently rouses quiescent phthisis into activity, but is only seldom the actual predisposing cause of tubercular infection. Typhoid fever is occasionally followed by phthisis, but in view of the fact that both diseases are so common the sequence does not seem to be very frequent. Diabetes is often associated with phthisis. Probably nearly one-half of the subjects of diabetes ultimately succumb to tuberculosis. Insanity seems to predispose to phthisis, as would on general considerations be likely. Of cases of idiocy and congenital imbecility, two-thirds ultimately die of tubercular disease. 'Consumption was startlingly more frequent as a cause of death among the inmates of the older asylums than in the modern institutions; but still it is in all asylums for the insane between three and four times more common than in the general population of the same ages.'* In most cases, no doubt, insanity is the antecedent and tuberculosis the sequel, but it is probable that this relation may be occasionally reversed. Possibly the *spes phthisica* of the tubercular patient and the *bien-être* of some forms of insanity may have something in common. The influence of the meningeal complications of tuberculosis in causing mental derangement has also to be borne in mind. Chronic syphilis is frequently associated with phthisis. The so-called 'syphilitic phthisis' is usually tubercular. Cancer is often associated with tuberculosis, the proportion of patients dying of cancer who present tubercular deposit in some organ being large. Severe dyspepsia and anæmia may be either predisposing causes or early manifestations of phthisis. My experience tends to confirm the time-honoured observation

* T. S. Clouston, 'Mental Diseases,' fifth edition, p. 507.

that distaste for, or inability to assimilate, fatty foods is a frequent precursor of phthisis.

The personal history may, then, assist us in the diagnosis of pulmonary tuberculosis by suggesting either that the patient's constitution and general development, or his occupation, or his previous diseases make him a likely subject for bacillary invasion.

I pass on to consider the remaining three lines of inquiry, which are by far the most important in the early diagnosis of phthisis, viz.:

C. The symptoms and modes of onset.
D. The examination of the sputum.
E. The physical signs.

C. The Symptoms and Modes of Onset.—Phthisis may develop to a certain point without symptoms. It is probable that the number of cases which never attract attention, soon become obsolescent, and give no trouble is not inconsiderable. Oftener, however, our difficulty is the reverse of this. There are symptoms and there are not signs. Phthisis very seldom develops without cough. It is true that just as a few isolated tubercles may exist in the lungs and yield no physical signs, so a similar condition may exist and yield no symptoms. Yet the cases are rare indeed where tuberculization of the lungs exists without exciting some degree of localized catarrh, and, as a consequence, cough. In any suspicious case of debility or loss of flesh, if we can satisfy ourselves that cough has been absent from the beginning, the presumption is against phthisis. It must be borne in mind, however, that some patients deny the existence of cough when it is really present, and also that cough may be really absent in quiescent cases. On the other hand, a persistent cough, with at first little or no expectoration, without the physical signs of general bronchial catarrh or other obvious explanation, especially if occurring in a young

adolescent, whose weight and general health are not quite satisfactory, is always suspicious. In such cases we should be careful to exclude such causes of persistent cough as an elongated uvula, pharyngeal catarrh, papilloma of the vocal cords, or pressure on the air passages by an enlarged bronchial gland or aneurysm.

Hæmoptysis is one of the most important points in the history, and we shall never regret attaching great weight to it. If we can exclude the other causes of hæmoptysis—viz., heart disease, anæmia, scurvy, varicose veins of pharynx, purpura, pulmonary gangrene, bronchiectasis, and a few other conditions*—the probability of pulmonary tuberculosis is very strong. The 'hæmorrhage from the throat,' of which we hear so much, is usually mythical. The so-called 'arthritic hæmoptysis' had better be viewed in most cases with considerable scepticism. The hæmoptysis of phthisis usually recurs, and if the bleeding has been moderately free it does not subside suddenly, a sharp bleeding being succeeded by a certain amount of gradually diminishing blood-spitting.

Pyrexia, impairment of appetite or digestive power, and wasting, form a group of symptoms closely related to each other and highly characteristic of incipient phthisis. If the patient has fever and digestive disorder he will certainly waste, unless the tendency to waste in such cases is kept under control, as often happens in the course of sanatorium treatment, by continuous rest and high-feeding. If, on the other hand, as sometimes happens, phthisis comes on without fever or digestive impairment, the patient may waste little, if at all. Upon the whole, however, wasting in some degree is one of the most constant symptoms, and we may say with substantial accuracy that in the diagnosis of incipient phthisis the weighing-machine is one of our main helps in

* See Lecture XXI. for a more complete account of the causes of hæmoptysis.

diagnosis. Wasting attended by fever, when not accounted for by some obvious condition, such as empyema, bone or joint disease, suggests the possibility of tuberculosis of the lungs. If to these symptoms cough be added, the suspicion becomes strong; and if to wasting, fever, and cough we add hæmoptysis, the suspicion becomes a practical certainty. The pyrexia at the outset most often takes the form of a moderate afternoon rise, and is frequently much under the influence of exercise and rest.

Opinions differ as to whether well-marked and obtrusive dyspepsia is, in the absence of other symptoms, a frequent precursor of phthisis. Upon the whole, my opinion would be in the negative. If we reflect upon the history and progress of the great army of dyspeptics, it would appear that the percentage of them who become tubercular is a small one, smaller than might have been expected on the view that dyspepsia involves 'lowered vitality,' and hence diminished resisting power to disease. As a broad rule, the dyspeptic remains a dyspeptic, and does not become tubercular. There are, however, exceptions to this rule. There is a type of dyspepsia where the gastric symptoms are not very marked, but where the patient becomes anæmic and wastes. A not inconsiderable proportion of such cases ultimately become tubercular. The voluble dyspeptic who complains loudly of many miseries, but retains a fair degree of nutrition (the type immortalized in the letters of Thomas Carlyle), seems to me in no greater danger of tuberculosis than the healthy man. If tuberculosis be suspected in a case of dyspepsia, the thermometer will be one of our chief aids in diagnosis.

Pain in the chest, especially in the apical region, is common in incipient phthisis. It is often due to local pleurisy; sometimes, I think, to nervous erethism. It may be accompanied by local tenderness.

Persistent frequency of the pulse in a young adolescent,

not otherwise accounted for, should lead to inquiry for phthisis. It may be present in incipient cases apart from pyrexia.

The combination of the following symptoms—languor, debility, and proneness to sweat on slight exertion—requires to be interpreted with caution. When such conditions are present, the suspicion of phthisis is apt to arise; but in the absence of cough, wasting, afternoon pyrexia, acceleration of the pulse, or hæmoptysis, I do not think there is strong foundation for it. Obviously, under such circumstances, the indications afforded by the stethoscope, thermometer, and weighing-machine ought to be scrutinized with special care. It is unfortunate to suggest the fear of tuberculosis to a patient who is only suffering from some form of nervous debility. We may get assistance in such cases by asking such questions as—Is this condition of debility recent or of long standing? A condition of debility of a standing of months or years, unaccompanied by any definite signs or symptoms of phthisis, is unlikely to be tubercular. Can any adequate cause be assigned for it—*e.g.*, over-study, anxiety, prolonged nursing of sick relatives, disappointment? This is not a point to be pressed for, but we may say that incipient phthisis more often *appears* uncaused than does nervous debility.

Chlorosis is often viewed by anxious parents as the harbinger of phthisis, usually without sufficient cause. Chlorotic girls do not in any considerable proportion become tubercular. Trousseau thought the contrary.

I have always a strong suspicion of incipient phthisis in the following type of case, of which I have seen many examples: The patient, generally an adolescent, protests that he is practically quite well, and submits to examination with reluctance. He at first denies all symptoms, but after a little conversation recollects that he has a slight cough in the mornings, that on one or more occasions he has seen

traces of blood in the sputum (but 'he is sure it came from his teeth,' or 'he had a bleeding from the nose the previous day'), he has lost weight lately (but this is 'due to having cycled so much,' or to late hours of work, or so forth), he perspires rather freely at night (but this 'is due to the hot weather'). The chest is found to be rather flat and inexpansile, the temperature is slightly raised in the afternoons, the patient is thin, he has a bright eye, a slight flush over the malar bones, his eyelashes are long and silky, his hands are rather hot, his intelligence is alert and above the average. We all know the type. In such cases the physician does not need to be a wizard to detect the projected shadow of tuberculosis.

D. The Examination of the Sputum.—It is hardly necessary to insist upon the primary importance of an examination of the sputum in all cases of suspected incipient phthisis. This examination may at once set all doubt at rest. Numerous cases have occurred in my practice in which tubercle bacilli were detected, although no definite physical signs could be made out, and the symptoms were merely suspicious. The diagnostic characters of the sputum have already been fully dealt with.* It will suffice to recapitulate here the chief working rules which we call to our aid in connection with the examination of the sputum in incipient phthisis:

1. Bacilli may be present in the sputum from the earliest stage. This is exceptional, but less rare than was formerly thought.

2. Bacilli are occasionally present in the blood of early hæmoptysis; more often not.

3. Bacilli are present in the great majority of cases of active disease attended by free expectoration; but exceptions to this rule are not unknown.

* See Lecture VII.

4. Bacilli are often absent from the sputum in acute miliary tuberculosis, in fibroid tuberculosis, and in quiescent cases of ordinary chronic phthisis.

5. Failure to find bacilli in the sputum, except after repeated trials, is entitled to slight weight in diagnosis.

6. The number of bacilli in the sputum has in general some relation to the activity of the case, but this rule is not without exceptions.

7. Cases of quiescent phthisis where the constitutional state is quite favourable may still present bacilli in the sputum.

8. A declining number of bacilli in the sputum is in general of favourable import, but many casual fluctuations occur, and are of little significance.

E. Physical Signs in Incipient Phthisis.—These may be entirely absent, or slight and indefinite, or suspicious and gradually approximating to specific characters. We shall do well, when the signs are slight, to attach more weight to their localization than to their character. Signs, whatever their precise nature, which tend to be confined to one apex; or, if present at both apices, are more pronounced on one side than upon the other; or which involve chiefly the upper portion of the lower lobe or lobes; or which begin in a median or basic position, but creep upwards towards the apex: are almost certainly due to tubercle.

The earliest physical signs in incipient phthisis are not always the same. Tubercular invasion of any organ is 'asystemic.' It does not respect anatomical boundaries or physiological frontiers. Bacilli reach the lung by two channels: (*a*) By way of the blood or lymph stream; (*b*) by way of the air passages. In the former case, the alveoli are first affected; in the latter, the finer bronchi and bronchioles. The earliest effects of tubercular invasion of the lungs would, therefore, seem to be catarrh limited to certain

alveoli or bronchioles. It is clear that flattening, dulness, and consonating râles, must belong to a later, and comparatively advanced, stage of the malady. These signs presuppose changes in the lung which require time and alterations in the pulmonary parenchyma. We should expect, *a priori*, that the earliest signs of commencing phthisis would be: (*a*) Certain alterations in the breath sounds; (*b*) fine, bubbling (soon to become crepitant) râles. Clinical experience will be found to confirm this expectation if we rid our minds of the fallacious picture given in many text-books of the physical signs of incipient phthisis —viz., flattening, dulness, localized crepitant râle. It is most important to recognise that when these signs are present the case is no longer incipient, but relatively advanced.

(*a*) *The Breath Sounds.*—Much attention should be devoted to the quality of the breath sounds in a case of suspected incipient phthisis, and we must be fully alive to the limits of physiological variation. The following rules will be found useful:

1. The alternations in the quality of the breath sounds are always local, and in the great majority of cases they are apical in situation.

2. The breathing may be harsh, weak, or cog-wheel in character.

3. The expiration is usually somewhat prolonged.

4. When the breathing is weak, coughing often increases its distinctness.

It has been doubted whether harsh breathing or weak breathing is the earlier condition. After much attention to the subject, I am inclined to think that there is no fixed rule. Both types of breathing are frequent, both are often early. It is doubtful if either is the invariable antecedent or sequent. It cannot, I think, be said that harsh breathing always gives way to weak breathing, or *vice versâ*. Some

authorities are of opinion that where extensive areas of tuberculization are present in the lungs, weak breathing is present over the older, harsh breathing over the more recently involved, areas. Cog-wheel breathing occurring alone is not a trustworthy sign, but it is certainly common in incipient phthisis, and cannot be dismissed as a sign of absolutely no significance.*

(*b*) *Râles.*—A fine bubbling, or obscurely crepitating, râle, limited to a small area of one apical region, often audible only after coughing, is usually an early sign. When we hear a limited, rather coarse, and definitely consonating râle, the case is no longer incipient. This râle may vary from day to day. It may come and go, and this variability may raise unfounded doubts regarding the correctness of the diagnosis. It is important to insist that the presence of this form of râle is not necessary to the diagnosis. If, on the other hand, the râle is fairly constant from day to day, the diagnosis is confirmed.

In a small number of incipient cases of phthisis I have heard over a portion of one apical region rhonchi and soft bubbling râles, not distinguishable from those present in ordinary bronchitis. It is the limitation of such sounds to one apical region, and their persistence, which give them significance.

I have stated my opinion that when well-marked dulness on percussion is present at one apex the case is not usually incipient. What are we to say of the slighter departures from normal percussion resonance? Considerable assistance may be obtained by attention to these minuter shades of difference in the percussion note. On the side of commencing tuberculization it may be possible to satisfy oneself that the percussion note in suprascapular, supraclavicular,

* I cannot agree with Gee that cog-wheel breathing 'is a sign of no practical importance' ('Auscultation and Percussion,' third edition, p. 127).

or infraclavicular regions, though not dull, has a slight flatness of tone, and that the pitch of the note is raised. It may also be possible to determine that the highest point of pulmonary resonance extends less above the clavicle on the affected than on the sound side. The 'sense of resistance' may also be increased on the affected side. Strümpell is of opinion that 'he who lays too great stress on the uncertain results of percussion will often make a false diagnosis.'* This is true in so far that slight variations in the percussion note, *if unaccompanied by auscultatory signs*, require to be appraised guardedly. It is also true, however, that a slight, but definite, departure from normal percussion resonance at one apex strengthens to a material extent the inferences which may be safely drawn from slight or indecisive auscultatory signs.

The following passage from the well-known and excellent work of Fowler and Godlee calls for some observations: 'The diagnosis may be attended with difficulty when the symptoms suggest tubercular disease, but the physical signs are of doubtful import, and there is no expectoration. The cases in which the above conditions are fulfilled are very often delicate-looking young women suffering from anæmia. In such cases, and in all others included in this category, it is a golden rule never to make a diagnosis of tuberculosis from doubtful physical signs, and particularly not from a single sign. The examination of the chest must be systematic; the evidence obtained on inspection and palpation must be considered with, and must agree with, that derived from percussion and auscultation. When the signs separately considered are of doubtful import, but are yet in harmony, and all point in one direction, a positive opinion may be given. But the observer *must beware of relying upon auscultation with its many fallacies*,† and a definite opinion should

* 'A Text-book of Medicine,' English edition, p. 252.

† The italics are mine.

rarely be given in doubtful cases, unless the record of the morning and evening temperature for at least a week is available.'*

Much of the foregoing is quite sound, some of it goes without saying, but if we are always to wait until all the above conditions are fulfilled, we shall seldom make a diagnosis of incipient phthisis in cases of any obscurity. Feeling strongly as I do the disastrous results of a procrastinating policy in these cases, I am unable to subscribe to rules which would make an early and prompt diagnosis often impossible. We need not expect that in incipient cases the signs obtainable by inspection, palpation, percussion, and auscultation will present a consistent and congruent whole. To distrust auscultation is not a safe rule. Auscultation is, on the whole, our best resource in these cases. To withhold an opinion in the absence of afternoon pyrexia is not always necessary. Tuberculization of the lung, as of other organs, may proceed a considerable way without pyrexia. We must act here upon the rule which has such a wide applicability in practical medicine—viz., attach much positive weight to the presence of certain signs; attach little negative weight to the absence of those signs. In a doubtful case, the presence of afternoon pyrexia is confirmatory evidence of much value. Its absence is to be noted, but must not be allowed too much weight in diagnosis. It is to be noted that while Strümpell cautions us against over-confidence in the results of percussion, Fowler advises us to distrust auscultation. In cases such as those under consideration, which are often of extreme difficulty, but where the necessity for a prompt diagnosis is urgent, these general maxims do not really help us. No doubt either percussion or auscultation may mislead, but nothing is gained by discrediting these methods in advance. It is certainly true that where the signs of incipient phthisis are slight and vague we should attach much weight to the

* Fowler and Godlee, 'The Diseases of the Lungs,' p. 378.

corroboration of one class of sign with another. But this is a self-evident principle.

Leube is of opinion that an apical catarrh is almost invariably tubercular, and in this opinion all will concur. The possibility of a simple (*i.e.*, non-tubercular) catarrh of one apex may be admitted, but almost nothing is known of such cases, and if the catarrh persist or recur, we may accept its tubercular origin with the most insignificant risk of error. Leube believes that some degree of percussion dulness is usually present in these cases, and that the diminution of the height of percussion resonance above the clavicle is highly significant. He proceeds as follows: 'Still more important is it for the certainty of diagnosis when auscultatory abnormalities correspond to the departure from the normal as regards percussion. Even the slightest alterations in the respiratory murmur are in such cases sufficient: cog-wheel breathing; prolonged expiration; weak, harsh, or rough vesicular sound; indeterminate vesicular sound. If the latter has a bronchial character or if râles are present, although such râles may be isolated and not consonating in character, the slightest dulness on percussion at the apex of the lung possesses much diagnostic significance.'*

Of cases of apical catarrh where definite proof of tuberculosis was wanting I have seen several examples, but in no single case do I feel certain that it was non-tubercular. In one instance in my practice, a well-defined apical catarrh in a young lad was followed by apparently complete recovery and disappearance of physical signs, but eight or nine years subsequently he died of phthisis.

In connection with these cases, croupous pneumonia affecting one apex will not give rise to any serious difficulty. Broncho-pneumonia confined to one apex is almost certainly tubercular.

* William von Leube, 'Specielle Diagnose der Innern Krankheiten,' vol. i., p. 149.

No rule is more borne in upon us by an extended experience than that we should regard with extreme suspicion localized signs, which are limited to, or tend to concentrate themselves upon, one apex, either of the upper or the lower lobe.

The increased conduction of the heart sounds to one apex has been thought by some observers to possess some diagnostic importance, but I have not found this sign of any definite value in practice. Increase of tactile fremitus, on the other hand, in one apical region is always deserving of close attention. We must carefully remember that in health the fremitus is often much more marked upon the right side than upon the left. This sign will assist us more in investigating a suspicious left apex than a suspicious right apex.

Pityriasis versicolor is common on the chest and back of tubercular patients.

Variations in the Physical Signs in Incipient Phthisis.—The physical signs in incipient phthisis may vary considerably from time to time, and the practitioner may sometimes have doubts regarding the correctness of a diagnosis which he has arrived at on what seemed conclusive evidence. Especially may the adventitious sounds vary, being now more numerous and characteristic, and, again, few, absent, or untypical. Probably such variations may be related to meteorological conditions, transient variations in the patient's general health, and, perhaps, other causes. Variations in the area of distribution of adventitious signs is not always a safe index of variations in the activity of tuberculization. Transient inflammatory causes often produce such variations. Compensatory emphysema of the sound lung may produce important variations in the percussion note and the vesicular murmur in certain parts of the chest. Intercurrent attacks of bronchitis, broncho-pneumonia, or croupous pneumonia will, of course, produce their characteristic physical signs.

*The Röntgen Rays in the Diagnosis of Incipient Phthisis.**— The general question of the value of the Röntgen rays in the diagnosis of pulmonary disease has already been fully considered. For the convenience of the reader, I shall here briefly recapitulate the rules regarding the application of this method in cases of incipient phthisis:

1. A slight shadow may be discernible in the apical area of the skiagram at a very early stage of the disease. Whether such a shadow can be recognised before there are any definite signs on percussion and auscultation is doubtful.

2. A limited excursion of the diaphragm on one side of the thorax, if not otherwise accounted for, affords ground for suspicion that the lung on that side is undergoing tuberculization.

3. The Röntgen rays may afford interesting facts and evidence confirmatory of that obtained by the usual methods, but there is not at present any sufficient ground for maintaining that this method of examination can take a place beside percussion and auscultation in the diagnosis of pulmonary diseases.

4. The sources of fallacy in the interpretation of the skiagram of pulmonary diseases are numerous, and require careful elimination before any assured conclusions can be drawn.

* See p. 64 *et seq.*

LECTURE XVI

CONDITIONS WHICH SIMULATE PULMONARY TUBERCULOSIS

SUMMARY:

Three groups of cases which simulate pulmonary tuberculosis:

A. A group of cases which may closely simulate phthisis, but which, owing to their infrequency, give rise to little difficulty in practice, viz.: Actinomyces of the lungs, malignant disease of the lungs, syphilis of the lungs, hydatids of the lungs.

B. A group of cases which may closely simulate phthisis, but in which, owing to well-marked differences in history, symptoms, and physical signs, the differential diagnosis is, as a rule, easy, viz.: Pulmonary collapse, bronchiectasis, pulmonary embolism.

C. A group of cases which may closely simulate phthisis, and in which the differential diagnosis, though often easy, sometimes presents great difficulty, viz.: Broncho-pneumonia, pleurisy, bronchitis, asthma, anæmia, neurotic dyspepsia.

Rules for differential diagnosis, canons, and cautions.

The conditions which simulate pulmonary phthisis are many. It will be of some practical service if we classify them into groups as in the foregoing summary.

We have a few rare conditions where the simulation of phthisis may be close, but which practically give rise to little

difficulty—viz., actinomyces, malignant disease of the lungs, syphilis, hydatids.

We have certain conditions which are upon the whole readily differentiated — viz., pulmonary collapse, bronchiectasis, pulmonary embolism.

Finally, we have some conditions which, although in the main easily differentiated, yet exceptionally cause serious difficulties in diagnosis—viz., broncho-pneumonia, pleurisy, bronchitis, asthma, anæmia, and certain neuroses.

A. The First Group includes the following cases: Actinomyces, malignant disease, syphilis, hydatids.

Actinomyces.—The lungs are affected in about 13 per cent. of cases of actinomyces: in only a very small proportion of such cases is the pulmonary affection primary. The course of these cases presents a somewhat close simulation of that of phthisis. While the diagnosis of actinomyces can be certainly made only by detecting the ray-fungus either in the sputum or in the discharge from an abscess, attention to the following considerations will usually serve to prevent confusion between this rare disease and phthisis:

1. The patient's occupation. About 75 per cent. of cases of actinomyces occur in persons who in the course of their occupation have to deal largely with cereals—viz., labourers, millers, farmers, and grooms. The source of infection is usually one of the grasses, especially the genus *Hordeum*.

2. The presence of actinomycotic lesions in other organs —*e.g.*, jaw, tongue, mouth, intestines, liver, skin.

3. The detection of the ray-fungus in the sputum or in the pus from an abscess.

4. The involvement of the chest wall, which is frequent in actinomycosis.

5. The localization of the physical signs in the lungs. In actinomycosis the physical signs are usually basic; they do not tend to invade the apices, nor do they exhibit that

'march of the lesion' which, in a certain proportion of cases, is characteristic of chronic phthisis.

6. The favourable influence of iodide of potash upon actinomycosis.

7. In actinomycosis there are no elastic fibres found in the sputum.

It must be borne in mind that actinomycosis and pulmonary tuberculosis are not uncommonly found in the same patient.

MALIGNANT DISEASE OF THE LUNGS.—This subject will hereafter be fully considered * I shall here consider only the question of its differentiation from pulmonary tuberculosis.

The simulation of phthisis by malignant disease of the lungs may be very close. In both conditions we may have cough, hæmoptysis, pyrexia, night-sweating, and wasting. The physical signs of the two diseases, though usually more or less contrasted, cannot always be distinguished with certainty at an early stage. The history, although usually of great importance, is not always decisive.

We may expect to differentiate malignant disease of the lungs from phthisis by attention to the following points, which are characteristic of the former condition :

1. There is in most cases a history of pre-existing malignant disease in the mamma, uterus, bones, glands, or other organs.

2. The bronchial, supraclavicular, infraclavicular, or axillary glands are usually involved.

3. Signs of pressure by enlarged glands upon veins, nerves, or bronchi may be present.

4. Pain is usually a prominent feature. It is severe, persistent, and localized.

5. The physical signs vary much. They may simulate phthisis in presenting areas of consolidation, but these areas are often median or median-basic in position, and do not

* See Lecture XXII.

follow the laws of localization which are more or less characteristic of phthisis. The right upper lobe is often the seat of cancer. If the growth be of considerable size, there will be some enlargement of the corresponding side of the thorax. Pleural effusion may be present in either malignant disease of the lungs or tuberculosis, and in either the fluid may be serous or hæmorrhagic.

6. The sputum in malignant disease may be mucoid, muco-purulent, or sanguineous. It may contain carcinomatous fragments. It is sometimes of the 'raspberry' or 'red-currant' jelly type, which is rare in tuberculosis. It is not often abundant. Tubercle bacilli are, of course, absent.

7. Cachexia is rapid, marked, and progressive. A few months commonly serve to bring the patient to a condition of marked dyscrasia, but in some cases a fair degree of nutrition is maintained until near the end. The rapid downward progress of malignant disease of the lungs is usually in marked contrast to the chronic course, the periods of amendment or quiescence, which are common in phthisis.

8. Treatment is quite unavailing, and there is little or no response to the tonic and fortifying lines of treatment which so often produce a marked effect upon phthisis.

PULMONARY SYPHILIS.—Pulmonary syphilis is a rare condition. Only a few undoubted cases are on record, but some recent observers are inclined to regard the condition as much more common than has generally been supposed.* Its differential diagnosis from phthisis will be assisted by attention to the following points:

1. The history of syphilitic infection. It need hardly be pointed out that such a history does not make the existence of tuberculosis improbable. Pulmonary syphilis appears from five to fifteen years after the primary infection (Fournier).

* 'Nothnagel's Encyclopædia of Practical Medicine,' 'Tuberculosis,' pp. 472, 473.

2. The co-existence of syphilitic lesions of larynx, bones, liver, spleen, or testis.

3. The localization of the lesions, which do not present the well-known peculiarities of tuberculosis, either as regards the seat or seats of the primary deposit, or the 'march of the lesion.' Syphilitic lesions affect chiefly the middle portions of the lungs.

4. The nature of the lesion. Tubercular lesions tend to excavate, syphilitic lesions do not. Softening of a gumma may, however, lead to cavity formation.

5. The presence in an important proportion of syphilitic cases of stenosis of the trachea or bronchi.

6. The absence of tubercle bacilli from the sputum, which is often profuse and fœtid.

7. The general progress of the case, the constitutional condition of the patient, and the results of treatment.

Tuberculosis not uncommonly develops in the course of chronic syphilis.

HYDATIDS OF THE LUNG.—Hydatid disease of the lungs may closely simulate phthisis. It may cause such symptoms as cough, expectoration, dyspnœa, fever, emaciation, and hæmoptysis. If to these symptoms be added the physical signs of excavation in one apex, it is evident that the distinction from phthisis may be very difficult.

Attention to the following rules will assist the diagnosis:

1. A close association with domestic animals will probably be a feature of cases of pulmonary hydatids.

2. Hydatids more often involve the base of the lung than other parts, and the right lung is more frequently affected than the left.

3. Hydatid disease of other organs, especially of the liver, usually co-exists with the pulmonary affection.

4. The auscultatory signs in hydatids are often slight and uncharacteristic, but may be those of excavation.

5. The sputum in hydatids is often fœtid or bloody, and

may contain hooklets or pieces of the hydatid membrane, in which case the diagnosis is easy.

6. Shortness of breath is, on the whole, more marked in hydatids than in phthisis in proportion to the other symptoms.

7. Signs of rupture of the sac—viz., dyspnœa, hæmoptysis, and expectoration of hydatid fluid or membrane—may occur in pulmonary hydatids.

B. The Second Group includes the following cases—viz.: Pulmonary collapse, bronchiectasis, pulmonary embolism.

Pulmonary Collapse.—This condition can be usually diagnosed from phthisis by attention to the following points:

1. *The History of the Case.*—In pulmonary collapse there is usually a history of—

(*a*) Such conditions as acute bronchitis, broncho-pneumonia, measles with pulmonary complications.

(*b*) Pressure on the lung or bronchi by tumour or aneurysm.

(*c*) Foreign body in the larynx or bronchi.

2. *The Character of the Symptoms.*—Dyspnœa is often a prominent feature in pulmonary collapse; not usually prominent in phthisis. Hæmoptysis is rare in collapse. Pyrexia may be absent in collapse, or, if present, will be conditioned by the antecedent disease, and will not present the characters of phthisical pyrexia.

3. *The Nature of the Physical Signs.*—In collapse we usually find retraction of the side, weak or feebly bronchial breathing, and a diminution of vocal fremitus and vocal resonance.

4. *The Localization of the Physical Signs.*—In collapse the signs are usually basic or median-basic; very rarely apical.

Bronchiectasis.—In this condition there will probably be a history of chronic bronchitis, pleurisy, or pulmonary cirrhosis; the sputum is usually abundant, fœtid, discharged in large quantities at considerable intervals of time, and does not contain tubercle bacilli or elastic fibres; hæmoptysis is not uncommon; the constitutional condition is often fairly

good; pyrexia may be present, owing to septic absorption, but does not present the characters of phthisical pyrexia; the physical signs are usually basic in situation and unilateral in distribution, and consist of cavernous or diffuse blowing breathing, and amphoric phenomena. The character of the sputum is the most important point in the differential diagnosis. It must be remembered that bronchiectasis is a common feature in advanced phthisis. In such cases it is usually apical in seat, and its presence may modify the physical signs in important particulars, and may sometimes create unfounded doubts as to the accuracy of the primary diagnosis. The history of the case, the examination of the sputum, and the constitutional symptoms, will serve to clear up these cases.

PULMONARY EMBOLISM.—Pulmonary embolism may give rise to signs and symptoms which may lead to difficulty in the differential diagnosis from phthisis. Our concern here is not with large emboli, which cause such violent symptoms that their diagnosis is easy, but with small or medium-sized emboli, which sometimes give rise to hæmoptysis, pyrexia, and the formation of abscesses in the lungs. The physical signs may be those of consolidation and œdema, and the distinction from phthisis may in exceptional cases present some difficulty. Our main reliance must be—(*a*) on the detection of a cause for embolism, *e.g.*, valvular disease of the heart or phlebitis; and (*b*) on the mode of onset, which in embolism will be sudden, but may be ill-marked where the emboli are small; and (*c*) on the localization of the lesions, which in embolism will not present any of the features characteristic of the distribution of tubercular lesions. According to West, a rigor sometimes occurs at the time of the embolism, even when the embolus is of a non-infective character.* Repeated rigors are common when the emboli are septic or infective in nature.

* S. West, *op. cit.*, vol. i., p. 357.

My experience of pulmonary embolism is not large, and I have known considerable difficulty to arise in its diagnosis. The absence of tubercle bacilli is, of course, of much importance.

C. The Third Group of cases includes the following: Broncho-pneumonia, pleurisy, bronchitis, asthma, anæmia, certain neuroses.

Some of these are a frequent source of difficulty in the differential diagnosis of pulmonary tuberculosis, others only exceptionally require consideration.

Broncho-Pneumonia. — The distinction of broncho-pneumonia from phthisis is often difficult and not seldom impossible. Nor is this fact surprising. Phthisis *is* a form of broncho-pneumonia, but of special character and specific origin. It is well to recognise that the physical signs of broncho-pneumonia may be identical with those of phthisis, and that in many cases the symptoms of the two diseases may also be indistinguishable. Assistance may be obtained by attention to the following points:

1. The history of broncho-pneumonia will probably cover a period of days or weeks; that of phthisis probably a period of weeks, months, or even years.

2. The co-existence of some of the frequent causes of broncho-pneumonia, *e.g.*, measles, whooping-cough, rickets, —is important. It must be borne in mind, however, that some of these conditions are not rarely associated with phthisis.

3. The symptoms of broncho-pneumonia are in general more acute and more obtrusive than those of phthisis.

4. The curve of the temperature may assist the diagnosis, though it must be admitted that in some cases of broncho-pneumonia the chart is indistinguishable from that of phthisis.

5. The absence of tubercle bacilli from the sputum is very important, but, as before pointed out, must not be allowed that negative weight which rightly belongs to the presence of bacilli as a positive sign.

6. The physical signs of broncho-pneumonia are generally basic or median in seat and bilateral from the first. The physical signs of phthisis are usually apical or interscapular in seat, and at first unilateral, becoming later bilateral.

The question of the differentiation of broncho-pneumonia and phthisis generally arises in the following form :

A child has passed through an attack of measles, whooping-cough, typhoid fever, or other disease, in which pulmonary complications have been present. The convalescence is lingering, the constitutional condition indifferent or bad, and certain physical signs persist in the lungs. The question arises, Is the case simply one of delayed convalescence, or has tuberculosis supervened ? This question cannot always be answered with confidence until some time has elapsed, but the following considerations will serve to assist the diagnosis:

1. *The Character of the Sputum.*—The presence of tubercle bacilli is, of course, decisive of tuberculosis. Their absence on repeated examination would point to the probability of broncho-pneumonia.

2. The physical signs must be cautiously appraised. The principal rules have already been given. In broncho-pneumonia the physical signs are almost always bilateral from the first, which is a point of much diagnostic importance, and they show no preference for the apical region, but rather the contrary. Any tendency for the physical signs to limit themselves to the apical region has, in my experience, almost invariably coincided with the development of tuberculosis. This rule cannot, however, be reversed with safety. I have seen a small number of cases where the physical signs began at the base and gradually spread upwards, and in which the subsequent development of the cases left no doubt of their tubercular character. Such cases are, however, rare.

Dulness, bronchial breathing, and crepitant râles are equally characteristic of broncho-pneumonia and phthisis.

Signs of commencing excavation, not always easy of recognition, would make phthisis practically certain if abscess and gangrene could be excluded.

3. The symptoms require to be interpreted cautiously. Hectic fever, wasting, and night-sweating may all be well marked in non-tubercular cases. Hæmoptysis, if present, would go far to determine the diagnosis, but this symptom is often absent in the commencing phthisis of young subjects.

A point upon which I have sometimes relied with success is the following: The tubercular patient is *less obviously ill* than the patient with simple broncho-pneumonia; the symptoms and the physical signs are less accordant in the former than in the latter case. This rule requires to be applied with caution, but it has a certain value.

When all has been said, the differential diagnosis of broncho-pneumonia from phthisis is sometimes one of the most difficult tasks of the physician. Osler does not overstate the difficulty when he says: 'It is well to emphasize the fact that there are many cases of broncho-pneumonia in children which time alone enables us to distinguish from tuberculosis.'* The practitioner would do well to be guarded in giving an opinion upon these cases, and in particular to abstain from giving a diagnosis of tuberculosis until he feels himself on sure ground.

Pleurisy.—Bearing in mind that a large proportion of pleurisies are tubercular in origin, and that a certain degree of pleurisy nearly always accompanies phthisis, it is in no way surprising that great difficulty may be encountered in differentiating the two conditions. Sometimes we may be seeking for a means of distinguishing two conditions which are fundamentally the same.

Nevertheless, the question of the differentiation of pleurisy from phthisis is one of great practical importance, which commonly arises in one or other of the following forms:

* Osler, 'Practice of Medicine,' p. 646.

(*a*) During the course of an acute pleurisy crepitations are heard at the apex on the affected side, and the question of the existence of phthisis arises. It should be a cardinal rule to give no opinion regarding these cases during the persistence of the effusion. The signs in question are often due to adhesions of the lung at the apex.

(*b*) A patient has passed through an attack of acute pleurisy; convalescence is tedious, expansion of the lung is imperfect; perhaps some residual effusion persists. Are we dealing with the residue of a so-called simple pleurisy, or has tuberculosis supervened?

This question does not always admit of a definite answer. The sputum must, of course, be repeatedly examined. A persistently hectic temperature is suspicious of tubercle, but the absence of fever may be consistent with either diagnosis. If fluid be present, it should be examined both microscopically and, if possible, by inoculation. The fluid may be serous, bloody, or purulent. A serous effusion throws no light on the differential diagnosis. A bloody effusion lends weight to the suspicion of tubercle. A purulent effusion is usually non-tubercular. The bacillus of tubercle is very rarely found in tubercular effusions. The results of inoculation experiments may be decisive. The local physical signs may present nothing pathognomonic, but any tendency of the signs to extend towards the apex, or any development of fresh signs at one or both apices, is strong confirmation of the suspicion of tubercle.

When a positive opinion is impossible, it is better to lean towards the more hopeful view of these cases, and to treat them simply as chronic pleurisies. The treatment thus adopted—viz., hygienic measures, good feeding, pulmonary gymnastics, upland or mountain air—will, in most cases, be useful, even if tuberculosis should ultimately prove to be present. If the case be tubercular, it will often be found that moderate exercise raises the temperature one or two degrees.

Bronchitis. — The differentiation of bronchitis from phthisis is usually a simple matter. In bronchitis the physical signs are bilateral in distribution, usually equally marked on the two sides, more pronounced at the base than at the apex; the tendency is to over-expansion and hyper-resonance of the chest; the inspiratory murmur is roughened; the râles, in the absence of complications, are bubbling and not crepitant. In all these points phthisis presents a contrast. The physical signs are at first unilateral, never equally marked on the two sides, more pronounced at the apex than elsewhere; the tendency is towards retraction and deficient resonance; the expiratory murmur is roughened; the râles are crepitant in character.

The departures from the above rules are few, but it must be admitted—

(*a*) That in certain extremely rare cases bronchitis is for a time unilateral in distribution.*

(*b*) That during convalescence from bronchitis the signs sometimes become localized before clearing up. If in such cases the constitutional condition be unsatisfactory, the suspicion of phthisis is likely to arise.

(*c*) That phthisis may set in with physical signs not distinguishable from those of ordinary bronchitis.

In such cases we may obtain assistance by attention to the following points:

1. The presence or absence of tubercle bacilli in the sputum.

2. The expansion of the chest — normal or increased in bronchitis, diminished in phthisis.

3. The state of the vocal fremitus—normal in bronchitis, probably increased in phthisis.

4. The percussion sound — hyper-resonant in bronchitis deficient in resonance in phthisis.

* S. Gee, 'Auscultation and Percussion,' third edition, p. 202; Lenhartz in Ebstein and Schwalbe's 'Handbuch der Praktischen Medizin,' Bd. i. 1., p. 109.

5. The character of the râles — bubbling in bronchitis, crepitant in phthisis.

6. The temperature, weight, presence or absence of hæmoptysis, night-sweating.

A hectic temperature, marked wasting, and night-sweating may all be present in the bronchitis of debilitated subjects. Hæmoptysis is strong presumptive proof of phthisis.

In rare cases phthisis may set in with the physical signs of general bronchitis, and the diagnosis may remain in doubt until the examination of the sputum, the occurrence of hæmoptysis, or the change from bubbling to crepitant râles, clears up the case.

ASTHMA. — The association of asthma and pulmonary tuberculosis is a rare one, and the differentiation of the two conditions is usually obvious. Nevertheless, there are exceptions to this rule, and these exceptional cases are, from their rarity, apt to mislead. The following type of case is occasionally met with in practice: A limited and retrocedent tubercular lesion of one apex is attended by fibroid changes and bronchiectasis. Attacks of spasmodic dyspnœa occur, and the case is mistaken for one of asthma. In such cases the differential diagnosis is usually easy. The history of the two types of case is different. The 'asthma' in the tubercular case is rarely typical. There is more or less debility or emaciation; the temperature may assist; tubercle bacilli may be found in the sputum, but are often absent in these cases; localized signs at one or both apices will probably be present. Allowance must be made for the possibly misleading physical signs of bronchiectasis in the neighbourhood of the tubercular lesion.

It should be a rule to examine the sputum for tubercle bacilli in cases of supposed asthma, and to exercise special care when the patient is young, and when debility or emaciation is present.

ANÆMIA, DYSPEPSIA, VARIOUS NEUROSES.—These condi-

tions, which may be conveniently considered together, often simulate incipient phthisis, and the difficulties of a differential diagnosis may be great. In one type of case anæmia and dyspepsia, in another type nervous debility, are the prominent features. In many cases the two groups of symptoms are conjoined.

A typical case may be sketched as follows: A young girl, eighteen to twenty-five years of age, has been getting out of health for weeks or months. She has lost flesh, and is becoming anæmic. Dyspepsia, often under the form of pronounced anorexia, is marked. She has a short cough, and occasionally there has been a little blood in the expectoration. The temperature is normal, there is no night-sweating, and there are no physical signs in the lungs. Such a case may simulate phthisis closely, and if there should be tuberculosis in the family, the fears of the patient and of her friends are naturally aroused.

A case such as the above may be: (1) Genuine incipient phthisis; (2) hysteria, with anæmia and debility; (3) nervous mimicry (*i.e.*, phthisiophobia). It is important to observe that the simulation by pseudo-phthisis (if I may employ so questionable a term) of the genuine disease may be very close. Anæmia, wasting, debility, and dyspepsia may be marked in both cases. Cough and hæmoptysis may also be present in both. Yet, on carefully analyzing the symptoms, points of contrast will usually emerge. The cough of nervous mimicry is more constant, obtrusive, hard, and clanging than that of incipient phthisis, and there is either no expectoration or it is chiefly composed of saliva. If hæmoptysis is present in such cases, it is usually produced by the patient sucking her gums, and the blood is watery and mixed with saliva. The spurious cases do not develop pyrexia or night-sweating, and of course there are no definite physical signs in the lungs and no tubercle bacilli in the sputum. In pseudo-phthisis the patient complains much of

her symptoms, in genuine phthisis she often complains little. The former draws our attention to her symptoms, the latter glosses them over; the former courts physical examination, the latter shuns it. If the blood of the alleged hæmoptysis can be inspected, a diagnosis may sometimes be easily made on the lines already indicated. Hysterical young women are fond of bringing handkerchiefs with a few dubious stains to the doctor; the genuine tubercular patient less often does so. The former invites a serious diagnosis; the latter dreads it.

We must be specially on our guard in these cases lest in neurotic or hypochondriacal patients actual disease may really be present. A neurotic girl may become tubercular, like anyone else; and the presence of an obvious neurosis must not blind us to the possibility of actual disease. Yet, in general, we have to deal either with commencing disease or mere neurosis, not with both. *Anorexia nervosa* requires special care. In cases of diagnosis between pseudo-phthisis and actual phthisis, it is not desirable to lay much stress on family history, or upon such points as 'tubercular aspect,' 'phthsical habitus,' 'flat chest,' or the like, for it is precisely where the family history or the patient's physique presents special ground for uneasiness that the spurious cases are most likely to arise. Indeed, we must exercise the greatest caution when we can perceive some obvious ground for the patient's agitations. In these difficult cases it is most important not to be betrayed into a premature diagnosis. A second or a third examination, and the regular use of the thermometer, will often be advisable. Under the circumstances described, parents are often panic-stricken, and fully prepared for heroic measures. If, therefore, the physician without due warrant endorses their apprehensions, the patient may be forthwith haled off to a sanatorium, where the true nature of the case will soon become evident.

LECTURE XVII

PROGNOSIS IN PULMONARY TUBERCULOSIS

SUMMARY :

Increased hopefulness of prognosis at the present day.
Need for caution in prognosis.
Influence upon prognosis of—
(*a*) Age.
(*b*) Sex.
(*c*) Occupation and social position.
(*d*) Family history.
(*e*) Constitutional type of patient.
(*f*) Mode of onset of the disease.
(*g*) State of the lungs.
(*h*) Extension of tuberculosis to other organs.
(*i*) Symptoms.
(*j*) Complications.
(*k*) Response to treatment.
Modes of onset in their relation to prognosis :
(*a*) The insidious onset.
(*b*) The acute onset.
(*c*) The catarrhal onset.
(*d*) The hæmorrhagic onset.
(*e*) The consecutive onset.
Type cases and the prognostic inferences to be drawn from them.

The prognosis in pulmonary phthisis involves peculiar difficulties which are inherent in the nature of the disease. Some writers go so far as to say that no prognosis is possible,

and that the physician of large experience and knowledge will abstain from any attempt to forecast the course of a malady so variable in its progress and so characterized by unforeseen contingencies. This is, however, an over-statement. The prognosis is not impossible, but difficult and always subject to a large margin of possible error.

The keynote of present-day prognosis in phthisis is an increased hopefulness, as compared with the despair of past times. The pendulum would seem, indeed, to have swung from one extreme to the other. It is well to remember that pulmonary tuberculosis is in all cases and under all circumstances a most grave condition, that the most promising cases may do badly, and that even when arrest seems complete, some casual catarrh or wayward hæmorrhage, some intercurrent and wholly incalculable malady, may disappoint apparently well-founded hopes, and relight the embers of quiescent fires. On the other hand, we have all seen the case of the chronic consumptive, with perhaps a cavity in his lungs and an unfavourable constitutional state, who disappoints the gloomy forecast given of his prospects, who makes a good rally, and preserves fair health for an indefinite period.

It is a great gain to recognise that the disease is not necessarily incurable, and that even when treatment cannot cure it can often do much to prolong life, and to render it at least tolerable. Cure in the sense of a complete *restitutio in integrum* is, no doubt, a rare event, and practically confined to incipient and favourable cases. But cure, in the sense of relative cure—*i.e.*, the arrest of the pulmonary mischief and the restoration of the patient to fair general health and working power, for a longer or a shorter period—is not by any means rare. And even when a 'relative cure' is not achieved, varying degrees of improvement, often marked and of considerable duration, are frequently obtained by treatment. Therefore the hopeful tone of modern therapeutics

has much to justify it. We have learnt that pessimism and inaction are unjustifiable, that treatment will, in a fair proportion of cases, repay its heavy price in time and money, and that the consumptive can make his appeal to us upon economic grounds, as well as upon those of our common humanity.

I shall first state the broad principles of prognosis, about which there is more or less general agreement, and I shall then enter more fully into those points which are to some extent *sub judice*. It must be freely admitted that the rules to be laid down are only of general application, and may require modification in any individual case.

Prognosis may be considered under the following heads :

1. Age.—The prognosis is worst in the very young, and in persons over sixty. It is best probably in early adult life—*i.e.*, from thirty to forty years of age.

2. Sex.—According to most observers, the prognosis is more favourable in the male sex. It is probable that this is not an ultimate fact—*i.e.*, a real sexual difference—but that it has relation to the habits, occupation, and mode of life of the two sexes. The influence of child-bearing also requires consideration. Pregnancy appears to retard the progress of phthisis, which often advances with increased rapidity after parturition. Prolonged lactation has naturally a bad effect.

3. Family History.—Many observers are of opinion that phthisis tends to develop at an earlier age, and to run a more rapid course when the family predisposition to the disease is marked, but this is denied by others.

4. Constitutional Type of the Patient, Previous Health, Occupation, Social Position, Habits.—These are factors of great importance. We shall resume their consideration presently.

5. Mode of Onset of the Disease.—An acute onset is always ominous, but does not necessarily portend an acute

course. We shall deal with this question in detail on a later page.

6. STATE OF THE LUNGS.—The larger the area of lung involved; the earlier the extension to the second lung; the more the pathological process tends to softening and excavation—the worse the prognosis. Bronchiectasis is unfavourable. Gangrene, which is rare, portends a rapidly fatal issue.

7. EXTENSION OF THE TUBERCULAR PROCESS TO OTHER ORGANS.—Extension of the tuberculizing process to the larynx, intestines, kidneys, or brain usually involves a definitely bad prognosis. Some laryngeal cases, however, do well. Extension of the disease to the glands has not much weight in prognosis. Extension to the abdominal lymphatic glands is more serious than extension to the cervical, thoracic, or mediastinal glands.

8. THE NATURE OF THE SYMPTOMS.—The following symptoms are unfavourable: Much pyrexia, wasting, marked loss of strength, rapid weak pulse, failure of appetite, inability to digest fatty foods, diarrhœa.

Certain types of pyrexia are specially ominous—*e.g.*, a high continuous temperature; a very high afternoon temperature with a fall to several degrees below normal in the early morning. Profuse and repeated hæmoptysis is decidedly unfavourable, but casual attacks of moderate bleeding have very little prognostic import. Of the various symptoms in phthisis, the most important with regard to prognosis is the state of the digestion. Patients with a healthy appetite and vigorous digestive power fight the disease well, and *vice versâ*.

9. THE COMPLICATIONS.—Amyloid degeneration of kidneys, intestines, or spleen is very unfavourable. Pneumothorax is in a majority of cases fatal within a month, but in certain rare cases it seems to exercise a retarding influence upon the disease. Complications which involve dysphagia are harbingers of the end.

Albuminuria is very unfavourable. It may depend on

lardaceous disease of the kidneys, interstitial nephritis, or tuberculosis of the kidneys.

Ulceration of the bowels, peritonitis, diabetes, typhoid fever, puerperal septicæmia, usually involve a definitely bad prognosis.

Bronchitis, if moderate in degree, has not much prognostic weight; if severe, it is unfavourable for two reasons—viz.: (*a*) It may point to a somewhat wide tuberculization of the lungs, or (*b*) it may serve to embarrass treatment.

Pleurisy is not definitely unfavourable. In the opinion of some observers pleural effusion arising in the course of phthisis points to a probably chronic course of the disease (Osler).

Fistula in ano is not unfavourable.

10. Response to Treatment.—As in many other diseases, one of the most essential facts in throwing light upon prognosis in phthisis is the response of the patient to treatment, which gives us some measure of what we vaguely call his 'vital energy' or 'resisting power.' Some patients at once begin to improve under more favourable conditions of life and the adoption of a methodized régime. Others, not obviously more gravely affected, make little or no response. The difference between the two classes is, *quoad* prognosis, a very vital difference.

The above may serve as a brief outline of the factors which influence prognosis in phthisis. Let us now consider the subject a little more closely, and enter into some of its more obscure and controversial aspects.

Age, sex, and family history are not factors of the first importance from the prognostic point of view. I have known very few favourable cases of phthisis in patients under fifteen years of age. Senile phthisis does badly, as might be expected, and is not always chronic in its course. I am inclined to think that the most favourable age at which phthisis can arise is between thirty and forty. Patients

between these ages sometimes respond well to treatment, while younger patients are apt to exhibit the disease in its more active forms, and older patients present special difficulties as regards the maintenance of nutrition.

Sex, not in itself a matter of great prognostic importance, may have influence indirectly—*e.g.*, in its bearing upon treatment. Phthisis developing in a young girl of from seventeen to twenty-five years of age involves more difficult problems from the therapeutic point of view than a case in the other sex within the same age limits. Travel, change of climate, the adoption of an out-of-door life and a healthy occupation, 'roughing it' in a new country, are obviously more easily available for the male, than for the female, adolescent. Women do as well as men at sanatoria, nor, so far as I am aware, is there any difference in the results obtained in the two sexes at some of the most frequented health-resorts.

Family history requires a cautious appraisement. Its influence has been overrated in the past, but it is in danger of being unjustly ignored in the present. It is probable that its importance depends mainly upon this consideration—viz., that families differ much not only in their tendency to resist, or succumb to, tubercular invasion, but also in their capacity to withstand the disease when once established. A history that several members of a family have succumbed to phthisis either at an early age or after a brief illness is very ominous. Much depends upon whether the activity of the disease in a certain family has been coincident with unfavourable hygienic conditions or not. The disease has been known to ravage a family when living under unhealthy conditions, but to cease entirely on removal of the family to a more favourable environment. In other cases, the disease has been known to continue to infect members of a family remaining at home, but has not pursued those who have left the family circle. It must be admitted, however, that exceptions to these rules seem to occur. It is true, nevertheless,

in not a few cases that 'family predisposition,' being interpreted in modern phrase, might often read 'house infection.'

The patient's temperament, social position, occupation, economic resources, his capability of obtaining early and prompt medical advice and of adopting and persevering in appropriate treatment, are factors of the first importance. It is hardly necessary to labour these points, the relevance of which is obvious. It must be borne in mind that the prospects of treatment are better in cases where the patient's occupation is with just ground held to be largely responsible for his condition than in cases where this circumstance is not present. Early diagnosis and prompt treatment are amongst the most important of all prognostic factors. The constitutional type of the patient is a matter of much moment in prognosis, but it is not easy to lay down rules regarding this point which can be applied in practice. It would be quite erroneous to say that the typical thin, flat-chested consumptive always does badly, or that the well-developed and fairly robust patient usually does well. Some of the worst cases in my experience have belonged to the latter category. West says truly: 'Galloping consumption may develop in a person apparently previously robust; and, on the other hand, in a weakly, delicate person phthisis may last for years.'* The following types of patient seem to be relatively unfavourable:

(*a*) The nervous, irritable type—*eretische constitution*—*i.e.*, the patient whose vaso-motor control is weak, who has hot hands and a moist skin; who sleeps badly, has a capricious appetite, an indifferent digestion, and whose capacity for fatigue is small.

(*b*) The 'lymphatic' type—*i.e.*, the patient who does not at first waste or show much pyrexia, but who is obviously ill; whose symptoms of languor, debility, and malaise are

* S. West, *op. cit.*, vol. ii., p. 535.

out of proportion to the physical signs in the lungs, the pyrexia, or the wasting.

The following types of patient seem relatively favourable:

(*a*) The 'wiry' type—*i.e.*, the patient who is thin but not debilitated, whose energy is but little impaired, whose appetite and digestive power are fair, whose pulse is good, and whose lungs seem prone to fibrosis rather than to softening and excavation.

(*b*) The 'hæmorrhagic' type—*i.e.*, the patient who has either repeated small, or occasional large, bleedings, but whose general health remains fairly good, and whose lungs present few definite signs of disease.

There is nothing more difficult to gauge, nothing more elusive of precise definition, than the 'constitutional type' in any given case of phthisis, yet we feel assured that it is a point of cardinal importance—that, in fact, the patient's personality weighs heavily in determining his prospects in the struggle with disease.

Modes of Onset.—The mode of onset of the disease has considerable prognostic significance. The following modes of onset may be enumerated:

(*a*) *The Insidious Onset—i.e.*, where the disease develops gradually, with ill-defined failure of general health, slight cough, loss of flesh and strength, dyspepsia, and perhaps some rise of afternoon temperature.

(*b*) *The Acute Onset—i.e.*, where the disease develops rapidly, with high fever and rapid wasting.

(*c*) *The Catarrhal Onset—i.e.*, where bronchial catarrh is the leading feature of the initial stage.

(*d*) *The Hæmorrhagic Onset—i.e.*, where hæmoptysis is the earliest, or, at least, the most outstanding, symptom of the initial stage.

(*e*) *The Consecutive Onset—i.e.*, where phthisis obviously follows on measles, whooping-cough, broncho-pneumonia, or typhoid fever.

Speaking generally, I think it may be said that the following modes of onset are relatively unfavourable—viz., the acute onset, the catarrhal onset, and the consecutive onset; that the hæmorrhagic onset is relatively favourable; and that the insidious onset is of doubtful prognostic import. Upon these points, however, there is by no means general agreement. Jaccoud is of opinion that pneumonic phthisis—*i.e.*, one of the cases where an acute onset occurs—has a prognosis which 'at first is more unfavourable than in any other form of the complaint, miliary granulosis alone excepted, while during the time which follows the acute outbreak it is the least serious of all the different varieties.'* Percy Kidd says: 'An acute onset is commonly followed by progressive invasion of both lungs, and has the gravest significance. An insidious, bronchitic, or hæmoptoic onset is more favourable.'† West says: 'The most unsatisfactory cases are those in which the onset has been insidious, and where the patient has been losing health, flesh, and strength, without obvious cause.'‡ Wilson Fox says: 'An acute pneumonic invasion, with rapid consolidation of one apex, rarely escapes softening and excavation.'§

I incline to the view that an acute onset is, in general, decidedly unfavourable, but I have seen striking exceptions to this rule; that the insidious onset has no definite prognostic significance; that the catarrhal onset is, upon the whole, unfavourable; and that the hæmorrhagic onset is, with many important exceptions, somewhat favourable. The consecutive onset is unfavourable, because in such cases we have usually to deal with a debilitated patient. No doubt, a good deal turns upon the nature of the pre-existing disease—*e.g.*, phthisis supervening upon measles or typhoid fever is

* S. Jaccoud, 'Pulmonary Phthisis,' translated by Lubbock, p. 43.

† Allbutt's 'System of Medicine,' vol. v., p. 226.

‡ S. West, *op. cit.*, vol. ii., p. 535.

§ Wilson Fox, 'Treatise on Diseases of the Lungs and Pleura,' p. 842.

probably more unfavourable than phthisis supervening upon influenza or pleurisy.

The rate of progress of the case has much prognostic significance. Evidence that the disease has lasted for a considerable period without serious impairment of the general health, or the development of pronounced signs in the lungs, is very important. In such cases it will usually be reasonable to assume that the patient's resisting power is good, and that the tubercular invasion of the lungs tends towards fibrosis rather than towards softening and excavation. On the other hand, pronounced constitutional dyscrasia and wide involvement of the lungs occurring in a short space of time point in an opposite direction, and are very unfavourable. Until sufficient time has elapsed to give us a clue to the probable rate of progress, prognosis must be very guarded. The course of phthisis is often erratic, and either much better or much worse than seemed at one time probable.

The state of the lungs is one of the most important aids to prognosis. Extensive disease, early involvement of the second lung, the lighting up of fresh foci of tuberculization, the steady increase of the area of physical signs, are of evil import. We cannot, however, entirely reverse these rules. Ill-marked and limited physical signs are no guarantee of a favourable course of the disease. One of the worst types of phthisis is the case where the constitutional symptoms—fever, wasting, and debility—are out of proportion to the physical signs. Further, marked and advanced signs, if of limited distribution, are not necessarily very unfavourable. Flattening, dulness, and bronchial breathing at one apex may be consistent with a limited lesion and a more or less satisfactory arrest of the disease. Signs of fibroid change are naturally much more hopeful than signs of increasing infiltration and softening. Hence, shrinkage of one apical region, with weak or faintly bronchial breathing, some dulness on percussion, and a comparative paucity of râles, constitute a

relatively favourable combination of physical signs, while the gradual extension of moist crepitant râles indicates the spread of the disease. The extent of the physical signs requires to be interpreted in the light both of their nature and of their probable duration. The presence of excavation is always unfavourable, but Lenhartz and Cornet are of opinion that even cases with cavities may recover completely.* Most authorities are of opinion that cavities once formed never close or become completely obliterated, though they may dry up and remain quiescent for long periods. A case where there is a single chronic, dry cavity at one apex, and little evidence of other involvement of the lungs, constitutes a relatively favourable type. One of the best cases of arrest of the disease, with complete restoration of the general health and practical abeyance of all symptoms for several years, which have come under my observation was that of a young lady who had a large cavity in the right infraclavicular region. Nevertheless, cavity cases, with rare exceptions, ultimately do badly.

Where physical signs and symptoms conflict as regards their prognostic indications, it is safer to trust to symptoms than to signs. The nature of the symptoms gives us the most trustworthy of all prognostic indications. Amongst the most unfavourable symptoms are the following: Severe and persistent pyrexia, especially if characterized either by high continuous fever or by a high afternoon maximum and a low morning minimum; rapid and progressive wasting, especially if it continue in spite of active alimentation; failure of appetite, discomfort during digestion, diarrhœa; rapid weak pulse; profuse night-sweating; profuse purulent sputum, containing many elastic fibres. An accentuated pulmonary second sound is believed to indicate that the right ventricle is vigorously combating the obstruction in the pulmonary circuit, while a decrease of an existing accentua-

* Lenhartz, *op. cit.*, Bd. i. 1, p. 319; Cornet, *op. cit.*, p. 493.

tion may point either to an easing of the pulmonary circulation or to exhaustion of the right ventricle.*

The following axioms may be laid down:

The absence of pyrexia, though nearly always a highly favourable symptom, is not inconsistent with progress of the disease. A gain in weight is usually favourable, but may occur even in cases which are otherwise doing badly. The hyper-alimentation of modern treatment not uncommonly produces the melancholy spectacle of a patient who has grown fat, but not well. Nevertheless, the indications of the weighing-machine are among the most valuable of prognostic aids. Marked dyspepsia may be due to dietetic errors. If it persists in spite of a regulated dietary and careful treatment, the outlook is gloomy. Persistent frequency and feebleness of the pulse, in spite of rest, is very unfavourable. The number of tubercle bacilli in the sputum is no certain index of the activity or progress of the disease, but numerous bacilli are usually present when the process is active. Hæmoptysis, if profuse and repeated, is unfavourable. Slight and infrequent hæmorrhages have no prognostic significance. Dyspnœa, if marked, may point to miliary tuberculosis, weak heart, or profound debility, and is very ominous. Dropsy of the extremities, dysphagia, and thrush usually mark the imminence of the end. *Spes phthisica* is sometimes marked in the worst cases.

The complications are highly important with a view to prognosis. Amongst the gravest complications are the following: Meningitis, peritonitis, pneumothorax, ulceration of the bowels, amyloid degeneration of the kidneys, ulceration of the larynx, dysphagia, thrush. Pleurisy, fistula in ano, various skin eruptions, are not unfavourable. Phthisical patients often go through an attack of pneumonia fairly well, and their condition subsequently is not necessarily worsened by the attack. Persistent diarrhœa is one of the commonest

* Cornet, *op. cit.*, p. 495.

complications of phthisis. If it depends upon ulceration or amyloid degeneration of the bowel, the outlook is very bad. If it depends on mere dyspeptic causes, it should be controllable by dietetic and medicinal measures.

Percy Kidd thus sums up the prognostic problem: 'The best results may be expected in a case presenting the following features: Apyrexia or a subfebrile temperature; weight stationary or increasing; signs of disease confined to one lung or to limited portions of both lungs (especially if associated with contraction); a quiet pulse and nervous system; a good digestion; absence of serious complications; a good family and personal history; and favourable hygienic surroundings.'*

I propose to take a few types of phthisis, and to offer some observations on the prognosis which their characteristics seem to warrant:

Case I.—A young adolescent (eighteen to twenty-five years of age) develops phthisis of the 'insidious onset' type. There is cough, some loss of flesh, slight afternoon pyrexia, and some signs of commencing mischief at one apex.

Prognosis.—Doubtful, but not definitely bad. The main aids to prognosis in such a case are the following:

1. Whether the attack was predisposed to by hard work, a confined life, inattention to food or similar causes; or came on in the absence of such predisposing causes. The prognosis is better in the former case.

2. The state of the appetite and digestion. Vigorous digestive power is highly favourable, and *vice versâ*.

3. The state of the circulation. A pulse of good tension and moderate frequency is favourable, and *vice versâ*.

4. The family history; the patient's previous habits, his prudence, self-control, and resolution; and the prospects of being able to secure the best treatment. These points have already been sufficiently dwelt upon.

* Percy Kidd, *loc. cit.*

CASE II.—A young adolescent (eighteen to twenty-five), previously in apparently good health or with very slight symptoms, is suddenly attacked by sharp pulmonary hæmorrhage depending on tuberculosis. The bleeding recurs once or twice, and then ceases; the general constitutional state is good; the physical signs in the lung are obscure.

Prognosis.—Upon the whole, relatively favourable. Death from the effects of the hæmorrhage is rare. If pyrexia persists after the hæmoptysis, it is probable that septic pneumonia is present, and the case passes into a less favourable category. Pulse and digestive capacity are very important.

These cases sometimes do well under various forms of sanatorium or climatic treatment. They are often suitable for the high altitudes.

CASE III.—Phthisis of a chronic type is known to have existed for six or twelve months, or longer, in a patient of from twenty to thirty years of age. There are signs of a well-marked but quiescent lesion at one apex. The general constitutional state is fair. There are no complications.

Prognosis.—Unfavourable as regards cure, fairly favourable as regards duration of life. The main aids to prognosis are the predominance of fibroid or caseating changes in the lung; the state of the digestion, pulse, weight, and temperature; the occupation and mode of life; the prospects of securing adequate and persevering treatment. These cases sometimes go on indefinitely, enjoy fair health, and perhaps do useful work, but they rarely recover. In the long run they usually succumb to some form of pulmonary disease, not always to the direct effects of tuberculosis, but perhaps to an intercurrent pneumonia, pleurisy, or bronchitis.

CASE IV.—Tubercular disease of the pneumonic type has progressed rapidly to partial excavation of one upper lobe, and has then undergone arrest. The general condition is fair, but the patient is thin, rather short of breath, and incapable of severe exertion.

Prognosis.—Doubtful. A few of these cases get on well, but in most instances the extension of the disease to the rest of the lungs is only a question of time. It will be remembered that Jaccoud held this type of case to be specially favourable.

Case V.—Excavation exists at one apex, infiltration of a portion of the same lung, slight signs at the opposite apex.

Prognosis.—Unfavourable, but duration may be very prolonged. Temperature, weight, strength, digestive capacity, prospects of thorough and persevering treatment, must be accorded their due weight. These cases are practically incurable. A duration of over twenty years has been known to me.

Case VI.—Supervening upon an old chronic lesion, tubercle has become rapidly disseminated throughout a large area of the lungs. The temperature is high, and there is much wasting and prostration.

Prognosis.—Highly unfavourable; death probably in a few weeks. In a certain small proportion of these cases the acute process becomes arrested, and the case lapses into the chronic category.

Case VII.—Phthisis has supervened upon typhoid fever, measles, or whooping-cough, and has assumed the subacute caseating form.

Prognosis.—Unfavourable. These cases usually do badly. In exceptional instances the active symptoms of tuberculization abate, and the cases take on one of the ordinary types of chronic fibro-caseous phthisis, to which the broad rules of prognosis, already sufficiently commented upon, may be applied.

I put forward the foregoing types of phthisis and the prognostic indications which they suggest, fully realizing their very limited value. Nevertheless, they may afford the practitioner a certain small amount of guidance. Cases of phthisis are so numerous, and the prognosis varies within such wide limits, that we cannot rest satisfied with a vague

general prognosis, whether of the pessimistic type of past years or of the optimistic type of to-day. We must approximate to rules which will enable us to say with some approach to accuracy in any given case :

(*a*) Whether there is reasonable hope of complete recovery.

(*b*) Whether the case is, upon the whole, favourable, a good rally probable, and treatment likely to repay its cost in time and money.

(*c*) Whether the case is, upon the whole, unfavourable and likely to admit of only a moderate degree of improvement.

(*d*) Whether the case is definitely unfavourable, and likely to admit of only a slight degree of palliation.

(*e*) Whether the case is obviously hopeless and systematic treatment useless.

The economic aspects of these questions often come before us. Patients, or their friends, say with much reasonableness, 'If I make a great sacrifice in time and money, and spend a prolonged period at a sanatorium or at a health resort, can you give me any assurance that the result will be commensurate with the expense and trouble ?' No doubt a positive answer to this very natural question cannot always be given, and the patient's means, social position, and domestic circumstances may all require to be carefully weighed. But an answer of some sort to this reasonable question—the best that we can arrive at—should be given.

It hardly needs insisting, in concluding our survey of this subject, that in a disease where the unexpected so often happens, where the casual and the unforeseen play so large a part, where the patient's wisdom, courage, and self-control (or the absence of these qualities) count for so much, our prognosis should always be guarded, and a dogmatic attitude should be avoided. It will often, however, be the duty of the practitioner to tell the patient's friends candidly that the case is hopeless, the end only a question of time, and that sanatorium or other treatment will be futile.

LECTURE XVIII

THE PREVENTION OF PULMONARY TUBERCULOSIS

SUMMARY:

Great progress already achieved.

Preventive measures:

A. The disposal of sputum; the disinfection of rooms occupied by phthisical patients.

B. Attention to the food-supply—milk, meat, bread.

C. Measures of general hygiene, viz.:

1. Better housing of the lower classes.
2. Attention to cleanliness, ventilation, and the disposal of domestic refuse.
3. Draining of damp subsoils, removal of filth, efficient sewerage.
4. Regulation of mills, factories, ware-rooms, and workshops.
5. Care of the young, especially as regards attention to the hygiene of schools, the provision of crèches, and playgrounds.
6. The inculcation of temperance.
7. The provision of adequate air-spaces in large towns.

D. The segregation of tubercular subjects—question of hospitals, sanatoria, tubercular colonies.

E. Administrative reforms—Notification.

The prevention of pulmonary tuberculosis is one of the most important practical problems before the modern world, and the zeal and earnestness with which it is being faced

are of the happiest augury for the future. The task before us is one of the utmost magnitude and importance, but the ultimate victory of science and civilization over tuberculosis is certain. It will be the speedier or the more delayed according as the teaching of medical science is more or less fully accepted and acted upon. The success of any nation or community in stamping out tuberculosis is no bad index of its standard of civilization. The measures necessary for the prophylaxis of the disease are fully understood, although some difference of opinion still exists as to the relative frequency of the various channels of infection; and the great success which has in various countries attended their adoption is positive proof that we are on the right road, and that what we need is not so much further knowledge as diligence and conscientiousness in acting upon the knowledge which we already possess. When many great cities have reduced their death-rate from tuberculosis by 30 or 40 per cent. within the space of a single generation by vigorous hygienic measures, it is evident that it is apathy, rather than ignorance of the necessary prophylactic procedure, which we have to combat. Pending the final settlement of the great controversy whether the tuberculosis of the bovine species is or is not identical with human tuberculosis, it seems safer at present to regard the views of Koch and Behring as supplemental of, rather than antagonistic to, each other, and to promote measures which tend to prevent the dissemination of the virus of tuberculosis either by the agency of dried sputum or of tuberculous milk and meat. I entertain little doubt that the former is by far the more important matter, but in the present state of our knowledge it does not seem safe to neglect the latter.

We shall consider the preventive treatment of pulmonary tuberculosis under the following heads:

A. The disposal of sputum; the disinfection of rooms occupied by phthisical patients.

B. Attention to the food-supply—milk, meat, bread.

C. Measures of general hygiene, such as the following:

1. Better housing of the lower classes.

2. Attention to cleanliness, ventilation, and the disposal of domestic refuse.

3. Draining of damp subsoil, removal of filth, sewerage.

4. Regulation of mills, factories, ware-rooms, and workshops.

5. Care of the young, especially as regards attention to the hygiene of schools, the provision of crèches and playgrounds.

6. The inculcation of temperance.

7. The provision of adequate air-spaces in large towns.

D. The segregation of tubercular subjects.

E. Administrative reforms—notification.

Our limits will only permit a somewhat summary consideration of some of these large questions.

A. The Disposal of Tubercular Sputum.

The patient must be instructed always to spit into a vessel containing water, and on no account to spit on the floor or into a handkerchief. Spittoons, properly constructed and regularly cleansed, should be provided in public places, workrooms, factories, shops, corridors, closets, as it is quite impracticable to prevent spitting in such places. The proposal to forbid spitting in the streets, however laudable, is really a counsel of perfection which practical hygiene can hardly take account of. For ambulatory cases of phthisis Dettweiler's spitting-cups should be provided. The linen and bedding of tubercular patients should be frequently changed, and their rooms should be often mopped with damp dusters, the windows being thrown widely open, and fresh air and sunlight being freely admitted. Dry dusting is useless or dangerous. There should be as little furniture as possible in such rooms, and no thick carpets or heavy curtains. The tubercular patient should always have a sleeping-room to himself. After the death of a tubercular patient his room

should be thoroughly disinfected, including the walls, furniture, and bed equipment, and it should be allowed to remain unoccupied for several weeks, free ventilation and insolation being sedulously practised. Probably no single measure of prevention which could be devised is so important as this proceeding, which should be made compulsory by law. In public institutions — schools, reformatories, wash-houses, lunatic asylums, and prisons—it should be a strict injunction never to place a healthy individual in an apartment previously in occupation by a tubercular case without the most thorough disinfection. Melancholy cases have come under my observation where the neglect of this simple rule has led to disastrous consequences.

B. Attention to the Food-Supply—Milk, Meat, Bread, Water.

While we know that an acute cleavage of opinion exists as to whether the tubercular virus reaches the human subject in any large proportion of cases through the channel of food, it is clear that strict attention to the character of the food-supply of the people is incumbent upon us, not only because it is a possible source of infection, but also on general grounds—viz., as being intimately connected with the general question of the maintenance of nutrition.

Behring's theoretical views have not met with any general acceptance, and they seem to me to rest on no solid foundation of clinical experience, but his suggestions as regards preventive measures are worthy of every attention, even although the theory which underlies them may be a mistaken or exaggerated one. His essential contention is that the chief means of combating tuberculosis is the prevention of the introduction of tubercle with food, especially with milk, during infantile life; and the introduction of tubercular antibodies with the milk in earliest infancy, in order to render innocuous any inhaled tubercle bacilli. The following are

the chief points in the Bonn rules given by Behring with regard to these questions:

1. The milk of cows, which react positively to tuberculin, must not be used for feeding calves, nor, of course, for infant feeding.

2. Protectively inoculated animals should, as far as possible, be housed in a stable free from tuberculosis.

3. Particular attention should be given to the cleanliness of the feeding-troughs and the quality of the water supplied to cattle.

4. The disinfecting of infected stables is to be thoroughly carried out by the use of a 5 per cent. carbolic acid solution or a 2 per cent. hot soda solution.

5. Attention to the cleanliness of the attendants on the cattle, the use of towels and dish-cloths which have been rendered germ-free by means of hot water or dry heat.

6. The cleansing of milk bottles should be carefully attended to, the necessary measures including energetic rinsing with a 10 per cent. warm soda solution, rinsing with quartz gravel by means of a stream of hot water, cold rinsing, sterilization in the dry chamber.

7. The stables should be so constructed that they can be kept thoroughly clean. They should be well supplied with hot and cold water, well ventilated, and the arrangement of the stalls should be such that the excreta are received into an open drain connected with the main drain. The flooring should be water-tight.

8. The cows should be kept scrupulously clean, special care being given to the udders and tails.

9. The milk vessels should be of tinned sheet iron.

10. The milk should be strained through aluminium-nickel gauze or brass gauze, which is then to be cleaned and disinfected.

11. The milk from one milking should be pumped into the bottling room, and conducted into a collecting reservoir,

from which it is bottled by a bottling-machine, the milk having a temperature of 5° to 7° C. on bottling.

12. If the milk is required for exportation, and to be kept for three days before use, the addition of formaldehyde in the proportion of 1 to 40,000 is recommended.*

For the purpose of obviating the dangers of tuberculous meat, the following regulations are in force in some continental countries. Denmark and Saxony have been specially active in this matter:

(*a*) All meat should be rejected as dangerous to health, and useful only for manufacturing purposes, if the meat itself appear diseased, or if the bones contain pearly nodules, or if the disease is general and severe—*i.e.*, if it involves more than one of the body cavities, and is associated with much emaciation.

(*b*) If the disease involves two of the body cavities, but without necessitating the assumption of a dissemination by means of the greater circulation; if, moreover, there is no emaciation, and the meat appears to be sound, it may be designated as of inferior quality, but not dangerous to health. Such meat is to be sold only after being cooked, the cooking to be done in a steam chamber.

(*c*) If the tuberculosis is confined to only one of the body cavities, or to a single organ, so that dissemination by the greater circulation may be excluded, the meat is to be considered neither detrimental to health nor of inferior quality. Such meat may be freely offered for sale, after removal of the diseased portions.†

C. Measures of General Hygiene.

Of the value of measures of general hygiene in the prevention of tuberculosis there can be no doubt. The difficulty is

* Von Behring, 'The Suppression of Tuberculosis,' English translation, pp. 60-67.

† Cornet, 'Nothnagel's Encyclopædia of Practical Medicine,' 'Tuberculosis,' p. 513 *et seq.*

that such measures seem, for large classes of the community, to involve insuperable economic problems. Nevertheless, it cannot be too much insisted that, in general, the amount of tuberculosis in any community has a very definite relation to the standard of living found amongst the inhabitants, and that no other measures of prevention can attain any considerable degree of success if poverty, filth, overcrowding, and bad feeding generally prevail. The effort to combat tuberculosis is, in one of its aspects, an effort to raise the entire social level. Some authorities hold that it is wrong to emphasize these views, as the inference from them would seem to be that tuberculosis depends largely upon causes deep-rooted in the social mechanism and only slightly under our control, and that our efforts should be mainly concentrated upon measures to segregate tuberculous persons and prevent infection. I believe we shall attain the largest measure of success by frankly admitting that both factors possess enormous importance, that both are beset with the gravest social and economic difficulties, but that both must be faced by all civilized communities.

The facts which prove that the tuberculosis rate is, in general, in some sort of definite ratio to the amount of overcrowding, poverty, and privation in any community are innumerable, and admit of only one conclusion. I select the following evidence out of the vast mass of material which is available on this subject :

M. Gebhard has shown that in Hamburg the death-rate from tuberculosis among persons whose income exceeds £175 per annum is equal to 10·7 per 10,000 per annum, while the rate amongst persons whose income varies from £44 to £60 is 39·3 per 10,000. Similar facts have been elicited regarding Frankfurt, Munich, and Berlin by Dr. Raths, and regarding Vienna by Dr. Philipovitch. Dr. Romme has shown that in Paris the tuberculosis rate of mortality is 10·8 per 10,000 in the neighbourhood of the

Champs Elysées as compared with a rate of 104 per 10,000 in the very poor quarters of Grenelle and Plaisance. Professor Henschen of Stockholm has shown that in the Swedish capital the tuberculosis rate of mortality in districts where there are 100 bedrooms for every 100 inhabitants is 14 per 10,000, while in districts where there are only 100 bedrooms to every 340 inhabitants the rate is 38 per 10,000.

These facts, which might easily be multiplied a hundredfold, do not stand alone. They express a universal experience; they serve as a useful reminder of how large a part the economic factor plays in connection with tuberculosis. The general hygienic conditions which tend to prevent the spread of tuberculosis are sufficiently well understood. They include such points as the following: (*a*) Suitable housing of all classes, so that overcrowding shall be prevented, subsoil damp mitigated, light, air, and ventilation promoted, the removal of filth and domestic refuse rendered easy, and house sanitation rendered efficient; (*b*) the due regulation of trades and workshops, especially such as involve confinement in dusty air; (*c*) the provision of air-spaces in the large cities for the people, and the promotion of healthy exercise and recreation; (*d*) the control of the food-supply of the people, and the prevention of want and of intemperance. These are very large questions, which touch the field of public hygiene at almost every point. I must content myself with emphasizing a few of the most outstanding particulars.

The housing of the poor is a matter of the utmost moment in the fight against tuberculosis. Small, ill-ventilated, uncleanly rooms and overcrowding favour the propagation of tuberculosis from two points of view: (*a*) They increase the facilities for infection; and (*b*) they lower the resisting power of the individual. There can be no question that, without any economic revolution, much can be done to effect a betterment of the housing of the lower classes by attention to matters which admit of easy regulation. The construction of

houses so as to allow as much light and air as possible and to permit of ready ventilation is still very imperfectly attended to. The faulty construction of the houses of the Irish peasantry is undoubtedly one of the reasons why tuberculosis is so rife amongst them. 'Architects and builders,' says Dr. Ransome most truly, 'of all classes of dwellings have been in the past, and are still, largely responsible for a very large proportion of preventable mortality. It is only right that we should look to them in the future to rectify the mischievous modes of construction—both of mansions and cottages and public buildings of all kinds—that have proved such a fertile source of disease, and especially of consumption. It is incumbent upon them both to rectify existing buildings, and to erect new ones upon plans that are more in accordance with modern sanitary requirements. They must see to the exclusion of noxious ground-air from houses, to the prevention of the harbourage of dust, to giving sufficiently copious streams of air without draughts and to the extraction of foul air as soon as it is produced, and to the provision of abundance of light. . . . Back-to-back houses should be converted into double houses with thorough ventilation. Closed streets, courts, and alleys should be opened up, and decent habitable houses built in their place. Breathing-spaces and playgrounds should be left in their midst.'* It is satisfactory to know that public opinion has been aroused on this subject, and that the various Sanitary Acts give local bodies all needful powers.

The regulation of unhealthy trades is a vital element in the prophylaxis of tuberculosis, and some considerable progress has already been attained. The use of ventilating-fans in factories has now become general, and much greater care regarding cleanliness than was formerly usual has begun to be taken. The principles which should govern the hygiene of factories and workshops have already been

* A. Ransome, 'The Causes and Prevention of Phthisis,' pp. 117, 119.

sufficiently laid down. In addition to the mitigation of dust, dirt, and re-breathed air, it is important that the artisan population should not expose themselves habitually to sharp alternations of temperature on leaving their workrooms, that they should be instructed as regards suitable clothing, and that measures should be taken to give them facilities for obtaining wholesome meals, at a small cost and at suitable intervals of time. These points are obvious, but a good deal of patience and persistence will be necessary in working out the details. The hygiene of schools has an important bearing upon the prophylaxis of tuberculosis. There can be no doubt that the disease is often propagated in small, overcrowded, uncleanly, and ill-ventilated schools. The subject has in recent years excited much attention. In some countries—*e.g.*, Denmark, Saxony, the United States, and Japan—great progress has been made, and in the United Kingdom the policy of the London County Council has been eminently progressive and enlightened. Ireland, unhappily, still lags far behind in this important matter. The conditions necessary for the prevention of the spread of tuberculosis in schools are easily defined, and follow naturally from the general principles which have already been fully considered. A school should, if possible, be situated in a healthy locality where sufficient air-space and suitable playgrounds can be arranged for, and where the air is not rendered foul by a dense population, bad sanitation, or the proximity of unhealthy trades. The buildings should be fully adapted for all the purposes of education and health. A minimum of 10 square feet of floor-space for each scholar should be provided; the window-space should not be less than one-fifth of the floor-space; the lighting should be so arranged as to afford sufficient light without glare, and the heating and ventilation should be planned on some modern and efficient system. Slow-combustion stoves should be forbidden. Proper sanitary facilities, ample play-grounds, and

suitable cloak-rooms should be provided, and scrupulous care should be taken to secure thorough cleanliness in all the school premises. The regular medical inspection of the scholars should be maintained, with a view to the early detection of disease and the segregation of the sick from the healthy.

The instruction of the people in the principles of hygiene, the laws of food, the necessity for temperance, is an indispensable element in the battle against tuberculosis.

D. The Segregation of Tubercular Subjects.

We now approach a much more difficult and controversial part of our subject—viz., the question how far the segregation of tubercular persons from the rest of the population is feasible, desirable, or economically justifiable. Some of our greatest authorities are divided upon this question, and it would appear that we have not yet in sight a system which is likely to command universal approval. The question is further complicated by the additional controversy whether, assuming that segregation of the tubercular is necessary or desirable, sanatorium treatment for them is the best that can be devised, and whether it will repay its cost in time, trouble, and expense. In order that we may consider the question in a concrete form and fully realize its scientific and economic bearings and all the difficulties which it involves, let us take a hypothetical case--viz., that of a city with a quarter of a million of people, a tuberculous rate about the general average, and no special circumstances which either hinder or assist the management of the disease. We may assume a tuberculosis death-rate of about 2 per 1,000, which is a little over the average of some of the more hygienic English cities, but less than the average of many other cities, both at home and abroad.

A city with 250,000 inhabitants will thus lose on an average 500 persons annually from tuberculosis. If we reckon

that for one person who dies of the disease there are five affected by it, and this does not seem an extravagant estimate, we get the result that a city of the size mentioned will have at any one time about 2,500 tubercular patients within its bounds. I will assume that one-fifth of this total will belong to the well-to-do classes, who can look after themselves and select either sanatorium treatment or climatic change at their own option. The problem is, What should the hypothetical city do with regard to the remaining 2,000? It may be assumed that they cannot be treated in the ordinary hospitals —a method of little advantage to the tubercular patients, a source of danger to other patients and to the nurses and attendants, and from every point of view objectionable. If 2,000 patients are sent to sanatoria, the cost per head on the present sanatorium lines would not be less than £1 per week, or a total yearly outlay of £104,000. Now, it is certain that private charity will not yield this sum, and that municipal rates would be overburdened by such an outlay. There is the further economic consideration that a certain proportion of the hypothetical 2,000 patients would be, under certain limitations, capable of earning their own living. It is clear, then, that the question involves many difficulties, and is not to be solved by the off-hand recommendation of sanatoria for all cases. A little reflection will probably serve to convince us that the large total of tubercular patients is divisible into three classes, of which the needs are different:—

There is Class A, consisting of incipient cases with fair general physique, and a limited lesion.

There is Class B, consisting of more advanced cases with a considerable and progressive lesion and decided impairment of general health.

There is Class C, consisting of advanced and hopeless cases, for whom treatment can only be palliative. We shall not make much progress until we agree to distinguish these classes, and consider them separately.

For Class A sanatorium treatment is indicated, and by its means a certain not inconsiderable proportion of cases will attain actual or relative cure; while a much larger proportion will obtain improved health, sufficient to enable them to return to their work for several years. The reports of the German sanatoria, where the cases are not always rigorously selected, show that about two-thirds of the cases rally sufficiently to return to their avocations. No doubt most of these subsequently relapse, but the temporary gain is great and is sufficient to make sanatorium treatment for such cases economically justifiable. The sanatoria should be of simple construction, on the châlet principle, and the cost per bed need not exceed £100.

For Class B the suitability of sanatorium treatment is doubtful. Unquestionably, many of these patients would benefit by it to a certain extent, but cure is, *ex hypothesi*, not to be expected; sanatorium treatment would be prolonged and costly; relapse subsequently would be speedy; the system would not be economically justifiable. What seems to be required for such cases is some sort of home or colony, where the patient, with others of his class, could obtain the requisite treatment and supervision, pursue in some cases, either regularly or intermittently, some form of useful occupation, and thus lighten the burden which he throws upon the community.

For Class C sanatorium treatment, with its costliness and its elaborate machinery, is manifestly unsuitable. What these patients need is a home for incurables, managed on simple and inexpensive lines, but adequate for the relief of their sufferings, and for securing them an easy euthanasia. The importance of the segregation of Class C is, from the point of view of the social community, of great moment, as these patients are no doubt the great disseminators of infection.

It is to be hoped, then, that the ideal social community of the future will possess three types of institution for its battle against tuberculosis—viz:

1. Sanatoria for incipient cases, managed on simple lines but with all the scientific requisites for combating the disease, where patients would remain for a limited period, perhaps on an average from three to six months, or longer, if possible.

2. Tubercular colonies, where the patients could remain permanently and work, and which might be made to a certain extent self-supporting.

3. Homes for incurables, for the care of advanced cases until death.

Let us consider some of the economic problems involved in the above scheme. It has been reckoned that tuberculosis costs the kingdom of Prussia about 86,000,000 marks (£4,300,000) yearly.* This is probably an under-estimate rather than an over-estimate. Dr. Charles Reinhardt computes the annual cost of consumption in London alone at £4,000,000. The cost to Ireland from the disease cannot be under a million pounds sterling per annum—an enormous sum for a comparatively poor country. A reduction of the tubercular rate by 30 to 40 per cent., such as has been effected in England and Prussia within the last generation, would mean a saving to Ireland of at least £300,000 per annum—probably considerably more.† When we add the saving in suffering and sorrow which would be thus effected, we may safely conclude that if the battle against tuberculosis is somewhat expensive it will, if successful, prove fully worth its price. How far tubercular colonies could be made self-supporting is a question which has yet to be solved, but the subject opens up a large and by no means hopeless problem. A large proportion of cases of chronic phthisis are quite equal to light work, and sometimes to the finest and most lucrative work; but laborious and fatiguing work would, of course, be unsuitable. The cost of providing

* Cornet, *op. cit.*, p. 19.

† These estimates for Ireland are probably much under the mark, but I wish to understate rather than overstate the case.

sanatorium accommodation for incipient cases will be serious, but not beyond the possibilities of charitable effort. Probably a sanatorium with 250 beds would fulfil the necessary indications for the hypothetical specimen city of a quarter of a million inhabitants. The cost of the maintenance of such an institution would probably be about £15,000 per annum —a large sum, but one which would probably repay the outlay. The cost of maintaining a few homes for incurables for advanced cases would not be very heavy.

Another method of dealing with Class B—*i.e.*, tubercular patients who are in too advanced a stage to leave hope for cure by sanatorium treatment, and not sufficiently advanced to be consigned to a home for incurables—has been designed by Calmette, and adopted with great success in Germany, France, and elsewhere. The plan is thus described by Koch in his recent Nobel Lecture (1905): 'The merit . . . belongs to Calmette, to whom occurred the happy idea of providing for this class of patients by the *dispensaries* organized by him. Calmette's suggestion has met with approval everywhere, especially in Germany, where more than fifty such dispensaries already exist, and where many cities are about to provide themselves with such. . . . The patient is visited in his dwelling, and instruction and advice as to cleanliness and the treatment of the sputum are given to him and to his family. If the domiciliary conditions are bad, money is granted in order to render the separation of the patient from the healthy members of his family possible, and thus to convert a dangerous patient into a comparatively harmless one by hiring a suitable room or even another dwelling. . . . The dispensary itself does not undertake the treatment of the patients, in order not to get into conflict with practitioners; but it takes care that they are placed under medical treatment, and, if advisable, admitted to a hospital, a sanatorium, or a health-recruiting home. One specially important part of their work is that they supervise the

family, and especially the children, and have them examined from time to time to see whether infection has taken place, in order to be able to bring help as early as possible. In this way these dispensaries really take care of poor consumptives, and they have therefore with perfect justice been named "care-stations." I regard them as one of the most powerful means of combating tuberculosis, if not the most powerful of all, and I believe that when, as we may hope, a dense net of care-stations overspreads the land they are destined to do a most blessed work.'*

There can hardly be any doubt that Calmette's scheme is happily devised and forms a most necessary link in the chain of protective mechanisms in the fight against tuberculosis. These dispensaries are cheap and efficient; they help the poor to help themselves; they serve to spread the light of hygienic principles through the crowded communities which have been wise enough to adopt them.

As to the general utility of the segregation of tubercular patients, and placing them in suitable institutions, Koch speaks in the most emphatic terms. Speaking of the great decrease of tuberculosis in England and Germany during the last two or three decades, he says: 'I am firmly convinced that the better provision for patients in the last stage of pulmonary phthisis —namely, the lodging of them in hospitals, which is done in England and in Prussia to a comparatively large extent—has contributed most to the improvement.'†

Dr. Arthur Newsholme, in a communication to the last International Congress on Tuberculosis (Paris, 1905), showed that the great decline in the tuberculosis rate in England and Wales during the last forty years had coincided with the treatment of a much larger proportion of cases in institutions. Thus, in the years 1861-1865 the percentge of cases of tuberculosis dying in public institutions was 14, while in the years 1901-1903 the proportion had risen to 31. This

* R. Koch, 'The Nobel Lecture,' *Lancet*, 1906. † *Ibid.*

does not mean that the special hospitals for consumption had played a large part in the diminution of the tuberculosis rate, as they were too few to exercise any preponderating influence. The number of beds in such institutions did not much exceed 1,000, while the number of deaths from pulmonary tuberculosis in the United Kingdom in 1902 was 56,643. It was rather the workhouse infirmaries which had been the means of separating tuberculous patients from the rest of the population, and thus preventing the spread of the disease. As the number of persons who die at home decreases, so does the spread of tuberculosis also decrease.

In Berlin 40 per cent. of all fatal cases of pulmonary tuberculosis die in hospitals, a fact which gives us some index of the extent to which hospital treatment is available for such cases in that city.

The treatment of all cases of pulmonary tuberculosis in hospitals, sanatoria, and homes for incurables would be a gigantic task, for which public opinion is not yet ripe. But the beginning which has been made in this direction in all civilized countries should be encouraged and persevered with. How small that beginning has been in the United Kingdom relative to the mass of disease to be dealt with is evident from the fact that, while the sufferers from pulmonary tuberculosis are reckoned to number at least 200,000, the number of beds in special institutions for the treatment of the disease hardly exceeds 1,000. Many sufferers, of course, obtain treatment in the ordinary hospitals and in the workhouse hospitals. While this disproportion between the number of available beds and of tubercular patients continues, it is evident that a selection of cases for treatment is necessary. Two principles should govern this selection—viz., to provide treatment for cases which offer a fair prospect of cure, and to offer an asylum in institutions for advanced cases which are most likely to spread infection.

E. Administrative Reforms; Notification.

Notification of some sort must form a necessary factor in the battle against tuberculosis. Universal compulsory notification has been adopted by Norway and Denmark, and the results of that courageous experiment will be watched with interest and sympathy by other nations. The objections to such a course are serious and not to be lightly put aside. It is urged that compulsory notification would make the tubercular patient a marked man, and would tend to deprive many bread-winners of their occupation and livelihood, as employers of labour would encounter much prejudice in retaining such persons in their employment; that it would make tubercular persons reluctant to seek medical advice, lest their condition should become known and certain disabilities ensue; that it would curtail the liberty of many persons who are not likely to be sources of infection; and that it would throw upon municipal authorities a vast volume of work with which they are at present without the means of successfully dealing. How far these objections ought to weigh in the scale against the probable advantages to the community of a well-regulated system of notification, it is difficult to say. The tendency of modern opinion is to regard the general good rather than individual convenience, and it is impossible to deny that this tendency is, upon the whole, beneficent, although it may lead to hardship in individual cases. As preliminary measures, clearly inadequate but fulfilling the most urgent necessities of the case, and perhaps going as far as public opinion in this country is prepared for, I would suggest a limited form of notification on some such lines as the following: That medical practitioners should be required to notify all cases of pulmonary tuberculosis where the disease exists under domestic conditions rendering the segregation of patients and the prevention of infection difficult or impossible. The notification of

incipient cases might, with the spread of more enlightened views regarding the disease, become unnecessary, as such cases would probably resort without compulsion to sanatoria, if such institutions were generally available at a moderate cost. If, however, compulsory notification is found to work well in other countries and not to involve any intolerable hardships, the demand for it in this country will become irresistible.

LECTURE XIX

THE TREATMENT OF PULMONARY TUBERCULOSIS

SUMMARY.

A. Specific treatment :

History of tuberculin.

Koch's rules for the administration of tuberculin.

Views of Lenhartz, Pottenger, West, Klebs, Behring, Maragliano, Marmorek, Maksutow, Hirschfelder, Landmann, Béraneck, and others.

B. Antiseptic treatment :

Aims and methods of antiseptic treatment.

Objections to intra-tracheal and intra-venous injections.

Antiseptic inhalations.

Internal use of creosote, creosotal, guaiacol.

C. Hygienic treatment :

Historical note : Views of Bodington and MacCormac, Brehmer and Dettweiler.

General principles of hygienic treatment.

Results of the treatment : Returns of the German Imperial Health Office. Opinions of Hermann Weber, Turban, Burton-Fanning, Morin, Schmidt, Bang, Kayserling, West.

Educational value of sanatorium treatment.

Criteria of patient's improvement.

Cornet's cautions against expecting too much from sanatorium treatment.

Need for combined effort on the part of assurance and benefit societies and of municipal authorities.

The large subject of the treatment of pulmonary tuberculosis may be conveniently dealt with under the following heads:

A. Specific treatment.
B. Antiseptic treatment.
C. Hygienic treatment.
D. Symptomatic treatment.
E. Climatic treatment.

A. Specific Treatment.

The search for a specific remedy for tuberculosis is not chimerical or irrational. There are sufficient analogies in the field of medicine to make the possibility of the discovery of such a remedy one that must be freely admitted. Universal interest was therefore excited when in 1890 Koch announced that he had discovered a substance which was capable of rendering healthy guinea-pigs immune to tuberculosis, and of bringing about complete arrest of tubercular processes already far advanced in these creatures without injury to the animals themselves. This substance was a sterilized glycerine extract made from pure cultures of the human tubercle bacilli. The assumption that similar effects would follow in the human subject from the use of this remedy, to which the name 'tuberculin' was applied, has not been justified by experience. At a later stage Koch introduced a new form of tuberculin, called Tuberculin R, prepared by pounding dried cultures of the tubercle bacilli, mixing them with distilled water, centrifugalizing, and removing the fluid at the top, The remainder is dried and pounded, distilled water is again added, and the whole again centrifugalized. The process is several times repeated, and the combined super-incumbent solutions, except the first, are mixed with 20 per cent. glycerine. The dose is $\frac{1}{500}$ milligramme, to be subsequently increased. There seems no satisfactory evidence that this new tuberculin—Tuberculin R

—possesses any advantages over the former preparation, Tuberculin-Alt. When the latter first came into general use, it was employed in all classes of case, without regard to special symptoms, and with only vague notions on the question of dosage. That the results were sometimes distinctly injurious, and that the remedy, as Virchow contended at a very early stage, led to destruction of the tissues around the tuberculous areas and thus set free the bacilli in the blood, is incontestable. I saw sufficient evidence in my own experience of these injurious effects. Naturally, the remedy rapidly fell into disrepute. But the question still remains open whether tuberculin may not prove useful in certain types of case and in carefully limited doses. In favour of the use of the remedy we have the opinions of Koch, Spengler, Turban, Liebermeister, Lenhartz, Krause, Heron, McAll Anderson, Goetsch, Osler, Latham, Pottenger, Rembold, Kirchner, and others. Koch now teaches that the medicament should be used only in early and non-febrile cases, and that the doses should at first be very small and should be cautiously increased. Regarding the use of tuberculin in early, non-febrile, and uncomplicated cases, he says: 'That tuberculin exercises an exceedingly favourable influence on all such cases, and even completely cures them, as a rule, is a fact of which I have repeatedly convinced myself.'* His rules for the use of the remedy are as follows:

1. Only patients who have no fever, and in whom the process has not advanced too far, are suited for the treatment.

2. One begins with a very small dose, and increases it so slowly, that only very slight reactions, or even none, take place.

3. If reactions take place, tuberculin must not be injected again until the temperature has been normal for one or several days.

* R. Koch, Transactions of the British Congress on Tuberculosis, vol. iii., p. 95.

4. The treatment with tuberculin must be repeated till, after an interval of three or four months, the capability of reaction is permanently extinct.*

Lenhartz rejects Tuberculin R, but makes regular use of Tuberculin-Alt, and speaks highly of his results. He is of opinion that the cure of tuberculosis is materially promoted by the use of tuberculin. He begins with a dose of $\frac{1}{10}$ milligramme, and increases the dose only when no serious febrile reaction takes place. The dose is gradually increased to $\frac{1}{4}$ or $\frac{1}{2}$ milligramme. The injections are made at considerable intervals of time, and are repeated only after all signs of reaction have subsided and the temperature has been normal for several days. If no reaction occurs, the injections are repeated every second day. This delay is necessary, as in some individuals a 'late' reaction, beginning from twenty-four to thirty hours after the injection, takes place. Lenhartz advises that after a series of injections a considerable pause should intervene before they are renewed. While the injections are proceeding, the patient should remain in bed, and have as good nourishment as possible.† Pottenger has collected statistics of 1,200 cases of incipient pulmonary tuberculosis, of which 589 were treated with culture products, and 611 by ordinary hygienic methods. Of the former, 496—*i.e.*, 84·2 per cent.—were reported as apparently cured, while of the latter only 391—*i.e.*, 64 per cent.—were reported as apparently cured. This author reminds us that tuberculin and the allied substances have no influence on germs other than the tubercle bacilli, and that they are, therefore, of little use where mixed infections are present. Also, that when active immunization is attempted, the cells of the organism are stimulated to react

* R. Koch, Transactions of the British Congress on Tuberculosis, vol. iii., p. 95.

† H. Lenhartz in Ebstein and Schwalbe's 'Handbuch der Praktischen Medizin,' Bd. i. 1, pp. 324, 325. The doses recommended by Lenhartz are much larger than those usually employed.

and produce substances antagonistic to the toxic material produced; this process involves a tax upon the organism, and hence tuberculin and the allied products are not useful in the later stages of the disease. Lastly, that when using tuberculin all other useful modes of treatment should be persevered in. Streptolytic serum may be employed when streptococci are present.*

As a distinctly adverse opinion upon the use of tuberculin, I may quote West: 'Clinical observation, however, has demonstrated that the remedy has unfortunately by no means the value which Koch originally claimed for it. Even in the case of lupus, where the effect is most striking and the conditions most favourable for cure, there has been no case of complete cure, while in phthisis the results have been utterly disappointing. Even in the most successful cases the improvement has not been greater than is often seen under various other forms of treatment when patients are taken into hospital and carefully treated. In many instances the disease runs its course unaffected and in others it is aggravated.'† Klebs has prepared a modification of tuberculin, to which he has given the name *antiphthisin*. Its preparation is based upon the theory that the harmful constituents of tubercle are stored in the bodies of the bacilli, and that the specific healing substances are contained in the cultural fluid as secretory products. Other serums have been prepared by Maragliano, Marmorek, Maksutow, Hirschfelder, Landmann, Béraneck, and others. Our limits will not permit the description of the modes of preparation of these sera, or of the experience which has up to the present been gained in their use. There is no evidence to warrant the belief that any of these preparations are likely to supersede Koch's tuberculin. My recent experience has been

* F. M. Pottenger, *Zeitschrift für Tuberkulose und Heilstättenwesen*, December, 1904.

† S. West, *op. cit.*, vol. ii., p. 541.

confined to the use of the old tuberculin, in minimal doses, and chiefly in cases of tubercular peritonitis. The results have been sufficiently encouraging to convince me that the remedy is at least worthy of further trial. As I write, the remedy of Behring has been brought before the world, but the evidence at present available as to its utility is still inconclusive. Further trials of this remedy will be awaited with much interest.

Anti-streptococcic serum has been used in the treatment of pulmonary tuberculosis, with a view of combating secondary infections, and good results have been reported by Menzer, Bonney, and others.

To Wright we owe the terms 'opsonins' and 'opsonic index,' and the interesting facts in connection therewith. Opsonins are substances which modify tubercle bacilli in a manner which renders them an easy prey for the phagocytes. Opsonins are contained in the blood fluids—serum or plasma—and exercise their influence by effecting a modification of the bacilli, and not by exerting a direct stimulating effect upon the phagocytes.

The 'opsonic index' of the individual is the ratio between the average number of tubercle bacilli ingested by each polynuclear white corpuscle in his blood as compared with the average of the healthy individual. The opsonic power is approximately the same for all healthy people, and in such persons it varies but slightly, or not at all, from day to day.

According to the teaching of Wright, an increased opsonic power is to be expected in those cases in which there has been an active response on the part of the machinery of immunization to the stimulus of infection; and a decreased opsonic power is to be expected in individuals in whom there is an inherent deficiency in opsonic power, or in whom this machinery is becoming exhausted.

The opsonic power of the majority of patients suffering from pulmonary tuberculosis is higher than normal, the

explanation probably being the absorption of toxins from the lungs. While the opsonic index of the healthy individual is stable, that of the phthisical subject is subject to many and considerable variations. When the opsonic index of a tubercular patient is low, the inference is that there is either some inherent deficiency or an exhaustion of the machinery of immunization; and a low opsonic index thus, in general, involves an unfavourable prognosis.*

After inoculation of tubercular subjects with tuberculin, there is first a negative phase, during which the opsonic power is decreased; this negative phase being succeeded by a positive phase, during which the opsonic power is increased. If the inoculations are successful, the result is a permanent increase of opsonic power. In healthy persons it would appear that after injections of tuberculin the opsonic index rises at once, without any 'negative phase' or preliminary fall.

In nineteen cases of pulmonary tuberculosis, Bulloch found the opsonic index very variable, figures being often attained considerably above the normal. A large number of cases of 'arrest' or 'cure' of early pulmonary tuberculosis showed an opsonic index considerably below the normal.†

B. Antiseptic Treatment.

The antiseptic treatment of pulmonary tuberculosis may be conceived as contemplating the following objects:

(*a*) The destruction of the tubercle bacilli *in situ*, or the limitation of their activity by remedies reaching the seat of the disease *per viam respirationis*.

(*b*) The destruction of the bacilli, or the limitation of their activity by remedies acting through the blood.

* See a paper by R. H. Urwick, M.D., 'Observations on the Opsonic Power of People suffering from Tuberculosis,' *British Medical Journal*, July 22, 1905.

† *Lancet*, January 21, 1905.

(*c*) The immunization of the pulmonary tissues, so as to render them resistant to the action of the bacilli.

These ends have been attempted by the following methods:

1. By inhalations of creosote, carbolic acid, thymol, formaldehyde, or resorcin.

2. By intra-tracheal injections of menthol, izal, or some analogous agent.

3. By intra-venous injections of formalin, protargol, or cinnamic acid.

4. By the internal administration of creosote or guaiacol.

5. By the introduction of antiseptic medicaments to the seat of the disease through the pores of the skin by static high currents of electricity of high or medium tension (Crôtte method).

I may say at once that my own somewhat extensive trial of antiseptic treatment in phthisis has been limited to two of the above methods—viz., antiseptic inhalations, and the internal use of creosote and guaiacol. Intra-tracheal injections strike me as ill conceived and hazardous. That injections of menthol in olive-oil or izal in glycerine into the trachea ever reach the foci of the disease seems dubious. The bronchial tree is complicated: in such a labyrinth it would seem a matter of chance whether remedies of this class ever reach their destined mark, and there are well-known anatomical reasons for believing that the apical region is peculiarly ill adapted to receive remedies by way of the bronchi. That treatment of this type is not without serious risks is evident from the fact that it has in several cases been followed by gangrene of the lung. I am decidedly of opinion that the treatment of phthisis by intra-tracheal injections is not within the scope of legitimate therapeutics. Intra-venous injections of formalin, protargol, and cinnamic acid have been lauded by various authorities. I have no personal experience of this method, and I should desire stronger evidence than that which is at present available before giving it a trial.

From what we know of the capacity of tubercle bacilli to withstand chemical substances, there seems little prospect of being able to convey such substances in sufficiently strong solution to the foci of disease by way of the blood current. Of the Crôtte electrical method I have no experience.

There remain for consideration two antiseptic methods, of which it can at least be said that they are theoretically sound, and that they involve no risk to the patient—viz., the use of antiseptic inhalations, and the internal administration of remedies of the creosote and guaiacol class. Antiseptic inhalations had at one time an extensive vogue in the treatment of phthisis, and it is not to be denied that remedies such as formalin have a favourable influence upon the secretions from bronchi or vomicæ, and may tend to check some of the secondary infections—*e.g.*, streptococci, staphylococci, pneumococci, the influenzal bacteria—which are so often associated with phthisis. That such remedies exert any direct controlling influence over the bacilli of tubercle seems doubtful, and I imagine their use has fallen into general, and not ill-deserved, neglect. A prolonged trial of various remedies thus administered failed to convince me of their utility. It has also to be borne in mind that the use of inhalers, mouth-pieces, and respirators of various kinds, is open to the grave initial objection that it tends to interfere with what is without question the primary condition of all successful treatment of pulmonary tuberculosis—viz., the constant flooding of the lungs with pure air.

Creosote, creosotal, and guaiacol, administered either by the mouth or subcutaneously, have now had a prolonged trial in pulmonary tuberculosis, and are still amongst the most favourite remedies. Beech-wood creosote has been specially recommended, and we are advised to give the remedy in capsule form after meals. There is some evidence to show that creosote and its congeners lessen cough and expectoration, and in certain cases have a favourable influence upon

the digestion. As regards this latter point, however, it is important to bear in mind that the limit of toleration for creosote is in some patients soon reached, and that the too vigorous use of the remedy may excite nausea and disagreeable eructations. Cornet's experiments on animals seem to show that creosote has no power of hindering or delaying the growth of bacilli in the body.* Klemperer believes that creosote has a favourable general influence upon the digestion and nutrition. I have used creosote and its derivatives somewhat extensively in the treatment of phthisis, and there can be no doubt that some patients improve under their use, but as I never employ it without the adoption of general tonic and hygienic methods, I have a difficulty in deciding to what extent the benefit often observed is attributable to the use of creosote. I am inclined to the opinion that it has no specific virtues, but that it suits some cases well, and that, in default of better remedies, it may be used with advantage from time to time. It should never be persevered with if its effect upon the appetite and digestion is unfavourable. It has also to be borne in mind that the systematic use of remedies of doubtful utility in so chronic a disease as phthisis is in general to be deprecated. Patients are so ready to take our drugs, so unready to accept our admonitions as regards wholesome living. The systematic use of such remedies as creosote and guaiacol may in certain cases serve to divert the attention of both doctor and patient from measures of really proved utility.

C. The Hygienic Treatment of Pulmonary Tuberculosis.

Two of the earliest pioneers of hygienic methods in the treatment of phthisis (although such ideas were foreshadowed by Hippocrates and Sydenham) were George Bodington of

* G. Cornet, 'Nothnagel's Encyclopædia of Practical Medicine,' English edition, 'Tuberculosis,' p. 582.

Sutton Coldfield, Warwickshire, and Henry MacCormac of Belfast. Writing in 1840 Bodington insisted on the importance of a generous diet, fresh air day and night, exercise, and medical supervision. 'Cold,' he said, 'is never too intense for a consumptive patient; the apartment should be kept well-aired, so that it should resemble the pure air of the outside, pure air being used in the treatment as much as possible.' Writing in 1856 Henry MacCormac said: 'It is impossible to urge in terms too strenuous, too explicit, the indispensableness of open-air life and effort in respect of the prevention of disease in general, and of phthisis in particular.' MacCormac's view of the causation of phthisis was that it is due to breathing 're-breathed' air. His pathology was erroneous, or at least inadequate—Koch's discovery was still a quarter of a century in the future—but his views on treatment were sound, and in some degree original.

To Brehmer and Dettweiler, however, rightly belongs the credit of having established the modern hygienic treatment of phthisis upon a firm and, I believe, unassailable basis. This treatment has now had a trial of forty years, and the amount of experience gained in this department has been very large. In Germany, the birthplace of these methods, the number of sanatoria available for this treatment exceeds 100, and the number of cases treated annually is more than 30,000. My own experience of the treatment has been considerable, and has had relation to sanatoria in Ireland, England, Scotland, Wales, Germany, Switzerland, Austria, and the United States of America. A recent animated controversy in the medical press has amply proved that there is still much difference of opinion amongst medical practitioners regarding the value of sanatorium treatment.* Some writers flatly deny that there are any cases of cure, and maintain that this method of treatment is costly and wasteful, its

* 'The Sanatorium Treatment of Phthisis: Is it Worth While?' *British Medical Journal*, 1905.

results wholly disappointing, and that the statistics afforded by the various sanatoria are quite misleading. Against such extreme views we have the solid facts that sanatorium treatment continues to make steady headway in nearly all civilized countries, and that the large German experience shows that about two-thirds of the cases subjected to the treatment recover, at all events, sufficiently to return to their avocations for a longer or a shorter period. A large majority of these 'cured' cases relapse within four years.

The hygienic treatment of phthisis is, of course, not necessarily synonymous with the sanatorium or institutional treatment, but both these methods rest on the same principles and are essentially identical in their general outlines. Let us consider this subject from three points of view—viz.:

(*a*) What are the principles and methods of the hygienic treatment of phthisis?

(*b*) What are the results hitherto attained, or fairly to be expected, from this system?

(*c*) What are the drawbacks of the system, and why is the system still viewed in many quarters with disfavour?

(*a*) The hygienic treatment of phthisis rests upon the sound principle that, in default of a trustworthy specific, the best means of combating the malady is to raise the general health to the highest possible level—flood, as it were, the tuberculized area with healthy blood and pure air—in the conviction that when these conditions are fulfilled the disease will in many cases abate. In well-nourished and well-ventilated lungs, it is not rash to assert, tubercle maintains itself with difficulty. Whether the patient will do better in an organized sanatorium or in his own extemporized and solitary shelter; how much medical supervision counts for; how far certain auxiliary lines of treatment and the control of symptoms influence the final result; what weight is to be attached to locality, climate, meteorological and telluric conditions—these are interesting

and important questions, but I think we should teach that the absolutely indispensable conditions of the hygienic treatment of phthisis are few and simple. They are these:

That the patient shall breathe absolutely pure air at all times, day and night;

That he shall take abundance—even an excess—of simple, nourishing food;

That he shall follow certain well-recognised rules as regards rest and exercise; and

That he shall dispose of his sputum so as not to re-infect himself.

This is the essence of the case, though by no means all. The choice of food, the improvement of digestion, the regulation of the bowels, the hygiene of the skin—these are points of real importance. I repeat, however, that the essentials of the hygienic method are few and simple; that they may be applied in most places and by most persons, with suitable guidance; and that, if this method of treatment is to become universally applicable, it is desirable to render it as simple as possible, and to shun any unnecessary elaboration. I have seen the most excellent results when the patient has simply built himself a common wooden shelter on his father's farm, lived on milk, eggs, and such-like food, and made it a rule to rest when his temperature was above normal. No doubt, we must allow for the influence of the personal equation. Not every patient can be trusted to manage his own case; many imperatively require supervision and control. But this is not because the treatment involves any real difficulties; it is because system, intelligence, and perseverance are so often lacking on the part of the patient. The due regulation of rest and exercise is one of the most vital points in hygienic treatment, and here sanatoria possess a great advantage, as in these institutions these matters can be carefully attended to. The chief guide is the state of the temperature. Febrile

patients, with very few exceptions, should have complete rest. Non-febrile patients should take regulated exercise, duly proportioned to the state of the pulse and the general vigour. Any form of exercise which raises the temperature 1° C. or more is doing harm. A too long persistence in rest tends to the deposit of adipose tissue, and makes the patient less fitted to resume habits of activity on leaving the sanatorium. The after-care of patients who have undergone sanatorium treatment with more or less benefit is only beginning to receive attention, but it is a vital matter. If these patients return to indoor occupations with doubtful hygienic surroundings, relapse is almost a certainty, and will take place probably in from one to four years. Some form of tubercular colony, with a systematized hygienic routine and various grades of labour, is required for these cases. We shall return to this point on a later page.

(*b*) What are the results hitherto attained, or fairly to be expected from, the hygienic treatment of phthisis? On this subject wide differences of opinion still prevail. Satisfactory data on the subject do not exist, and I put forward the following items rather as indicating opinions than assured results:

The German Imperial Health Office analyzed the results of treatment in 6,273 cases treated in sanatoria in the years 1899 and 1900, with the following results: In the opinion of the sanatorium physicians 87·7 per cent. were cured or improved, of whom 67·3 per cent. were regarded as sufficiently well to resume work at their former occupation. The after-history of 2,147 patients was inquired into, and it was found that of each 100 cases dismissed as being able to work, only 21 per cent. were able to work four years later. At first sight this seems very disappointing, but it is to be observed that this heavy rate of relapse in patients resuming their former more or less unhealthy occupations gives us no fair gauge of the permanent results of sanatorium treatment, if hygienic surroundings could be secured for patients on

leaving a sanatorium. Another return of the German Imperial Health Office shows that of 1660 cases of early phthisis subjected to sanatorium treatment, 44 per cent. were able for their work four years after leaving the sanatorium. This serves to emphasize the relatively good results which may be expected in early cases.

Sir Hermann Weber informs me that, as the outcome of his inquiries into the results of sanatorium treatment in and out of England, he would be inclined to put the proportion of cures as follows:

1st stage cases—	cured,	74	per	cent.
2nd ,,	,,	48	,,	
3rd ,,	,,	19	,,	

Dr. Turban (Davos) reports:

1st stage cases—97 patients: improved, 95; not improved, 2.

2nd stage cases—205 patients: improved, 174; not improved, 31.

3rd stage cases—106 patients: improved, 56; not improved, 50.

In 116 cases the sputum was free from bacilli at the time of the patient's discharge. Fourteen patients (3·4 per cent.) died while under treatment. Capacity for work (*Leistungsfähigkeit*) on discharge from the sanatorium was noted as unimpaired in 80 of the 95 cases of improvement in the 1st stage; in 76 of the 174 cases of improvement in the 2nd stage; in 5 of the 56 cases of improvement in the 3rd stage.*

Dr. Burton-Fanning reports: 716 cases collected from various sanatoria in England, Scotland, and Ireland.†

* K. Turban, 'Beitrage zur Kenntniss der Lungen-Tuberkulose,' 1899.

† Burton-Fanning, Transactions of the British Congress on Tuberculosis, vol. iii., p. 199.

Quiescence of the disease or relative recovery was obtained in 37·4 per cent. of cases; amelioration in 40·2 per cent; no improvement in 22·3 per cent. A gain in weight took place in 92 per cent. Only 52 of these cases (7·4 per cent.) could be described as 'cases of slight lung mischief.' Of this group 46 (*i.e.*, 88·4 per cent.) 'did well and obtained relative recovery with arrest of the disease.' Tubercle bacilli disappeared from the sputum in only 19 (*i.e.*, 41·3 per cent.) of these 46 patients. In some of the remaining cases the presence of bacilli was doubtful.

Dr. Morin (Leysin) reports:

1st stage—cured,	50	per cent. ;	improved,	40	per cent.
2nd	,,	,,	10	,,	,,	67	,,	,,
3rd	,,	,,	3·2	,,	,,	48	,,	,,

The Basler sanatorium at Davos reports that 64·3 per cent. of all cases were still able to work four years after their discharge from the sanatorium.*

Meissen of Hohenhonnef reports that of 248 patients, who spent an average of five months in the sanatorium and whose condition was inquired into from three to eleven years later, 84 per cent. were sound and able to work, 16 per cent. had relapsed, of whom about 3·6 per cent. had died.†

Dr. Chowry-Muthu reports that of 152 patients treated at the Mendip Hills Sanatorium during the four years ending August, 1903, 78 were restored to health and returned to work, while of the remaining 74 cases the majority had been admitted in an advanced stage of the disease.‡

Dr. Thurnam, of Nordrach-on-Mendip, reports that of 183 advanced cases under his care during a term of four years 35 (*i.e.*, 19·1 per cent.) were perfectly well and in full work at the end of that period, while 39 (*i.e.*, 21·3 per cent.) were

* *Centralblatt für Innere Medizin*, 1902.
† *Zeitschrift für Tuberkulose und Heilstättenwesen*, 1903.
‡ *British Medical Journal*, July 1, 1905.

in fair health and able to do a certain amount of work.* This evidence is the more important as the general tendency is to regard sanatorium treatment as of little avail in advanced cases.

The Rutland Sanatorium (Massachusetts), a large institution for early cases, with an average of about 250 patients, reports arrest of the disease and apparent cure in 73 per cent. of cases.† There are now 135 institutions of various kinds for tuberculous patients with accommodation for 8,400 cases in the United States and Canada.

Dr. Schmidt, chief of the Sanitary Bureau of the Swiss Confederation, reported to the last International Congress on Tuberculosis (Paris, 1905) that Switzerland had one sanatorium bed for every 4,000 of the population; that 71 per cent. of the patients were restored to their full working capacity; and that after five years 43 per cent. still retained this capacity.

Dr. Sophus Bang, chief physician of the Silkeborg Sanatorium in Denmark, reported to the same Congress that Denmark possessed one sanatorium bed for every 3,000 of the population, and that the results were very good. The average duration of treatment in the Danish sanatoria is six months.

Dr. Kayserling, general secretary of the Berlin Poor Relief Organization, speaking at the same Congress, stated that the popularity of the sanatoria was so great, and the conviction of their usefulness so strong, that any attempt to close them in Germany 'would be enough to provoke a revolution.'

Similar evidence in favour of the utility of sanatoria could be multiplied indefinitely. In spite of discrepancies in the statistics, we find a considerable degree of accord amongst those best qualified to speak with authority on the following points—viz.:

* *British Medical Journal*, March 4, 1905.

† *Ibid.*, August 19, 1905.

(*a*) That a large degree of improvement, amounting in many cases to complete arrest or 'relative cure,' may be expected from sanatorium treatment.

(*b*) That the degree of success attained by this method of treatment has a close relation to the stage of the disease at which the treatment is undertaken and the thoroughness with which it is carried out.

(*c*) That the proportion of relapses is very high amongst sanatorium patients who return to their former unhealthy occupations.

(*d*) That in order that sanatorium treatment should prove more successful, and justify the large outlay involved, it is necessary that a wise selection of cases for treatment should be made, and that sanatoria should not stand alone, but be simply a link (though a very important link) in the chain of preventive and curative machinery employed in the battle against tuberculosis.

In what proportion of cases subjected to sanatorium treatment can the word 'cure' be fairly used? This is a very difficult question to answer. We have seen that 'cure' is claimed by various authorities in a proportion varying from 37 to 74 per cent. of cases, while, as the recent controversy in the *British Medical Journal* showed, many observers deny that any cures are affected, and even so judicious a writer as West speaks as follows: 'Sanatorium treatment cannot change the natural course of the disease. If this is expected, disappointment will follow. It will not cure the disease, but it will assist the patient to resist the inroads of it.'* In this connection the very embarrassing question arises, How often does cure take place in phthisis without treatment, or where a diagnosis of the disease has never been made? The proportion of persons dying of other diseases whose lungs present evidence of healed tuberculosis is not less than 10, and may be 20, per cent. I think it highly probable that in

* S. West, 'Diseases of the Organs of Respiration,' vol. ii., p. 565.

many of these cases the disease never progressed to the extent of causing definite signs and symptoms, but this is theoretical. Experience has taught me that if we make a point of examining the sputum in every case of 'lingering cold,' or repeated 'bronchial attack,' we shall sometimes find tubercle bacilli when not in the least expected, and that in a certain proportion of these cases the disease does not proceed further. If this form of arrest sometimes occurs without treatment, *a fortiori* it may be expected to occur in connection with the favourable conditions involved in sanatorium treatment. My own experience of the treatment, while highly favourable as regards such results as 'great improvement,' 'relief of all serious symptoms,' 'restoration to fair general health,' does not incline me to speak confidently on the subject of 'cure,' but I cannot subscribe to the pessimistic dictum, 'Once a consumptive, always a consumptive.' Morbid anatomy is decisive on the point that healed tubercular scars may give no further trouble, and involve no impairment of health or energy. The term 'economic cure' is much used by German authorities in the sense that the patient has been restored to full working capacity. On the whole, the term seems to me somewhat misleading. The patient is not, in most cases, cured in any real sense of the term. He is restored to a condition of more or less precarious health-equilibrium, if we may use such an expression.

When 'arrest of the disease' or 'great improvement' has been obtained, Are these results lasting? Of all questions in this connection this is most vital. The treatment of phthisis on a large scale is an economic question. Granting that great benefits may be obtained from sanatorium treatment, Are these benefits permanent or at least fairly enduring? Are they worth their cost? This question has already been glanced at; let us now consider it a little more fully. On the answer which we can give to it probably turns the whole attitude of civilized nations towards the

sanatorium treatment of phthisis. We have seen that, according to the careful returns prepared by the German and Swiss health authorities of patients discharged from sanatoria as 'arrested,' from 21 to 44 per cent. retained full working capacity four or five years after discharge, the higher percentage referring to early, the lower to unclassified, cases. Turban* supplies the following evidence on this point: Queries were sent to discharged patients, and 225 replies were obtained. In 68 cases the patient's condition was described as 'stationary since discharge,' in 127 as 'improved,' in 30 as 'worse.' In 126 other cases the patient was found to be dead. The cases were spread over a period of seven years, but the length of time which had elapsed from the patient's discharge from the sanatorium to the date of the inquiry—viz., 1897—is not stated. Turban's results, therefore, work out as follows:

Total number of patients in one septennium	408
Obtained marked improvement	325
Died while under treatment	14
Died before 1897	126
Improved after discharge from sanatorium ...	127
Stationary since	68
Lost ground since	30

A further query was put to former patients—viz., Is the patient's activity (*Leistungs-fähigkeit*) 'not impaired,' 'slightly impaired,' 'much impaired,' or 'annulled'?—and to this query 226 replies were obtained as follows:

Not impaired	151 cases
Slightly impaired	45 ,,
Much impaired	18 ,,
Annulled	12 ,,

These results, if not exactly brilliant, are at least encouraging. They point to the conclusion that the improve-

* K. Turban, *op. cit.*

ment obtained by sanatorium methods is not a mere transient rally, but possesses some elements of permanence. It must be borne in mind that a patient usually leaves a sanatorium a thorough convert to hygienic methods. He is fanatical for fresh air and cleanliness; he has sound views upon food, rest, exercise, and the disposal of sputum; he knows the dangers incident to certain occupations. Hence it is not unduly sanguine to hope that the benefits which he has obtained may endure. He has started upon a new career, and may have a new and happier fate. It is important to emphasize these points, as a widespread prejudice exists, which is not without some foundation, that the benefits obtained in sanatoria are brief and illusory. In any fair estimate of the value of sanatoria we must give a high place on the credit side of the account to their unquestionable educational influence, which is doing something to spread the leaven of sound hygienic principle precisely in the quarter where it is most needed.

The criteria of a patient's improvement under sanatorium treatment are the following:

(*a*) Gain in weight, strength, capacity for exertion, breathing power, pulse.

(*b*) Freedom from pyrexia.

(*c*) Diminution or disappearance of physical signs.

(*d*) Disappearance of tubercle bacilli from the sputum.

Gain in weight, taken alone, is a fallacious test of improvement. Rest and high-feeding will produce a temporary gain in weight in most patients (probably at least 90 per cent.), whether real improvement is in progress or not. Some most melancholy examples have occurred in my experience of patients who, in response to rest and hyper-alimentation, have gained several stones in weight without any real or lasting improvement. In these disappointing cases the patient, in spite of the gain in weight, is weak and short of breath, his pulse keeps frequent, and the physical signs in

the lungs do not recede. The prognosis in such cases is highly unfavourable. On the other hand, a steady and progressive gain in weight, if accompanied by improvement in pulse, energy, and local signs, is most important. Freedom from pyrexia is found in nearly all cases which make satisfactory progress, but a slight degree of afternoon pyrexia is not inconsistent with marked improvement. Retrocession of the physical signs in the chest is highly important. In favourable cases crepitations become fewer and drier, or disappear altogether; signs of shrinkage of the affected apex appear; compensatory emphysema of the sound lung can be detected. Disappearance of the bacilli from the sputum is of favourable augury, but bacilli may persist in the sputum of patients whose general condition is quite favourable.

Let us consider in concluding our survey of this subject how it comes to pass that, if the case for sanatoria is seemingly so strong, if this method of treatment is making rapid strides in most civilized countries, adverse opinions should be so freely expressed and even the utility of sanatorium treatment denied. Even Cornet, who is decidedly in favour of this method of treatment, writes as follows: 'As one of the first who championed these establishments by word and deed, I cannot refrain from issuing a warning against the too roseate hopes which have been aroused, lest the disillusionment be overwhelming. According to my experience, the three-months' cure is too brief; it may bring about improvement, but rarely, even in milder cases, a definite cure. The patients are discharged relatively cured, return to their old surroundings, to their hard labour in dusty workshops—most of them being workmen—and to their unhealthy dwellings, and it is not strange that the process begins anew. . . . The establishment of these institutions is only a half-measure, the enormous outlay of money useless, the success only apparent, if we do not also give heed to the immediate future of the patient, if we do not improve the conditions of the

home, do not relieve him of a part of his responsibility toward his family, and supply him for some time with a less injurious occupation, and keep him busy, if possible, in the open air—requirements which it is easier to enumerate than to fulfil.'* All this I take to be sound and not over-stated, but let us be a little cautious as to the conclusions which we draw from these arguments. The true conclusion is not that sanatorium treatment is ill-designed or ineffective, but that it is only part of an indispensable system, and that standing alone it is both economically wasteful and disappointing in its present results. Two things are absolutely indispensable if sanatorium treatment is to achieve its best results—viz. (*a*) a proper selection of cases for treatment; and (*b*) the recognition of the fact that the discharged patient, who has undergone 'arrest' of his disease, or 'economic cure,' is not in a fit state to return to his former avocation, but requires further guidance and assistance, for which end some form of 'tubercular colony' is indispensable.† I am also of opinion that this subject will remain in its present confused and controverted condition until we frankly distinguish sanatoria for the cure or arrest of the disease, and homes for incurables for the assuagement of the sufferings of hopeless cases. These two lines of treatment can never be successfully pursued in the same institution.

Sanatorium treatment has also incurred odium from its costliness, and from a fear that the moral effects of prolonged rest and good feeding are in many instances undesirable. There is no objection to luxurious institutions costing nearly £1 per head per diem for wealthy patients, but for the masses of the people simple buildings and a plain régime should be aimed at, and the cost should never exceed £2 per head per week, and, if possible, should

* G. Cornet, *op. cit.*, pp. 576, 577.

† See an able letter by Dr. R. C. Macfie, 'Sanatoriums and the Eradication of Consumption,' *Lancet*, September 30, 1905.

be much less than this sum. It is also clear that charity will never undertake the immense task of the treatment of tubercular cases, that some form of compulsory insurance or of combined action on the part of assurance companies and benefit societies will be necessary. Such bodies will gradually discover that it is cheaper to give their tubercular clients treatment at a sanatorium and restore them to, at all events, several years of working life than to allow them to draw unlimited sick-pay and die off by degrees. The State and municipal authorities should assist in this work.

LECTURE XX

THE TREATMENT OF PULMONARY TUBERCULOSIS

SUMMARY:

D. Symptomatic treatment:

(*a*) Remedies to improve nutrition.

(*b*) Remedies to control pyrexia.

(*c*) Remedies to control cough, expectoration, hæmoptysis, etc.

(*d*) Remedies to promote the hygiene of the skin.

(*e*) Remedies directed against complications—*e.g.*, ulceration of the larynx, ulceration of the intestines, amyloid degenerations, anæmia, dysphagia, fistula in ano.

E. Climatic treatment:

Place of climatic treatment at the present day.

Direct and indirect influence of climate.

Classification of climates:

(*a*) The oceanic climate.

(*b*) The marine climate.

(*c*) The inland climate of slight or moderate elevation.

(*d*) The high-altitude climate.

The oceanic climate—meteorological characters, indications, and contra-indications.

The marine climate—meteorological characters, indications, and contra-indications:

The Riviera, the Home Resorts, the Canary Islands, Madeira, Sicily, Algiers, Tangier, Southern California, Florida.

The inland climate of low or moderate elevation —meteorological characters, indications, and contra-indications:

Egypt, Algeria, South Africa, Australia, New Zealand, Arizona.

The high-altitude climate—meteorological characters, indications, and contra-indications:

Davos, St. Moritz, Arosa, Leysin, Colorado.

Clinical types of phthisis, and the choice of climate which they indicate.

D. Symptomatic Treatment of Pulmonary Tuberculosis.

The symptomatic treatment of phthisis takes at the present day a very subordinate place. If the broad principles of hygienic treatment have been adopted, nothing can be more undesirable than a fussy attention to symptoms. The elaborate discussion of medicinal remedies for the disease in which some text-books indulge is apt to create an entirely erroneous impression of their value, which is rarely more than subsidiary. One may hope that the practitioner who fortifies his tubercular patient with a row of medicine bottles—one for his cough, another for his digestion, a third for his night-sweats, a fourth for his bowels, and so on—is nearly extinct. Such treatment is a parody on genuine therapeutics. Nevertheless, the phthisical patient is subject to many conditions which can be favourably influenced by remedies, and there can be no objection to such treatment, provided that its strictly subordinate character is steadily borne in mind. I shall consider these remedies under the following heads:

(*a*) Remedies to improve digestion, nutrition, and circulatory efficiency.

(*b*) Remedies to control pyrexia.

(*c*) Remedies to control cough, expectoration, hæmoptysis, pain in the chest.

(*d*) Remedies to promote the hygiene of the skin.

(*e*) Remedies directed against complications—viz., ulceration of the larynx, ulceration of the intestines, amyloid degenerations, anæmia, dysphagia, fistula.

(*a*) REMEDIES TO IMPROVE NUTRITION.—Our first thought in the treatment of phthisis should be the promotion of nutrition. The digestive condition of the consumptive is variable. Often he suffers much from dyspepsia, not seldom he surprises us by digestive vigour. The dyspepsia of phthisis takes on many forms, and cannot be usefully differentiated from the dyspepsia of other origins. In these days of high-feeding in phthisis, the catarrhal type of dyspepsia (*e.g.*, sub-acute gastritis) is common. Tuberculous ulceration of the oral cavity and aphthous stomatitis are occasionally observed. They are usually found in severe and advanced cases, and are of very evil augury. The tongue varies much. If pale and flabby with slight fur, atonic dyspepsia is probably present. If thickly coated, with prominent papillæ, gastric catarrh may be suspected. If red, raw-looking, or glazed, intestinal ulceration may be inferred. Diarrhœa may be troublesome.

These conditions must be treated on general lines. It is well to make sure that the food is not excessive in amount or unsuitable in character. It is no infrequent experience of mine to be informed by patients that they consume two or more quarts of milk and four to six eggs daily; yet such patients sometimes complain sadly that 'they never have any appetite.' It would be strange if they had. In the dyspepsia of phthisis the drugs most often useful are alkalies and bitters, especially nux vomica. Acids are seldom indicated. If signs of catarrhal gastritis are present, rest in bed for a few days, liquid diet, and warm compresses to the abdomen may be tried. Nervous anorexia may be combated by change of climate, the open-air cure, hydrotherapy, creosote, guaiacol,

lavage, electricity, mineral waters.* The bowels often require attention. Constipation may be treated by the usual dietetic rules and the moderate use of aperients. Diarrhœa is a more serious matter. If it depends on dyspeptic or catarrhal conditions the bowels should first be emptied with castor-oil, a bland non-irritating diet ordered, and, if required, some vegetable astringent given. The diarrhœa of intestinal ulceration is usually very intractable. The diet should consist of materials which leave little residue. Peptonized milk or raw-beef juice may be tried; in some cases raw-beef sandwiches seem to do well. Opium is the best medicinal remedy. Enemata of nitrate of silver may be tried. Rest should be enforced. Similar measures may be adopted in the diarrhœa of amyloid disease of the intestines, which also is very intractable.

Tonics and artificial nutrients have a considerable utility in phthisis. Vegetable bitters, nux vomica, quinine, arsenic, and the hypophosphites may be employed with advantage. Cod-liver-oil and maltine are valuable nutrients. Alcohol should be sparingly used, but the use of a good red wine sometimes improves appetite and digestion.

(*b*) Remedies to control Pyrexia.—The control of pyrexia is often one of the most important points in the management of phthisis, and it is to be feared that it is not always well directed. The practitioner too often is not sufficiently strict in enforcing rest, and too ready to order antipyretic drugs, which give a delusive show of improvement. The febrile patient should in most cases have absolute rest, unless the pyrexia is very slight—*e.g.*, under 99·5° F. The important point is to secure that rest shall be taken in the fresh air, for which purpose some form of balcony, shelter, or tent is indispensable. Too much exposure to the direct rays of the sun should be avoided. Rest should be enforced even in cases where the febrile rise is confined to two or three hours in the afternoon. In chronic cases

* G. Cornet, *op. cit.*, p. 617.

with excavation the rule regarding rest in the presence of pyrexia may be somewhat relaxed. Next to rest may be placed strict attention to the hygiene of the skin—viz., tepid or cold spongings, with the addition of some fragrant spirit to the water. Antipyretics, such as antipyrin, antifebrin, phenacetin, salipyrin, and salicylate of soda, have quite failed in my hands to do any permanent good, although their occasional use may be justifiable. Quinine often does good, but rather from its tonic than its antipyretic properties. Brehmer advises the use of alcohol to combat the phthisical pyrexia, and gives a glass of strong wine (8 to 12 per cent. of alcohol) at the onset of the more rapid rise, and a second similar dose upon a second rise. Arsenic has been recommended for the chills of phthisis. An ice-bag may be laid over the heart, but care should be taken not to retain it there too long. The pyrexia of phthisis is often aggravated by mixed infections. The diet in pyrexial cases should be as generous as the state of the stomach and digestion will permit. It is quite an error to keep these cases on fluid diet.

(*c*) REMEDIES TO CONTROL COUGH, EXPECTORATION, HÆMOPTYSIS, PAIN IN THE CHEST.—The broad rule in phthisis is to treat the constitutional state and ignore the cough, but cough may be irritating to the bronchial mucous membrane, interfere with sleep, or bring on vomiting. In such cases it fairly forms an object of treatment, but the routine administration of cough mixtures is, it need hardly be said, one of the most baneful of errors. Cough may be justifiably the object of treatment under the following conditions:

1. When it is irritative, more or less incessant, and interfering with digestion or sleep.

2. When it is due to ineffectual attempts to expel viscid mucus from a cavity.

3. When it is due to excessive bronchial secretion.

4. When it is due to laryngeal ulceration.

In the first class of case, morphia, codein, hydrocyanic acid, bromides, and Virginian prune are the best remedies.

In the second class, alkalies, given in hot water or hot milk, are useful.

In the third class of case we may try inhalations of hot saline waters, tar, benzoin, or formalin (2 to 3 per cent).

In the fourth class we may use sedative inhalations—benzoin, conium, hop; or antiseptic sprays; or powders such as iodoform, with or without the addition of morphia.

When the bronchial secretion is tough and difficult of expulsion, we may try the effect of open-air, moderate exercise, the use of the waters of the salt springs, warm moist inhalations, and hot compresses to the chest. For diminishing excessive secretion we may try terebene, creosote, creosotal, or myrtol.

A special lecture will be devoted to the subject of hæmoptysis.*

(*d*) Remedies to Promote the Hygiene of the Skin.—This line of treatment is important, and should on no account be neglected. With the adoption of hygienic methods, night-sweating usually ceases to be troublesome. The skin should be sponged over once or twice daily with tepid water, to which rectified or methylated spirit or eau de Cologne should be added. Atropin has a potent influence upon night-sweating, but in the long run it does more harm than good. I have abandoned its use, except in rare cases. Agaricin, picrotoxin, and sulphonal are also recommended. The sleeping-room should be kept cool, and the night-clothes should be light. Warm drinks at bedtime should be avoided. A few spoonfuls of cognac in milk before going to bed are sometimes useful.

The routine application of counter-irritants to the chest must be condemned. Where pleurisy is present, or where

* See Lecture XXI.

there is much pain in the chest, the occasional and purely temporary use of vesicants may be approved, but in general the cardinal rule in phthisis should be to keep the patient's skin scrupulously clean. There are few sadder sights in practice than that of the consumptive's chest plastered over with iodine, and in a state of positive filth—a sight, unhappily, by no means rare; it can only be called barbarous. The clothing should be warm, but not heavy. All forms of 'chest-protectors' are to be avoided. The patient should sleep in some light woollen material.

(*e*) REMEDIES DIRECTED AGAINST COMPLICATIONS. — In laryngeal ulceration the general treatment is the chief point. The local treatment consists in the use of antiseptic or sedative remedies in the form of inhalations, sprays, powders, solutions. A very active treatment of the local lesion is seldom advisable. The ulcers should be kept clean.

The treatment of intestinal ulceration has been already considered. Amyloid degenerations are little under therapeutic control. Anæmia requires iron, arsenic, and suitable diet. Dysphagia may demand feeding with the nasal tube. Fistula in ano may require the attention of the surgeon.

E. THE CLIMATIC TREATMENT OF PHTHISIS.

With the general recognition of the preponderating influence of hygienic measures, habits, and mode of life in the treatment of phthisis, the relative importance of locality and climate has in recent years somewhat receded. Yet these factors cannot be ignored, and their importance has been insisted on, amongst others, by Cornet and Nothnagel. It is alleged by some observers that 'weather' has little or no influence on the course of phthisis, that patients do as well in one resort as another, in bad seasons as in good. This is not my experience. I cannot doubt that weather counts for something with the consumptive—affects his

appetite and nutrition, as well as his cough and his breathing; affects him psychically as well as physically. If, as it is often alleged at the present day, climate has no influence upon phthisis, direct or indirect, then it follows, as a necessary inference, that to the consumptive sunshine and sunlessness are indifferent, tonic air and relaxing air, shelter and exposure, wind and calm. Will anyone accept this doctrine, thus nakedly stated? No doubt we cannot attribute to climate any definite, specific, antagonizing influence upon phthisis, and the notion that any climate is capable of preventing the development of the disease is erroneous; but climate, interpreted in a broad sense, so as to include all meteorological and telluric conditions, is a factor which only a very one-sided pathology can ignore. However, 'climatic treatment,' as I have written elsewhere, 'is not a complete therapeusis, and will be only a snare if so interpreted. It is a means to an end, not an end in itself; a powerful adjunct to hygienic and medicinal measures, not a substitute for them; a channel of escape from vicious habit and abnormal mode of life, not a mysterious remedy or an unfailing specific.'* I am quite unable to understand the views of those who hold that locality has no influence in the treatment of phthisis, one of the most outstanding facts in my experience being that patients may do well in one locality and do ill in another, although the conditions of life remain much the same. I may add that this is practically the unanimous opinion of patients themselves. The subject is, however, beset with many difficulties, and is hardly ripe for scientific definition. 'The choice of a particular climate for a particular case,' as Burney Yeo truly says, 'will frequently have to be determined by individual and personal, rather than by general and pathological, considerations.'†

* J. A. Lindsay, 'The Climatic Treatment of Consumption,' 1887.

† Burney Yeo, 'Manual of Medical Treatment,' vol. ii., p. 1.

Climate as a therapeutic agent in phthisis may be usefully regarded from two points of view—viz. :

(*a*) First, as regards its direct influence upon the patient's pulmonary condition, his nervous system, appetite, and nutrition.

(*b*) Secondly, as regards its indirect influence in either facilitating or impeding open-air life, hygienic measures, healthful occupations.

These two factors are often difficult to disentangle. No doubt the second is sometimes the more important of the two ; yet I cannot doubt that the former is also significant, however it may elude the attempt to give it precise scientific definition. That many tubercular patients under proper direction do well at home is a fact to which we have in recent years become increasingly alive. But do they, or do they not, make any better progress at Davos, Leysin, San Remo, on the South African veld, upon the uplands of Queensland, in Colorado, in Egypt ? Let those answer who have really a large experience in this matter. Some tubercular patients will do well almost anywhere, provided they adopt a rational mode of life. Some tubercular patients will do well nowhere, no matter how they live. Is there, however, or is there not, a class with whom locality and climate are factors which may incline the scale towards, or away from, recovery ? I believe there is. Experience must finally settle the question.

The question of climate confronts us also in relation to the complications of phthisis. Bronchial catarrh, anæmia, nervous irritability, sleeplessness, dyspepsia, laryngeal ulceration, may be present, and it cannot be doubted that such conditions are more or less affected by temperature, humidity of the atmosphere, wind, shelter, and the like. Again, we find that while successful sanatoria may be founded under diverse conditions of locality and climate, yet local conditions have not been ignored in the selection of their sites. A dry subsoil, an agreeable outlook, a free circulation of air com-

bined with adequate local shelter, a maximum of sunshine and a minimum of fog and mist—such are some of the conditions usually aimed at. Further, in this connection we have to consider the case of the patient who, having undergone a course of sanatorium treatment and secured arrest of his malady, desires advice as to the best localities for either temporary or permanent residence. We are asked the question whether a recrudescence of the disease is to be feared if life is resumed in the former locality, or whether a change would be advisable. Finally, there may be the question whether in advanced cases any change of locality might serve to lessen the miseries of the last months of life.

On all these counts—and the subject might easily be further developed—it will not be found advisable to act upon the assumption that locality and climate are matters which can be safely ignored in the management of phthisis. It is most desirable not to over-state their importance; it is equally essential to give their importance its due weight.

Climates are many; health-resorts are innumerable; in their choice it is indeed true that 'experience is fallacious and judgment difficult.'* We may fairly ask the question, Have the climates found useful in phthisis any common characters? Have Davos and St. Moritz any *commune vinculum* with Egypt and the Orange River Colony, San Remo and Mentone with Colorado and Queensland, Algeria and Sicily with Bournemouth and Ventnor? The answer often given to this question is that climates are useful in phthisis simply in proportion as they permit the patient to spend a longer or a shorter time in the open air with comfort and benefit. This somewhat simple formula hardly seems to exhaust the subject. The patient's age, constitution, temperament, the stage and activity of his disease, the complications which may be present, will be found to count

* Hippocrates, Aphorism I.

for something in relation to locality and climate. One of the wisest of the ancients has said: 'Those things which we use most and oftenest have the greatest influence on health; and water and air are of this nature.'* The favourite climates for phthisis are, for the most part, sunny without excessive heat; possessing the maximum of purity of the air; presenting long spells of continuous favourable weather; suitable for outdoor exercise; free from frequent violent winds; of a moderate or low grade of humidity. Some observers believe that coolness, rather than heat, is desirable. On the other hand, no climate is generally recommended in phthisis which involves much continuous bad weather, frequent high winds, excessive humidity, sunlessness.

The patient's temperament—lymphatic, neurotic, bilious, sanguine (vague as these terms are, and difficult of scientific application, there can be no doubt that they point to profound differences)—has much to say with regard to his response to climatic influence. This is even a more fundamental point than the precise state of the local lesion. We shall return to this question in connection with the various forms of climate.

The limits of these lectures will only permit a very summary treatment of the large subject of climate and phthisis. I wish to give in the briefest form the results of a considerable experience in this department. I shall treat the subject from two points of view, viz.:

(*a*) The leading types of climate, their meteorological features, advantages and disadvantages, indications and contra-indications.

(*b*) The chief types of phthisis and the indications as regards climate and locality which they afford.

There will be a certain convenience, if also some unavoidable repetition, in this twofold mode of considering the subject.

* Aristotle, 'Politics,' vii., 2-4.

The climates recommended in phthisis may be grouped under the following heads:

1. The oceanic climate, such as a patient enjoys during a long sea-voyage.

2. The marine climate — *e.g.*, Bournemouth, Ventnor, Torquay, Falmouth, St. Leonards, Rostrevor, Queenstown, Mentone, Bordighera, San Remo, Alassio, Rapallo, Jersey, Arcachon, Biarritz, Sorrento, Palermo, Malaga, Algiers, Tangier, Madeira, the Canary Islands, Southern California, Florida, Tasmania.

3. The inland climate of slight or moderate elevation — *e.g.*, Egypt, Pau, Montreux, Meran, Hammam R'Ihra, Biskra, Griqualand West, Orange River Colony, Natal, parts of New South Wales, Queensland, and New Zealand.

4. The high-altitude climate—*e.g.*, Davos, Clavadel, Arosa, St. Moritz, Pontresina, Andermatt, Maloja, Wiesen, Montana, Leysin, Arolla, Les Avants, Caux, the Andirondacks, the Andes, the Carpathians, Denver, Colorado Springs, Manitou Springs, Arizona, New Mexico.

The above classification is one of practical convenience rather than strict scientific accuracy. It must be freely admitted that the present state of our knowledge does not enable us to speak dogmatically upon the precise relation of meteorological factors and the influence of climates upon phthisis. We can, however, formulate some rules, more or less empirically, which may prove of substantial service in practice.

1. The Oceanic Climate.—The leading features of this climate are the following: Marked equability; a high average of humidity; a high degree of purity and free circulation of air; much ozone. The best sea-voyage in pulmonary cases is the all-round voyage to New Zealand, viâ the Cape of Good Hope and home by Cape Horn. It involves about twelve weeks at sea. Its chief drawback is the cold and windy weather often encountered in the high southern latitudes. No

voyage of shorter duration than the above can be expected to do much good in pulmonary cases. Well-arranged yachting cruises might sometimes be adopted with advantage.

The popularity of the sea-voyage as a remedy in phthisis has fallen off decidedly in recent years, and it is worthy of note that this change in medical practice has coincided with the effacement of the old clipper ship and the substitution of the modern steamship. That this change, however convenient from other points of view, has been a loss to the invalid, I entertain no doubt. The old-fashioned sailing-ship, with its roomy cabins, its leisurely life, absence of excitement, slower progress through different latitudes, and longer duration, was more suitable to the invalid than the crack liner with its swift speed, rapid progress through different degrees of latitude, its often crowded cabins, gayer life, and briefer duration.

The sea-voyage combines a very perfect form of physical and mental repose, continuous fresh air without fatigue, an agreeable sense of passive movement, life on a new plane. On the other hand, the ventilation of the cabins is not always good, the food is sometimes of mediocre quality, there is an element of possible boredom and ennui, and the discomforts of continuous bad weather are sometimes serious. I entertain no doubt, however, that, granting a well-found, well-ventilated ship, good cuisine, agreeable society, and moderate luck in weather, a sea-voyage is a powerful restorative in many conditions of debility, and has an important but very limited application in phthisis. The typical case for a sea-voyage is that of a young male adult (twenty to thirty-five) in whom tuberculosis is only suspected, or who has become slightly infected as the result of study, continuous indoor occupation, or the struggles of commercial or professional life; whose general health is not seriously impaired, who is fond of the sea, a good sailor, and attracted by the prospect of travel. Such patients, it may be observed, often

shrink from the tedium and rigid routine of sanatorium life, but readily acquiesce in the suggestion of a sea-voyage. The sea-voyage is also useful in the so-called 'strumous' cases, where a sluggish pulmonary lesion co-exists with affections of bones or joints.

The sea-voyage is unsuited for all cases of advanced disease and all cases with active symptoms. Pyrexia (unless very slight), marked debility, much dyspepsia, laryngeal and intestinal complications, all contra-indicate the sea-voyage. Recent hæmorrhage is also a contra-indication, but hæmorrhage of comparatively remote date need not be specially considered. A moderate degree of nervous irritability is often favourably influenced by a sea-voyage.

Some writers have laid much stress on the noises and distractions of life on shipboard, on the crash of the engines, the roar of wind and wave, the bustle of the saloon. My own recollections, based on a considerable experience of the sea, are rather of placid days, much fine weather, calm mental and physical; a life of repose and freedom from stress of any kind; a life sometimes, indeed, monotonous, but peaceful and restful. Insomnia is rarely complained of on shipboard after the first few days; sea-sickness in most cases soon abates; appetite and digestion are commonly active; weight in most cases increases. There are, however, exceptions to these rules, and sometimes it is clear that the patient has an intolerance of sea life.

Dr. Theodore Williams recommends sea-voyages in cases of chronic cavity, where the disease is unilateral and quiescent, and adds that in 'strumous' phthisis he has repeatedly seen cavities contract and a gain in weight of from one to two stones occur during a sea-voyage.* I believe this is so, but it is difficult to say how cavity cases will do on shipboard.

* C. T. Williams, Transactions of the British Congress on Tuberculosis, vol. iii., p. 7.

In the recommendation of a sea-voyage, age, sex, and the patient's tastes deserve weight. Upon the whole, the young do decidedly better at sea than the middle-aged or old, men better than women, and those who have a natural taste for the sea than those who have none. It is not well to advise a sea-voyage to a patient who has a natural dislike for the sea.

The management of the patient after landing from a sea-voyage is important. Indiscretions at this stage often undo the good obtained during the voyage. The patient should be warned that on landing he must exercise special care as regards over-fatigue, exposure, errors in eating and drinking. He must not make too abrupt a change in his habits.

2. THE MARINE CLIMATE.—Marine resorts for phthisis form a large class, and present fundamental variations as regards—(*a*) temperature, (*b*) humidity, (*c*) wind-exposure and shelter.* Most of the favourite marine resorts are characterized by warmth, moderate humidity, and a fair degree of shelter. The Rivieran resorts (San Remo, Mentone, Bordighera) represent the type of dry marine resorts. Algiers, Ajaccio, Palermo, Taormina, Corfu, Catania, Capri, represent the type of moderately moist marine resorts. Madeira, Teneriffe, Arcachon, Bournemouth, Torquay, Falmouth, represent the type of decidedly moist marine resorts. Dry marine resorts may be reckoned as 'bracing' or 'stimulating'; moist marine stations are in various degrees 'sedative' or 'relaxing.' Some stations seem to combine a tonic with a sedative influence.

The Rivieran resorts have lost something of their former popularity, in part owing to the competition of rivals—especially the mountain sanatoria—but in part, also, to their

* Dr. Gordon of Exeter has recently brought forward a good deal of evidence to show that in Devonshire exposed districts have more phthisis than sheltered districts.

inherent defects, especially as regards windiness and instability of temperature. Yet the winter in a favourable season is often very charming. Long spells of mild, sunny, gently stimulating and fairly calm weather, are not uncommon. For the consumptive, Mentone and San Remo are, perhaps, the best of the Rivieran resorts. Where quiet is desired, Bordighera may be tried. Grasse sometimes does well, but is apt to be rather cold. Where economy is a consideration, Alassio or Rapallo may be recommended. These latter resorts are moister and less stimulating than the towns farther west. Nice, Cannes, and Monte Carlo are less suitable for the invalid. Many patients get on best at places a little removed from the sea. Visitors to the Riviera must be advised that the climate demands watchfulness; that sudden changes of temperature, especially just after sundown, are common; that the combination of bright sun and cold winds is somewhat treacherous, and that clothing and habits must be arranged accordingly. It is a matter of common complaint that visitors to the Riviera do not submit themselves to medical control as much as might be desired. The atmosphere, traditions, and *milieu* of the Riviera are favourable to pleasure-seeking and enjoyment rather than to continuous, watchful, and serious treatment. The proximity of Monte Carlo acts as a disturbing influence.

According to Dr. Theodore Williams, the cases most suitable for the Riviera are the following:

(*a*) Phthisis in which inflammatory processes have played a large part in predisposing to the disease.

(*b*) Strumous phthisis.

(*c*) Laryngeal phthisis.

(*d*) Unilateral tuberculization rather than bilateral.

(*e*) The large class of consumptives who, either from extent of disease, or feebleness of circulation, or advancing years, are unable to endure the rarified atmosphere and cold of the high altitudes. Most of these patients love warmth,

and cannot take enough exercise when the thermometer is below zero to maintain it.

The Riviera is, in my experience, generally unsuitable for neurotic patients, for active disease, and for cases where dry irritable bronchitis is present. The *eretische constitution* does not do well on the Riviera.

The home resorts—Ventnor, Bournemouth, Torquay, St. Leonards, Dawlish, Sidmouth, Salcombe, Ilfracombe, Tenby, Rostrevor, Queenstown, Glengarriff—come under the class of marine stations, and suit many cases well for winter residence. They are appropriate for cases of phthisis which for various reasons—medical, financial, personal, or domestic—prefer not to go abroad, and the results obtained often compare not unfavourably with those obtained at more distant resorts. Some patients shrink from the fatigue of a long journey, some are repelled by the idea of foreign customs and a strange cuisine, others dislike separation from friends. No doubt these objections would be trivial if the advantages of foreign resorts were always striking and decisive. But this is far from being the case. Ventnor, Bournemouth, Torquay, St. Leonards, and Falmouth are good winter stations, and may be safely recommended in many cases. They are especially suitable for cases of chronic phthisis where digestive troubles are prominent, for patients who are reluctant to leave their own country, and for cases where there is more than the usual amount of uncertainty regarding the patient's prospects and progress. Advanced cases may obtain the stimulus which change of air and scene often affords, with a minimum of fatigue and risk, by selecting one of the home resorts. Where a bracing and tonic quality is desired we may select St. Leonards or Hastings; where soothing and sedative conditions seem indicated we may give the preference to Torquay or Falmouth.

The Canary Islands have a remarkably mild, equable, sunny, but somewhat damp, climate. Las Palmas in Grand

Canary is drier and more bracing than Orotava and Guimar in Teneriffe. Among the available resorts in these islands may be mentioned the following places: Orotava, La Laguna, Guimar, Santa Cruz (Teneriffe), Santa Cruz (La Palma), Las Palmas (Grand Canary). Facilities are now available in some of these islands for enabling the patient to have recourse to some of the more elevated spots on the approach of the hot season. There is much complaint of the frequency of diarrhœa and bowel affections amongst visitors, but some of my patients assure me that these troubles are mainly attributable to indiscretion in diet, especially eating excessively of fruit, or eating fruit of bad quality. My personal trial of the Canary Islands in cases of phthisis has not been large, but the results have been fairly favourable. These islands are worthy of recommendation in certain cases of phthisis where anæmia and debility are marked, where irritable bronchitis is present, or where the patient craves warmth and sunshine; also in advanced cases, where palliation of symptoms is the only object contemplated.

Madeira is, upon the whole, less desirable than the Canary Islands, but it may be tried in some cases of catarrhal phthisis, and in senile phthisis, or where emphysema or cardiac complications are present.

The Sicilian resorts — Palermo, Catania, Aci Reale, Taormina—belong to the same class as the Canary Islands, and may be tried in similar cases.

Algiers has a good winter climate, with a high average of fine weather, moderate humidity, and much sunshine. The sanitary condition is not good, and dust is often troublesome. Tangier has a somewhat similar climate, but moister, and more under the influence of winds from the Atlantic. It is not a suitable resort for consumptives. The hotels are good, but the sanitation is oriental, and there are practically no roads.

There are some good resorts upon the southern coast of California—*e.g.*, San Diego, Santa Barbara, Coronado Beach,

Catalina Island. In Northern California Monterey is the chief resort.

The coast of Florida is a favourite resort for patients from the Northern States. Jacksonville, St. Augustine, and Palm Beach are some of the best known places.

3. The Inland Climate of Low or Moderate Elevation.—The resorts enumerated under this category present many variations as regards elevation and temperature. In the main they are dry, sunny, and tonic. The marked diurnal oscillations of temperature which are found in most of these resorts do not seem to be disadvantageous, provided they occur with regularity, and can, therefore, be reckoned with. Such oscillations have a tonic influence. Egypt has great attractions for the patient of ample means who can command every luxury, and whose condition does not preclude him from travelling about and enjoying the scenery and the antiquities. The best cases for Egypt are those of chronic quiescent phthisis in persons of lymphatic or torpid constitution, or where there is much bronchial catarrh and emphysema. Egypt is, however, not well adapted for phthisis in general. In spite of the magnificent climate, and (in many places) excellent accommodation, the conditions of life and the social *milieu* are not suitable for consumptives. The Nile voyage, however charming, has a very limited utility for phthisical cases. Dust is troublesome in Egypt. The season is short, as patients cannot stay with advantage when March is past. The choice of locality lies between Cairo, Helouan, Mena House, Luxor, and Assouan. Cairo should be avoided by the consumptive; the hygiene is bad, and the social atmosphere is unsuitable for such cases. Dr. Sandwith records that 'after eighteen years of life in Egypt I have only as yet seen one case of a European living in the country becoming tubercular.'* He admits, however, that the natives suffer largely

* Transactions of the British Congress on Tuberculosis, vol. iii., p. 33.

from the disease. I should be inclined to limit the recommendation of Egypt to early or quiescent cases, especially in middle-aged patients where bronchitis or weak circulation or rheumatism is present, and in cases where the historic and antiquarian attractions of the country are likely to prove of special interest. The same remarks might apply with little qualification to the interior of Algeria. Hammam R'Ihra is a good spring resort, but not very suitable for winter. Biskra, on the edge of the Sahara, is recommended for cases where desert air and quiet are desired. The water-supply is, however, bad, and some patients find life there after a time monotonous and depressing.

South Africa has earned a reputation in phthisical cases, and I have made a somewhat extensive trial of it. The best parts are the high plateaus of the interior, especially certain districts of the Karoo, Griqualand West, Orange River Colony, the Transvaal, and Upper Natal. Ceres, eighty-four miles from Capetown, and at an elevation of 1,700 feet, is a convenient temporary stopping-place. The coast regions are, on the whole, unsuitable, but the environs of Capetown present some agreeable quarters for summer residence. Amongst the places which are recommended for phthisical cases the following may be enumerated: Cradock, Beaufort West, Matjesfontein, Graaf Reinet, Aliwal North, Bloemfontein, Bishop, Ladybrand, Harrismith, Heidelburg (Transvaal), Greytown (Natal). Many patients, however, wisely avoid the towns, and seek quarters on a farm. The mining towns are dusty, and in other respects undesirable. The climate presents many variations, but is in the main dry and sunny, with a good deal of atmospheric rarefaction, with hot summers, and cool rather than cold winters. In many parts of the interior the winter is practically rainless. Drought is often troublesome, dust-storms are frequent, and sanitation is generally defective. The accommodation is rather poor, but improving; and the food-supply, especially

as regards milk and fresh vegetables, leaves a good deal to be desired. My experience of cases sent to South Africa has been rather conflicting. I have had results beyond my expectations; I have had not a few disappointing failures. I have no doubt, however, that the climate is in the main a good one for phthisis if the patient goes well into the interior, and can secure comfortable quarters and proper food. The best type of case for South Africa is an early case of fairly quiescent phthisis in a young man who does not object to 'rough it,' who is in fair general health, and who looks forward to permanent residence in the country, if the results as regards health are found to be satisfactory. Cases with much bronchial secretion, some emphysema, rheumatism, or slight valvular mischief, may be sent to South Africa. Advanced, active, or febrile cases should on no account be sent thither; and, as living is expensive, it is indispensable that the seeker after health should be adequately provided with financial resources. The fate of poor patients with advanced disease, who go out to South Africa under the delusive impression that the climate will work miracles, is usually a deplorable one.

Australia has long had a repute in pulmonary disease, a repute which is somewhat on the wane, but which has a certain foundation. The climate is in many places a magnificent one for nine months in the year—dry, sunny, tonic, and exhilarating, with a high average of fine days, and affording exceptional opportunities for outdoor life. On the other hand, the summer season is in many places quite too hot, and the dust-storms which prevail at this season are only too likely to have a disastrous effect upon the consumptive. On the Riverine plain of New South Wales, where the climate during a large part of the year has great charm, the thermometer not uncommonly rises in summer to 105° F., 110° F., or even 120° F., in the shade. Dust-storms not infrequently destroy all vegetation, and drought

is sometimes prolonged and disastrous. It is impossible to approve of such conditions for the consumptive. Among the districts which are specially recommended for consumptives may be mentioned: Armidale (New South Wales), Beechworth (Victoria), Macedon (Victoria), Orange (New South Wales), Toowoomba (Queensland), Mount Victoria (New South Wales), Bathurst (New South Wales), Goulburn (New South Wales), and Maranoa (Queensland). The coastline of Australia is, in general, unsuitable for consumptive cases. The Australian climate is in a high degree tonic and stimulating. It is, therefore, best suited to quiescent cases and to torpid constitutions. It is unsuited to all irritable conditions, to neurotic patients, and to active disease. A patient should never be sent to Australia with a view of engaging in indoor work in one of the large Australian cities. Life on a sheep farm offers many advantages.

New Zealand has a milder, more humid, and more windy climate than Australia, but the climates of different parts present many variations. On the whole, the climate of New Zealand is not particularly well adapted to consumptive cases, though many patients undoubtedly do well there. Among the best districts are Napier and the Canterbury Plain.

The climates of some parts of Arizona belong to this class, and present great advantages, but many of the resorts in this region belong rather to the high-altitude class.

4. The High-Altitude Climate.—Sir Hermann Weber gives the following catalogue of the meteorological features of the climates of elevation exceeding 3,500 feet: Diminished density of the air; a lower degree of absolute and relative humidity of the air; absence or infrequency of mists; greater transparency of the air; greater diathermancy of the air; lower shade temperature; greater difference between sun and shade temperature; greater purity of the air; absence or rarity of microbes; more ozone; comparatively little wind

(in winter).* The conditions of life at a resort of the Davos type are a little difficult of realization by those who have no experience of them. In favourable weather the heat in the sun may be so strong as to call for sunshades, while in the shade icicles may be forming. Though the shade temperature may be many degrees below freezing-point, the cold is little felt unless there be wind. Exercise is freely taken, amusements are numerous, life is not dull. The proportion of bright, sunny days is high, though the seasons vary much in this particular, and when the weather is at its best it is absolutely perfect. For persons who can bear the cold and rarefaction of the air, and who can take sufficient exercise, the climate is in a high degree tonic and bracing, nutrition is in many cases successfully stimulated, nervous energy is promoted, the number of red corpuscles in the blood is increased,† and the expansion of the chest (with probably in many cases compensatory emphysema) is augmented.

Whether cold *per se* is desirable for consumptive cases is difficult to say. It seems probable that cold is inimical to bacillary activity, but in the case of man the question is much involved owing to the relation of cold to habits, exercise, occupation, and nutrition. If a cold climate involves confinement to the house, or interferes with the quality of the food-supply, it will be, *pro tanto*, injurious to the consumptive. It is worth recalling that Commandant Peary reports that several members of his crew in his last Arctic expedition (1902) who were slightly consumptive obtained much benefit from their stay in the Arctic regions. In all probability the patient with a fair degree of circulatory and digestive vigour, and capable of taking adequate exercise, will do better in a cold climate than in a warm one.

* H. Weber and F. P. Weber, 'The Mineral Waters and Health Resorts of Europe,' p. 352.

† Viault, Egger, Kündig, Mercier, and others.

The high-altitude climate may be tried in most cases of phthisis, provided certain well-understood contra-indications are not present—viz.:

(*a*) Circulatory weakness, whether due to organic disease or not.

(*b*) Much bronchitis and emphysema.

(*c*) Albuminuria.

(*d*) Rheumatism.

(*e*) Marked nervous irritability.

(*f*) Inability to take exercise and to withstand cold.

The best results at the high-altitude stations are obtained in young male adolescents with incipient lesions, in hæmorrhagic cases, and in cases consequent on pneumonia and pleurisy. Chronic one-sided cavity cases sometimes do well. It is said that cases of laryngeal ulceration sometimes do well, but I cannot confirm this opinion from my own experience.

No stage of the disease precludes resort to the high-altitude stations provided the constitutional state is good, but naturally the early cases do best. Some cavity cases, however, if the lesions are not extensive and the general, circulatory, digestive, and nervous condition satisfactory, do well at Davos, Arosa, or St. Moritz. A moderate degree of pyrexia is not a contra-indication, nor is hæmoptysis. Dr. Theodore Williams reports that resort to the high altitudes led to arrest of the disease in 58 per cent. of his cases; to improvement in 87 per cent.; in excavation cases arrest occurred in 21 per cent. of cases, and great improvement in 61 per cent.* Dr. Turban reports that of 408 cases treated in his sanatorium at Davos good results were obtained in 76·6 per cent. of cases.† Dr. Stephani reports that of 150 cases treated by him at Montana complete cure was obtained in 12 per cent., improvement in 50 per cent., arrest in 20 per cent., and no

* *Loc. cit.*

† 'Beiträge zur Kenntniss der Lungen-Tuberkulose,' p. 134, etc.

benefit in 18 per cent.* Sir Hermann Weber finds that 'the effect of these climates (*i.e.*, of the high altitudes) had been better than that of any other climatic group or sea-voyages.'†

I have given the high-altitude stations a prolonged trial, more especially Davos, St. Moritz, Leysin, Denver, Colorado Springs, Manitou Springs, and the highlands of South Africa. The results have been in the main satisfactory, and as good as could be hoped for from climatic treatment. The essential point is that there should be a careful selection of cases. Unsuitable cases do badly at the high altitudes, and bring discredit on the treatment.

Patients may go to the high-altitude stations at any time of the year, but the winter is the best season in Europe. There seems no foundation for the idea that special dangers attend the melting of the snow, but the spring at these stations, as elsewhere in Europe, is the least satisfactory season. A short stay at some intermediate station (*e.g.*, Ragatz, Montreux, Lugano) on leaving the high altitudes seems usually advisable. Vigilant medical supervision is especially obligatory at the high-altitude stations. The patient's habits and amusements must be strictly controlled.

I shall now select some well-known clinical types of phthisis, and enquire what can be said as regards the class of health-resort most likely to be found useful in their treatment. It must be understood that the suggestions given possess only a very limited validity, inasmuch as every case must be considered upon its merits, and personal idiosyncrasies cannot be ignored. The medical adviser must always endeavour to solve the personal equation in these cases.

TYPE I.—The common case where incipient phthisis is suspected but positive proof is lacking. Slight cough, perhaps an occasional trace of blood in the expectoration, slight loss

* Transactions of the British Congress on Tuberculosis, vol. iii., p. 80.

† *Ibid.*, p. 22.

of flesh, some degree of languor and debility—such is a common group of symptoms; there are no physical signs in the chest, and sputum is either absent or bacilli are wanting.

Probably many of these cases are really tubercular, but we may not be able to advance beyond the point of suspicion. Much judgment is required to avoid the opposite errors of premature panic and disastrous procrastination. It is a question whether these cases should be sent to a sanatorium. They are usually most reluctant to go. Constant association with more or less advanced cases of phthisis is of doubtful propriety when a doubt rests upon the diagnosis, and the psychical effect of sending mere 'suspicious' cases to a sanatorium is not free from objection. On the other hand, such patients readily welcome, as a rule, the suggestion of climatic change. Often their condition is due to over-pressure of some sort, and the release from arduous duties or the escape from an unfavourable environment may be an essential element in the treatment.

The choice of climate in such cases is obviously rather wide. A long sea-voyage often answers well; so does a winter in the Alps. The Riviera, Egypt, the Canary Islands, or the southern English coast, may in various cases be selected. It is essential that the patient's mode of life be carefully regulated.

TYPE II.—A case similar to the foregoing, but with definite though slight signs in the lungs or bacilli in the sputum.

Sanatorium treatment should be preferred, in most instances, for these cases. Its educative influence is particularly valuable. If, for any reason, sanatorium treatment is not adopted, the choice for these cases will usually lie between the high-altitude stations, the Riviera, South Africa, or Colorado, but the home resorts may often be selected with advantage. It is important not to send an incipient case on a distant journey until we have grounds for believing that there is no danger of the case running an acute or subacute course.

TYPE III.—A case where phthisis sets in suddenly with more or less profuse hæmorrhage in the midst of apparently good health; the physical signs are slight, and the constitutional state good.

A period of complete rest and treatment at home will first be necessary. These cases often do very well at the high altitudes. I have seen excellent results from sea-voyages. A preliminary course of sanatorium treatment is usually advisable.

TYPE IV.—A case where phthisis has supervened upon pleurisy or broncho-pneumonia, and where 'inflammatory' conditions may be supposed to have played a considerable part.

These cases do well at the high altitudes and on the Riviera. The state of the circulatory, digestive, and nervous systems will determine the choice between these two types of climate.

TYPE V.—A case of phthisis which has set in with active catarrhal symptoms. This type of case should be regarded with caution, as well-marked catarrh may point to widespread tuberculization.

These cases do not get on well in the mountains or on shipboard. They do better on the south coast of England, in the Riviera, the Canary Islands, Madeira, or Egypt. If we judge that, owing either to the bronchial or nervous condition, the case requires stimulation, the Riviera or Egypt may be selected; if a sedative effect seems preferable, we may select the Canary Islands or Madeira.

TYPE VI.—A case presenting laryngeal complications, irritative or ulcerative.

Many of these cases should be kept at home. Irritable laryngitis sometimes does well at Bournemouth or in the Canary Islands. Laryngeal ulceration is always unpromising. The Riviera may be tried. Some authorities advise a trial of the high altitudes.

TYPE VII.—A case in which albuminuria, rheumatism, or heart disease is present as a complication.

A dry, warm climate, such as the Riviera, Egypt, or the interior of Algeria, should be chosen. The home resorts, the long sea-voyage, and the high-altitude stations are all alike unsuitable.

TYPE VIII.—A case where excavation has taken place at one apex, but the disease is limited and the general condition fair.

These cases do fairly well in many climates, but rarely recover completely. The high-altitude resorts are best, if no contra-indications exist, but the inland stations, the long sea-voyage, or the home resorts, may sometimes be recommended.

TYPE IX.—A case of extensive lesion and steadily progressive disease, with more or less active symptoms.

These cases should usually be kept at home. If a change be thought advisable, one of the English stations may be chosen, or Madeira or the Canary Islands may be advised with a view to the palliation of symptoms. The friends of the patient should be informed that change of climate is a measure of doubtful expediency, and that not much is to be expected from it.

TYPE X.—A case of so-called 'strumous' phthisis—*i.e.*, where disease of bones, joints, or glands is present in addition to pulmonary lesions.

A bracing marine resort is, in general, best for these cases —*e.g.*, Hastings, Margate, San Remo. The long sea-voyage is sometimes useful.

TYPE XI.—A case of limited disease where dyspeptic symptoms are prominent, or where diarrhœa is present.

These cases often do best at one of the home stations.

TYPE XII.—A case where phthisis has supervened upon typhoid fever, influenza, or measles.

If the case be recent, and it is still doubtful whether its course is going to be subacute or chronic, it should be kept

at home, or may be sent to one of the southern English resorts. If it is clear that the case is likely to pursue a chronic and fairly favourable course, it may be sent to the high-altitude resorts, or one of the dry marine resorts, or the inland plains. Prognosis should be very guarded.

Speaking generally, the main guides to the selection of climate in phthisis are the activity of the morbid process and the constitutional condition of the patient. The stage of the disease is less important, but the extent of the disease is a consideration of weight, inasmuch as the more extensive the lesion the less is the patient likely to react to tonic climates. Often our most difficult task is to decide whether climatic change should be tried at all or not. If this question be decided in the affirmative, we have to determine whether our aim is the arrest and cure, or only the palliation, of the disease. Finally, we must consider whether the patient is likely to respond to an actively stimulating, moderately stimulating, slightly sedative, or decidedly sedative, climate. Our selection of locality will be largely governed by the answer which we give to these questions.

There is no necessary conflict between sanatorium treatment and climatic treatment. The two methods may be successfully combined, as in the well-known sanatoria at Davos, Arosa, Leysin, Montana, and elsewhere. A preliminary course of sanatorium treatment is often most advisable before change of climate is adopted.

Dr. Burney Yeo says most truly, 'What the consumptive patient most needs is a combination of climate and sanatorium treatment, for the patient, if left to his own devices, may make a bad use of a good climate, while with skilful guidance in a sanatorium he may make a good use of a bad one. Care without climate is better than climate without care.'*

* J. Burney Yeo, Transactions of the British Congress on Tuberculosis, vol. iii., p. 20.

LECTURE XXI

THE CAUSES AND MANAGEMENT OF HÆMOPTYSIS

SUMMARY:

Causes of hæmoptysis:

1. Pulmonary tuberculosis.
2. Valvular disease of the heart.
3. Pulmonary conditions other than tuberculosis—*e.g.*, infarction, capillary bronchitis, plastic bronchitis, pneumonia, abscess, gangrene, bronchiectasis, malignant disease, degeneration of pulmonary vessels, actinomycosis, hydatids.
4. Certain blood conditions—*e.g.*, scurvy, hæmophilia, leukæmia, pernicious anæmia, purpura, malignant types of the specific fevers.
5. Aortic aneurysm.
6. Epithelioma of the œsophagus.
7. Vaso-motor disturbance—*e.g.*, hysteria.
8. The endemic hæmoptysis of China and Japan.
9. Traumatism.

General problems involved in a case of hæmoptysis.

Question of 'arthritic' hæmoptysis.

Hæmoptysis in heart disease.

Hæmoptysis in pulmonary tuberculosis: its characters, dangers, differential diagnosis, and treatment.

Hæmoptysis is a frequent and important symptom which will repay careful study. The patient commonly exaggerates its dangers; the physician rightly attaches much weight to its diagnostic significance.

The causes of hæmoptysis may be enumerated as follows:

1. Pulmonary tuberculosis, the immediate cause in such cases being: (*a*) Hyperæmia of the bronchial mucous membrane; (*b*) ulceration of a bronchiole; or (*c*) rupture of an aneurysm of the pulmonary artery or of one of its branches.

2. Valvular diseases of the heart.

3. Pulmonary conditions other than tuberculosis—viz., infarction; capillary bronchitis; plastic bronchitis; pneumonia; abscess; gangrene; bronchiectasis; malignant disease; degeneration of the pulmonary vessels, as in Bright's disease and the so-called 'arthritic' hæmoptysis; actinomycosis; hydatids.

4. Certain blood conditions—viz., scurvy, hæmophilia, leukæmia, pernicious anæmia, purpura, malignant types of the specific fevers.

5. Aortic aneurysm.

6. Epithelioma of the œsophagus.

7. Vaso-motor disturbance—*e.g.*, hysteria.

8. The endemic hæmoptysis of Japan and Korea, depending on the *distomum pulmonale*.

9. Traumatism.

Whether a true 'vicarious' hæmoptysis occurs—*e.g.*, in patients suffering from amenorrhœa—is doubtful. Most writers affirm its existence.

We shall first consider the general diagnostic problem usually involved in a case of hæmoptysis, and then enter into the examination of details.

There is a definite history of hæmoptysis—How shall we proceed to discover its cause? The possibility that the blood came originally from the stomach, and has thence found its way into the air passages may, as a rule, be readily excluded. In gastric hæmorrhage the blood is generally in considerable quantity, dark, or even black in colour, acid in reaction; there is a history of pain in the stomach and previous vomiting;

portions of food may be found in the blood; there is no cough, and physical signs in the chest are wanting.

Often the cause of hæmoptysis will be self-evident. The patient may present clear signs of phthisis, mitral disease, scurvy, or one of the severe anæmias. Such causes as pulmonary infarction, capillary bronchitis, pneumonia, abscess, gangrene, will usually be evident from the history and other symptoms. Aortic aneurysm and bronchiectasis have their own clinical picture, and will not often give rise to serious difficulty. The possibility of phthisis being present as a complication of some other condition must always be borne in mind.

In a case of hæmoptysis without evident cause or typical history, we may say, with a high degree of probability, that the patient has either phthisis, heart disease, or some blood dyscrasia. As regards the frequent statement of patients, 'I had an attack of spitting of blood, but the doctor said it came from the throat,' it may be laid down with confidence that such a statement is hardly ever well founded. It generally means that no physical signs being discovered in the lungs or heart, and no evident source for the bleeding being recognisable, it has been assumed that the blood came from the throat. Now, I think it may be affirmed that free bleeding from the throat is not common in any condition. It may occur in advanced laryngeal ulceration, but such cases are usually clear. There is generally a definite history of tuberculosis, a poor constitutional state, hoarseness or aphonia, in some cases a certain degree of dysphagia. Again, blood may come from the naso-pharynx in a variety of conditions, but such cases rarely involve any serious difficulty. The pharynx or nasal passages may be seen to be congested or ulcerated, the blood may come in part from the nose, and if we have an opportunity of examining it we shall probably find it dark in colour, watery, and mixed with saliva or nasal mucus. We may lay down the following working rule:

If the nasal passages and pharynx are fairly healthy in appearance, if no marked congestion or ulceration is present, if no varicose veins can be seen, then it is highly improbable that the seat of the bleeding is in those regions.

The hæmoptysis of heart disease is not, as a rule, difficult of detection. It is not at all a common symptom in early cases with good compensation. It occurs chiefly in mitral disease, especially stenosis, and generally in cases where the signs of heart disease and of passive congestion are gross and evident, so that its recognition is commonly a simple matter.

If, then, no obvious explanation of an attack of hæmoptysis is forthcoming, if we can exclude heart disease, the blood dyscrasias, and other more or less obvious conditions, What is the probability that pulmonary tubercle is the cause of the hæmoptysis? That probability is, in my judgment, a high one. Nearly all apparently uncaused attacks of hæmoptysis, nearly all attacks where no other obvious morbid state co-exists, are in all probability due to pulmonary tuberculosis. This may seem a somewhat sweeping statement, but I believe it will bear the test of experience. No doubt many of these cases get well and never develop the clinical symptoms of phthisis, but then we have to bear in mind that evidence of healed tuberculosis is found in from 10 to 20 per cent. of the lungs of persons dying of all causes. We must, of course, make sure that the alleged attack of hæmoptysis was a genuine one, not a mere streak of blood in the sputum occurring after violent coughing, to which little weight can be attached, or an attack of bleeding from the gums, pharynx, œsophagus or stomach. I believe we can generally exclude these sources of fallacy by attention to the story of any intelligent patient. In genuine hæmoptysis the blood is usually coughed up; there is a history of cough preceding the hæmorrhage; the blood is more or less mixed with air;

its colour varies much, and is not to be relied upon, but it is often bright and 'arterial'; the amount of blood lost varies within the widest limits; the bleeding, if at all free, does not stop abruptly, but a smart hæmorrhage is followed by more or less blood-spitting for hours or days; some rise of temperature often follows the hæmorrhage. Of these various points, perhaps the one which gives us most assistance in diagnosis is the persistence of slight blood-spitting after a smart hæmorrhage. If a patient informs us that upon a certain date he had a sharp hæmorrhage, accompanied by cough, and followed by a gradually diminishing amount of blood for hours or days, we may conclude with confidence that the blood came from the lungs; and if heart disease, blood dyscrasias, and other more or less obvious morbid states can be excluded, we shall not be often wrong in concluding that pulmonary tuberculosis is present.

Are there any cases of recurring attacks of hæmoptysis in persons otherwise healthy where the attacks are really of no importance? Such cases are described by most writers upon the lungs, and it is undeniable that some persons suffer from periodic attacks of hæmoptysis, yet remain in good health, and never exhibit any definite pulmonary signs. The late Sir Andrew Clark believed that he could recognise a variety of hæmoptysis occurring in persons of the 'arthritic diathesis,' and not due to any definite pulmonary lesion. I doubt the correctness of this view. That sound and healthy lungs ever bleed recurrently is, I think, a most doubtful proposition. If these cases be followed to their termination, it will, I believe, be found that some are tubercular, some have heart disease, and others are the subjects of vascular degeneration and renal disease. It is dangerous in practice ever to make light of hæmoptysis, or to assume that it may be due to a constitutional peculiarity involving no serious issues.

Let us now study in some detail hæmoptysis due to tubercular disease of the lungs.

(*a*) As regards its frequency. Hæmoptysis occurs at some stage of the disease in considerably more than half the total number of cases of phthisis. The more carefully we inquire into the matter, the more frequent will this symptom prove to be. Often, no doubt, the amount of blood may be insignificant, and the occurrence of hæmoptysis may be forgotten by the patient. Wilson Fox found hæmoptysis in 161 out of 289 cases, Turban found it in 235 out of 408 cases, Walshe in 80 per cent. of cases, Brehmer in 66 per cent.

(*b*) As regards the cases in which hæmoptysis occurs. Hæmoptysis is quite rare in children, and continues infrequent until puberty. It is common in adolescence and early adult life, and again becomes infrequent in later life. Sex and family history do not appear to me to have any influence. Hæmoptysis seems to occur indifferently in patients of all constitutional types.

(*c*) As regards the stage of the disease when hæmoptysis occurs. Hæmoptysis may occur at any stage of phthisis. It is often alleged to be the first symptom, but if these cases are closely investigated it will usually be found that some cough or impairment of the general health preceded the hæmorrhage. Walshe found hæmoptysis to be the first symptom in 36 per cent. of cases. Laennec says: 'The first alarming symptom and the one calculated to draw attention to the malady with the majority of phthisical patients is an attack of hæmoptysis.'* Both these statements require some qualification. My own experience is more in accord with the statistics of Turban, who found hæmoptysis as the initial symptom in 47 out of 408 cases.† I feel sure, however, that in some of these cases slight but

* Laennec, 'Auscultation Médiate,' tome ii., p. 117.

† K. Turban, *op. cit.*

significant symptoms had preceded the occurrence of hæmoptysis.

Hæmoptysis may occur at any time in the course of phthisis, and may be the final symptom. It is commoner in subacute and chronic, than in acute, cases.

(*d*) As regards the amount of blood lost. This varies within the widest limits. There may be only a few spits of blood, or several pints may be lost. It might have been expected that early hæmorrhages would usually be small, late hæmorrhages large. This is common enough; but, on the other hand, the first hæmorrhage may be profuse, the latest may be scanty.

(*e*) As regards the precise pulmonary condition which is the physical cause of the hæmorrhage. A good deal of obscurity hangs over this subject. Hæmoptysis may be due to local hyperæmia of vessels, to rupture of capillaries, to ulceration of a vessel in the progress of softening and excavation, to rupture of a pulmonary aneurysm. Profuse bleedings are generally due to the last-mentioned cause. It is often impossible to determine which of the above conditions is present in any individual case.

(*f*) As regards the appearance of the blood and its chemical and microscopical characters. In a typical case of hæmoptysis the appearance of the blood is very characteristic. It is bright red in colour, frothy, and has a 'churned-up' appearance. Departures from these typical characters are, however, common. The blood may be dark and clotted, not mixed with air but rather with saliva; it may be salmon-coloured. These characters depend upon the seat of the bleeding, its rapidity, and the length of time which the blood has lain in the air passages. Blood may be effused into a cavity, remain there sufficiently long to become dark and clotted, and then be ejected in this state. Or blood may remain for a time in the bronchial tubes, or in the larynx or pharynx, and become more or less intimately mixed with the secre-

tions of those parts. Dried nodules of blood are sometimes ejected, but these are commoner in mitral disease than in phthisis. The blood in phthisical hæmoptysis sometimes contains tubercle bacilli, more often not.

(*g*) As regards the existence of a type of 'hæmorrhagic phthisis.' The division of cases of phthisis into a hæmorrhagic and a non-hæmorrhagic group would be an artificial one. The fact of hæmoptysis does not enable us to draw any definite inferences as regards the type of the disease, its probable course, or its special dangers. The term 'hæmorrhagic phthisis' is sometimes employed in a sense which has a certain limited degree of utility—viz., as applied to those cases where the principal feature is the occurrence of profuse hæmorrhages at long intervals, physical signs being slight or absent, and the constitutional state good. This is a well-known type, with a relatively favourable prognosis; but there is a certain awkwardness in labelling these cases hæmorrhagic phthisis, inasmuch as they form only a small proportion of the total number of cases in which hæmoptysis is present.

(*h*) As to the dangers of hæmoptysis in phthisis, and its prognosis. The dangers of hæmoptysis are either immediate or remote. Only 1 or 2 per cent. of phthisical patients die from the direct effects of hæmorrhage. The immediate danger is rarely urgent, except in cases of advanced disease and marked debility. Patients with early and limited disease hardly ever die of hæmoptysis. Death from hæmoptysis may be due to shock, suffocation, cerebral anæmia, syncope, or exhaustion. Shock is generally nervous in character, and rarely fatal. Suffocation may ensue from blocking of the air passages with blood. This is probably one of the commoner causes of death from hæmoptysis, and should always be remembered, inasmuch as it demands prompt and energetic treatment on quite different lines from those usually suitable for hæmoptysis. Death from cerebral anæmia and from

syncope is probably commoner than death from exhaustion. The remote dangers from hæmoptysis are chiefly the establishment of a septic broncho-pneumonia, with the development of fresh foci of tuberculization in the lungs, and the transformation of a chronic into an acute or subacute case. There is no definite prognosis in the hæmoptysis of phthisis. Its occurrence does not stamp the case as either better or worse than the average. Everything depends on the amount of blood lost, the frequency with which the hæmorrhage recurs, the effect upon the patient, and the state of the lungs after the hæmorrhage has subsided.

We have already considered in its broad outlines the question of diagnosis in connection with hæmoptysis. We shall now proceed to consider the differential diagnosis a little more in detail.

The distinction of hæmoptysis from hæmatemesis is usually easy. In the latter condition the blood is often dark or tarry in appearance; the reaction is acid; the blood is often mixed with food; it is vomited rather than coughed up; the vomit has often the well-known 'coffee-ground' character; the hæmorrhage is often preceded by gastric pain or other dyspeptic symptoms; the bleeding usually ceases at once, but may recur; the motions may be black. There is no cough, no persistent spitting of blood, and no rise of temperature; there are no signs in the lungs. Pain and tenderness in the epigastrium are usually present.

In hæmoptysis, on the other hand, the blood is often bright red in colour; alkaline in reaction; mixed with bronchial mucus or saliva; coughed up rather than vomited; preceded usually by cough and other pulmonary symptoms; does not cease immediately; and is often followed by a temporary fall and subsequent rise of temperature. There is no history of painful dyspepsia; no pain or tenderness in the epigastrium; no tarry motions; while there may be physical

signs in the chest. Tubercle bacilli are not usually present in the blood.

Difficulties in the differential diagnosis of hæmoptysis and hæmatemesis may arise in either of two ways—viz.:

(*a*) Blood from the lungs may be swallowed, undergo certain changes in the stomach, and then be vomited; or

(*b*) Blood from the stomach may find its way into the air passages and then be coughed up.

The history will usually distinguish these cases.

The distinction of genuine hæmoptysis from hæmorrhage from the upper air passages—pharynx, larynx, or bronchi—may be difficult. It is the first problem which should engage our attention in the differential diagnosis of hæmoptysis. In bleeding from the upper air passages the hæmorrhage is rarely profuse; often the sputum is only 'streaked' with blood, which is mixed with saliva or bronchial mucus; in some cases the seat of the hæmorrhage can be seen. Fallacy may creep in from the circumstance that blood from the mouth, nose, or pharynx may flow into the lungs during sleep. In all cases the gums and naso-pharynx should be carefully examined. Hysterical girls, intent on exciting sympathy, often suck their gums, and thus seek to add to the dignity of their sufferings by a specious show of hæmoptysis. Genuine hæmoptysis, depending on vaso-motor causes, is said sometimes to occur in hysteria.

Hæmoptysis from cardiac, usually mitral, disease does not often cause difficulty in the differential diagnosis. It is not uncommon, though rarer, I think, than might have been expected. It is commoner in mitral than in aortic disease, commoner in mitral stenosis than in mitral regurgitation, though not rare in this latter condition. It is generally found in cardiac cases where the other symptoms are prominent, but I have known hæmoptysis to recur persistently over a long series of years in a case of combined aortic and mitral disease, the patient meanwhile remaining fairly

well, continuing at work, and having hardly any other symptom except occasional attacks of vertigo. Such cases are rare. Hæmoptysis from cardiac cause is not often profuse; sometimes the blood consists chiefly of small pellets of clot, which sink in water. Shock is not usually marked, and not infrequently the patient professes himself relieved by the bleeding.

Hæmoptysis from certain blood dyscrasias—*e.g.*, leukæmia, purpura, scurvy, hæmophilia, malignant infective fevers—is rarely profuse, and the causal condition is usually obvious. Hæmoptysis in my experience has been rare, both in pernicious anæmia and in chlorosis.

Profuse hæmorrhage, the result of the rupture of an aneurysm, is usually obvious, and need not detain us. More important, from our present point of view, are those cases of repeated small hæmorrhages believed to be due to 'weeping of the sac' into the trachea or one of the large bronchi. These cases may simulate phthisis, and if it happen that the aneurysmal sac compresses the lung the simulation may be close. Often, however, the problem of differential diagnosis is not difficult. The subjects of aneurysm are commonly middle-aged men of robust physique. There is a history of syphilis, alcoholism or strain; deep-seated thoracic pain and breathlessness will probably be present; and the patient's arteries are atheromatous. The clinical picture is thus in most respects quite different from that of phthisis. Aneurysm may also cause hæmoptysis by pressure on, and rupture of, a pulmonary vessel.

Hæmoptysis occasionally occurs in asthma. In such cases we have to inquire whether it is due to violent coughing, to ulceration of the bronchi, or to a quiescent tubercular lesion, accompanied by bronchiectasis. An examination of the sputum may help to distinguish these conditions.

Pulmonary infarction is an important, though rare, cause of hæmoptysis. The presence of cardiac disease, the sudden

onset, and the detection of an area of consolidation in one of the lungs, will serve to clear up these cases.

Hæmoptysis may be a feature of emphysema, where it is probably due to degeneration of the pulmonary vessels; of plastic bronchitis, where an examination of the sputum will usually obviate any difficulty; of pulmonary gangrene, where the history and the fœtor of the sputum make diagnosis easy; of hydatids, actinomyces, or the presence of the *distomum pulmonale*, where in most cases a diagnosis is only possible by the detection of the special parasite present.

Bronchiectasis is an important cause of hæmoptysis. The diagnosis of this condition from phthisis rests upon the history; the evacuation at long intervals of large quantities of foul-smelling sputum, which does not contain tubercle bacilli; the situation of the physical signs; and the general progress of the case.

Hæmoptysis is much less common in chronic renal disease than might have been expected. It is rarely a source of danger. Hæmoptysis is common, but not invariable, in malignant disease of the lungs. In these cases it sometimes assumes the 'red-currant jelly' type, but may not present any characteristic feature.

TREATMENT OF HÆMOPTYSIS.—I shall first deal in some detail with the treatment of hæmoptysis depending on pulmonary tuberculosis. The other cases can then be dealt with somewhat summarily.

In any ordinary case of slight or moderate hæmoptysis occurring at an early stage of phthisis, active treatment is unnecessary, and usually does harm. The routine administration of internal astringents is a most pernicious practice. In the great majority of such cases the bleeding tends to subside spontaneously, and our task is simply to place the patient under the most favourable conditions for its arrest, to calm his fears, and to endeavour to prevent a recurrence of the hæmorrhage. We must not forget that a small bleeding

may be succeeded by a more profuse attack. The patient should be put to bed in a well-aired room at a moderate temperature, with the shoulders moderately raised. The bed-clothes should be light, but sufficient for comfortable warmth. Absolute quiet and abstinence from conversation should be enforced, and little or no food should be given for several hours. A hypodermic injection of morphia and a saline aperient should be administered. In many cases no further measures are necessary. Our chief task at this stage is often to reassure the patient, and prevent panic and fussy interference on the part of friends. In cases of slight bleeding where the hæmorrhage shows a tendency to speedy subsidence, it is unnecessary to prolong the period of rest, silence, and abstinence. The patient may safely be allowed to resume his former routine in a few days, provided there is no rise of temperature or other untoward symptom.

Where the bleeding is more profuse and persistent, a somewhat different line of treatment will be advisable. Rest, silence, and abstinence must be strictly enjoined. Care should be taken to obviate passive congestion of the lungs. The bowels must be kept open by some simple aperient, care being taken to avoid diarrhœa, which disturbs the patient. Opium is the best internal remedy, and, when necessary, may be pushed freely. Patients suffering from hæmorrhage bear opium well, and ½ to 1 grain may be given safely every three or four hours for several doses if the bleeding is severe and persistent. Next to opium I should be disposed to put turpentine, which has sometimes succeeded well in my hands. It is best given by the mouth. Ergot is lauded by some authorities, while others think it inert or even injurious. Calcium chloride is a favourite remedy at present. Acetate of lead is worthy of an occasional trial. Astringent inhalations should be avoided; they involve an abrogation of the cardinal rule of giving as much physiological rest as possible to the affected part.

Digitalis seems sometimes valuable. An ice-bag may be applied over the præcordium ; this helps to quiet the heart, and it does not seem to be of much moment whether it is applied over the seat of the hæmorrhage or not. Ice by the mouth has probably little effect beyond its psychical influence.

An extreme degree of syncope or cerebral anæmia may compel us to resort to various methods of stimulation. These should be avoided unless there is evidently urgent danger. It must be remembered that a slight degree of syncope is favourable to clotting of the blood, and hence desirable.

It must be insisted that in cases of moderate hæmoptysis in early phthisis the free exhibition of internal astringents is thoroughly bad practice ; they lock up the secretions, and aggravate the after-effects of the hæmorrhage. We should bear in mind that while the patient thinks most of his hæmorrhage, the physician should think most of his condition after the hæmorrhage.

What are we to do when the hæmorrhage is alarmingly profuse and persistent ? Much depends upon whether we have reason to believe that the disease in the lungs is early and limited in extent, or that there is extensive excavation. In the former case the prospects of the speedy cessation of the hæmorrhage are good, and the treatment already outlined may be adopted, perhaps somewhat more actively, but essentially on the same lines. If profuse hæmorrhage occurs in connection with extensive excavation of the lungs, the danger is serious, although death from the direct effects of the bleeding is unusual. In such cases rupture of an aneurysm in the wall of a cavity is to be surmised, and this accident is but little amenable to treatment. Opium is our best resource, and may be given freely. Nauseating doses of ipecacuanha, recommended by Trousseau ; aconite, praised by Andrew ; the artificial induction of pneumothorax, advised

by Cayley; venesection, suggested by many authorities, may be enumerated as possible resources in these dire extremities. I have no experience of these methods. Good results have been reported from the use of nitrite of amyl. Slow-acting astringents are obviously futile in the circumstances. It must not be forgotten that the patient's most urgent danger may be death from asphyxia, owing to blocking of the air passages with blood. In such cases, all other considerations must yield to the urgent necessity of averting the imminent danger of suffocation. Active stimulation must be employed. As Dr. Sutton used to say to his class at the London Hospital, 'Tell the patient to cough it up.' Transfusion may sometimes be necessary.

The after-treatment of hæmoptysis in phthisis is highly important, and is, perhaps, not sufficiently considered. We find several types of case:

(*a*) The patient may profess himself relieved by the hæmoptysis; the sense of 'tightness' in the chest is lessened, and the breathing is easier. These cases do not call for any special comment.

(*b*) The hæmorrhage may subside in a few hours or in a day or two, and leave the patient's condition unaltered. In such cases rest and light diet should be advised for a few days, and the use of cod-liver-oil or other nutrients and tonics should be temporarily suspended.

(*c*) The hæmorrhage may be followed by a septic bronchopneumonia; the temperature rises and becomes hectic; new physical signs appear in the lungs; the general clinical state is worsened. These cases call for much watchfulness. Rest, light diet, and a mild antiphlogistic regimen may be tried for a time, but lowering measures should be cautiously pursued. As soon as possible we should again resume tonic lines of treatment.

(*d*) The hæmorrhage may be followed by the development of fresh foci of tuberculization in the lungs; the case passes

from a chronic into a subacute condition. Rest, fresh air, cold or tepid sponging, a fairly generous diet, and a carefully regulated hygiene are indicated.

The treatment of hæmoptysis depending on causes other than phthisis need not detain us. In cardiac cases rest and purgation are the chief points. The use of digitalis need not be suspended. Astringents are hurtful. Hæmoptysis depending on blood conditions seldom requires active interference. If treatment seems called for, it may follow the lines suitable for moderate hæmorrhage in early phthisis. Hæmoptysis is rarely the leading feature of hæmophilia. Where it requires treatment in this condition, turpentine and perchloride of iron are the best remedies. In pneumonia, hæmoptysis does not call for any special treatment.

LECTURE XXII

SOME OF THE RARER FORMS OF PULMONARY DISEASE

SUMMARY:

Malignant disease of the lungs:

Analysis of the history, symptoms, physical signs, and result in seven cases.

Differential diagnosis of pulmonary malignancy.

Distinction of the disease from pulmonary tuberculosis.

Abscess of the lungs:

Causes of pulmonary abscess.

Diagnosis, prognosis, and treatment.

Gangrene of the lungs:

Causes, symptoms, sputum, complications.

Differential diagnosis, prognosis, and treatment.

Actinomycosis of the lungs:

Causes, pathology, signs, and symptoms.

Differential diagnosis, prognosis, and treatment.

Hydatids of the lungs:

Distribution of the disease.

Symptoms and physical signs.

Differential diagnosis, prognosis, and treatment.

Syphilis of the lungs:

Two forms of syphilitic lungs—viz., a gummatous form and a chronic interstitial indurative pneumonia.

Distribution of the lesions.

Symptoms and physical signs.

Differential diagnosis and treatment.

Malignant Disease of the Lungs.

Malignant disease of the lungs, though not extremely rare, is a sufficiently uncommon condition to make its diagnosis a matter of more than ordinary interest. In the earlier stages of the malady the diagnosis involves considerable difficulty, the physical signs being variable within wide limits, while the symptoms are not always characteristic. An affirmative diagnosis being equivalent to a death sentence, the practitioner is naturally reluctant to suggest the possibility of pulmonary malignancy; but, on the other hand, the possibility is one which should be faced as promptly as possible, in order that we may not hold out futile hopes or persist in abortive treatment.

I have notes of seven cases of pulmonary malignancy, and perhaps I cannot do better than depart somewhat from the general plan of these lectures, and simply give a summary of the facts which these cases bring to light. I propose to analyze these cases from the following points of view:

(*a*) Previous history of the case.
(*b*) Symptoms.
(*c*) Physical signs.
(*d*) Nature of the growth.
(*e*) Duration of the case.

(*a*) Previous History. — This is a point of the first importance, and often gives the earliest clue to diagnosis. In the great majority of cases of pulmonary malignancy the growth is secondary to tumour elsewhere. In two of my cases there had been excision of the mamma for scirrhus— in one case six weeks, in the other two years, before the advent of pulmonary symptoms. In a third case the patient had had his leg amputated for sarcoma five years before coming under my observation, and in two other cases the disease began in the glands of the mediastinum. In the

remaining two cases of my series of seven no primary growth could be discovered. We may take it as a point of the first importance to get a clue to a previous growth in a case where the symptoms and physical signs suggest the possibility of pulmonary malignancy.

(*b*) Symptoms.—As a rule, it is the symptoms which first attract attention, and which give us the greatest help in making a diagnosis. Persistent thoracic pain, gradually increasing dyspnœa, and cough were present in all my cases. This symptom-complex is highly suspicious in a patient who has had a growth elsewhere than in the lungs, especially if the possibility of aneurysm can be excluded. The prominence of pain and the increasing urgency of the dyspnœa are specially important, because these symptoms are, upon the whole, improbable in the conditions—*e.g.*, phthisis—most likely to be confounded with malignant disease of the lungs. Hæmoptysis was present in five of my cases, absent in two. Some observers believe that the hæmorrhage often has a red-currant-jelly-like appearance; others—*e.g.*, Stokes—describe it as dark and mucoid. My own experience would lead me to doubt if any rule can be laid down on this point. In several of my cases the hæmoptysis was not superficially distinguishable from that of phthisis. Fragments of cancerous material are only rarely detected. In one case the hæmorrhage was copious and uncontrollable. Pyrexia was also present in five out of my seven cases, and was somewhat hectic in character. Night-sweating was prominent in two cases, and emaciation was marked in four. Pulmonary malignancy may progress so rapidly that there is hardly time for much emaciation to develop. In the last case which has come under my notice the patient remained plump and well nourished until near the end. It is fallacious, therefore, to exclude pulmonary malignancy because there is little or no definite cachexia.

(*c*) Physical Signs.—These are extremely variable and

not at all to be relied upon in the earlier stages of the disease, while at an advanced stage they become pronounced and easily interpreted. In one of my cases the disease first showed itself by rhonchi and mucous râles in the right mammary region, which were suspicious from their localization and from the fact that the lady in question had had the right mamma removed six weeks previously for scirrhus, and because tuberculosis was on various grounds improbable. Râles of various kinds may occur; a pleuritic rub is not uncommon; but when the growth has become well developed we generally find the following group of signs: Dulness on percussion, usually pronounced; weak breathing; diminished vocal fremitus. Weak, rather than bronchial, breathing has been present in most of the cases under my observation, the inference being that the growth usually compresses one of the main bronchi. The localization of the signs presents many variations, the mid-region of the lungs being often involved. Some writers think that the right apex is a frequent seat of the disease. The dissemination of the growth does not follow any of the laws characteristic of the spread of tubercular infiltration. Bulging of the ribs and sternum is frequent in pulmonary malignancy, but it is a late symptom.

In addition to the physical signs directly due to the presence of a tumour, it is common in these cases to get evidences of pressure by enlarged glands or veins, nerves or bronchi; and signs of pleural effusion at the base posteriorly are frequent. The effusion is sometimes hæmorrhagic, but often serous.

(*d*) NATURE OF THE GROWTH.—An autopsy was performed in four of my cases: one example of each of the following growths was found—viz., encephaloid carcinoma, lympho-sarcoma, squamous-celled carcinoma, while in the fourth case the nature of the growth was doubtful.

(*e*) DURATION OF THE CASE.—The following was the

probable duration in the various cases: Case 1, four months; Case 2, eight months; Case 3, three months; Case 4, five months; Case 5, four months; Case 6, doubtful but brief; Case 7, doubtful but prolonged.

The above cases, so far as any conclusions can be based on a somewhat limited number, suggest the following observations:

The history is a matter of the first importance in investigating any case of suspected pulmonary malignancy. Fortunately, from the nature of the case, the history is usually clear. We must be on our guard against what is, perhaps, a somewhat natural error—viz., of too confidently diagnosing a growth in the lung in the presence of certain signs and symptoms, because the history seems to make the existence of such a growth probable.

The symptoms are fairly consistent, and, on the whole, to be relied on, especially in the latter stages of the affection, but at the outset they may be vague and inconclusive. The prominence of pain in the chest with oppression of the breathing is very significant in any case in which, on other grounds, the presence of a pulmonary new growth might be suspected. Aortic disease, which might give rise to similar symptoms, can usually be excluded without difficulty. Aneurysm may cause more difficulty, but in this case the patient's occupation and habits; the history of alcoholism, strain, or syphilis; and the presence of vascular degenerations, will assist us in arriving at a correct diagnosis.

In several of my cases the presence of hæmoptysis, pyrexia, night-sweating, and emaciation made the simulation of phthisis for a time close. In any case of doubt between pulmonary malignancy and phthisis, the following rules will be found helpful:

1. The history in the two cases is different: in phthisis we shall probably have a history of vague ill-health with debility, cough, and loss of weight; in pulmonary malignancy we

shall probably have a history of removal of the mamma, or of operations on bones, uterus, or rectum.

2. The age of the patients may present some contrast in the two cases. Phthisis is more likely to occur in adolescents, pulmonary malignancy in middle life or later. The average age in my seven cases was forty-two; only one was under thirty; two were over sixty.

3. The progress of pulmonary malignancy is usually more rapid than that of phthisis; there are no remissions of symptoms or periods of rally which are so common in phthisis.

4. The sputum may give decisive assistance, but the characters of the sputum in pulmonary malignancy are variable, and must be cautiously interpreted. The detection of tubercle bacilli would, of course, at once prove the presence of tuberculosis.

5. Enlargement of the glands at the root of the neck may be found in either disease.

6. Pain and dyspnœa are much more prominent in pulmonary malignancy than in phthisis, and much less under the influence of palliative measures.

7. Displacement of the heart, if not otherwise obviously explained, would be in favour of tumour.

As to the duration of cases of pulmonary malignancy, Walshe gives the average duration as 13·2 months. Osler thinks from six to eight months is more probable, and my statistics would tend to confirm this view. It is, of course, difficult and often impossible to determine the actual beginnings of the invasion of the lungs in these cases.

In some cases the history, signs, and symptoms point irresistibly to the presence of an intra-thoracic malignant growth, but it is difficult to say whether its seat is mediastinal, pleural, or pulmonary. A careful analysis of the early symptoms may give some assistance in determining this point, but in some cases the differential diagnosis is for a

time impossible. Early hæmoptysis would be a point in favour of a pulmonary lesion, while late hæmoptysis would possess less significance. The physical signs might assist the diagnosis—*e.g.*, if the earliest physical signs were those of infiltration, rather than of compression, of the lung substance.

In one of my cases the presence of malignant nodules on the abdominal wall gave an important clue to diagnosis. In any doubtful case the search for evidence of malignant disease elsewhere in the body is, of course, a point of the highest importance.

It is important to bear in mind that malignant disease of the lungs, like malignant disease of any organ, may be for a time almost or altogether latent. The clinical picture of malignancy usually present to our minds is essentially a picture of the advanced, or at least the fully-developed, disease. We cannot too carefully bear in mind that malignant disease of the lungs may for a time be practically without symptoms; that pain, dyspnœa, and cough may be absent; and that the patient may retain a full measure of nutrition until the case is far advanced.

The treatment of pulmonary malignancy does not offer much scope for therapeutic measures. The relief of pain may be effected by some of the usual means. For the relief of dyspnœa, hypodermic doses of strychnine and inhalations of oxygen are of some value. Hæmoptysis may be dealt with on the usual lines.

Abscess of the Lungs.

Abscess of the lungs is a rare condition. Amongst its causes may be enumerated:

(*a*) Croupous pneumonia, where it appears to supervene upon necrosis of the pulmonary tissue. Leyden, Biermer, and others have questioned the origin of pulmonary abscess in this manner, but apparently without sufficient reason.

(*b*) Pulmonary embolism, the emboli being in most cases septic. Puerperal fever is one of the chief causes of septic embolism.

(*c*) Empyema, subphrenic abscess, liver abscess.

(*d*) Suppuration of the bronchial glands.

(*e*) Typhoid fever.

Symptoms and Physical Signs. — The symptoms are variable, and depend much on cause. The important point is that the general constitutional symptoms of sepsis may be ill-marked or even altogether absent. In other cases, however, fever, dyspnœa, and severe cough may suggest the diagnosis, but in general our chief reliance must be upon the character of the physical signs and of the sputum. The physical signs are at first those of a limited collection of fluid—viz., dulness on percussion, diminished tactile fremitus, and absence of breath sounds, followed on evacuation of the pus cavity after coughing by those of excavation—viz., cavernous or amphoric respiration, gurgling râles, bronchophony, and perhaps pectoriloquy. As the abscess cavity again fills up, these latter physical signs are replaced by those of a limited collection of fluid. This alternation of physical signs, coupled with a characteristic sputum, may suffice for the diagnosis.

The sputum consists of pure, creamy pus; hæmatoidin crystals are present, elastic fibres are usually abundant; cholesterin is sometimes found. The sputum sometimes possesses a grass-green colour.

In some cases the pus from a pulmonary abscess finds its way into the pleura, pericardium, mediastinum, abdomen, or through the thoracic parietes.

The diagnosis turns mainly on—

(*a*) The presence of one of the usual causes of lung abscess.

(*b*) The alternations of the physical signs of a local collection of fluid with those of excavation.

(*c*) The evacuation of large quantities of purulent sputum

containing hæmatoidin crystals and elastic fibres, but not usually fœtid.

The prognosis must have regard to the antecedent cause of the abscess, the constitutional condition of the patient, the probability of spontaneous cure, or the feasibility of operative interference. The acute abscess is very fatal; the chronic abscess has many dangers, of which hæmorrhage is one of the most serious, but some cases do well.

The general treatment includes all measures, such as inhalations of carbolic acid, creosote, or thymol, which tend to prevent decomposition of the pus in a pus cavity; attention to the cleanliness of the mouth, and such measures as cold effusion to promote the expulsion of pus from the lung. General tonic and fortifying treatment is most important. If the progress of the case is not satisfactory, and the situation of the abscess can be fixed with any degree of probability, operative measures should be undertaken.

Gangrene of the Lungs.

Gangrene of the lungs is a somewhat rare condition. It occurs in two forms—the circumscribed and the diffuse, the former being the more frequent. In circumscribed gangrene the lung tissue surrounding the gangrenous area is acutely inflamed, and resembles hepatized lung. Within this zone the lung tissue may persist as a gangrenous mass, or a cavity may form. The contents of the cavity are horribly fœtid. It may contain blood. In the diffuse form there is no limiting zone of inflammation.

The causes of gangrene are as follows:

(*a*) Lobar pneumonia. Gangrene ensues in lobar pneumonia only in very exceptional cases, and chiefly in alcoholic subjects or diabetics. I have seen several cases in boys from rescue homes, whose constitutional state was very low.

(*b*) Aspiration pneumonia, depending on the lodgment in the lungs of particles of food such as fish-bones, cherry-

stones, or of pieces of malignant growth from the œsophagus or larynx.

(*c*) Putrid bronchitis, bronchiectasis, broncho-pneumonia, phthisis.

(*d*) Embolism of the pulmonary artery.

(*e*) Retro-pharyngeal, vertebral, or liver abscess.

(*f*) Carcinoma of the lungs.

The symptoms of pulmonary gangrene are chiefly profound physical prostration, hæmoptysis, cough, intense fœtor of the sputum, and pyrexia, usually asthenic in character and accompanied by chills. The pulse is very frequent, weak, and sometimes irregular; delirium may be present; cyanosis is frequent, and sordes may form on the lips and tongue. The cough is persistent and distressing.

The sputum is the most characteristic feature of the disease. It is commonly abundant, of an intensely foul and sickening odour, separates on standing into three layers, and contains connective tissue and elastic fibres. Traube denied that elastic tissue was present, but in this view he was apparently mistaken. This is one of the most important points in the differential diagnosis, as it helps to distinguish the disease from bronchiectasis. Numerous bacteria are present.

The physical signs are variable, and necessarily present many modifications, according as the affected area is consolidated, softened, or excavated. Sometimes after coughing and expectoration the usual signs of cavity may be recognised. Dulness and consonating râles are often present. In children the physical signs cannot in many cases be distinguished from those of broncho-pneumonia.

The chief complications are the following: Pyæmia; abscess of the brain; pleurisy, pneumonia, pericarditis; pneumothorax; hæmoptysis.

The differential diagnosis is often easy and rests upon the fœtor of the sputum and the presence of elastic fibres, the

signs of consolidation, softening or excavation of the lungs, the physical prostration, and the usually rapid progress of the case to a fatal termination. Putrid bronchitis, bronchiectasis, and phthisis can usually be excluded without much difficulty.

The prognosis is in general most unfavourable. Death usually takes place in from one or two to six, eight, or ten weeks. Recovery is more probable in the circumscribed than in the diffuse form. Recovery has often followed upon operative measures. Lenhartz states that in fifty-five cases operated on by him cure took place in 65 per cent.*

The treatment is both medicinal and operative. The former consists in generous diet, stimulants, and the use of such remedies as carbolic acid, creosote, or turpentine either by the mouth or by inhalation. Guaiacol may be given by hypodermic injection. Traube thought that acetate of lead was a valuable remedy.

Lenhartz warmly recommends operation in cases of circumscribed gangrene. The opening in the chest wall serves to afford the readiest exit for the gangrenous material, while it obviates the development of putrid bronchitis, and relieves the disgusting taste in the mouth which so often produces a repugnance for every kind of food.†

Actinomycosis of the Lungs.

Actinomycosis may attack the lungs either primarily or secondarily, the former in a small minority of cases. The ray-fungus gains access to the body chiefly by means of the food, especially cereals of the genus *Hordeum*. The jaw is the part most usually affected in man, as in the lower animals, and the intestinal tract, abdominal viscera, and skin may also be involved. The growth may reach the lungs either directly through the air tubes, or by metastasis through

* Lenhartz, *op. cit.*, Bd. i. 1, p. 346. † *Ibid.*

the channel of the blood. A form of broncho-pneumonia is set up, and small nodules develop, which have often been mistaken for tubercles. 'Round the fungus develops a mass of cellular inflammation, in which may ultimately be found both epithelial and giant cells. Round these nodules an abundant formation of connective tissue takes place. The vesicles in the neighbourhood become filled with catarrhal, epithelial, and small white cells, and ultimately they become completely obliterated by the growth of vascular connective tissue from the septa into their cavity. By the development of fresh nodules round, and their confluence with the original ones, a considerable mass may be formed, and the greater part or the whole of a lobe become involved. The longer the case lasts, the more connective tissue will be developed, and so a considerable and dense fibroid induration of the affected parts be produced.'* The pleura may become involved, also the neighbouring organs, and frequently the chest wall.

The symptoms are at first to a large extent latent, but as the disease progresses pain in the side and obstinate cough appear, the patient becomes febrile, night-sweating may be present, dyspnœa is more or less marked, and anæmia and emaciation develop.

The physical signs are those of pulmonary infiltration or consolidation or of chronic pleurisy, more rarely of excavation, and are not pathognomonic.

The character of the sputum is the most important point, and upon this in most cases the diagnosis turns. The sputum is muco-purulent or hæmorrhagic, and the characteristic yellowish granules may be recognised. Some observers have found elastic fibres, others deny their presence.

The diagnosis of actinomycosis has to be made from that of tuberculosis, empyema, new growths in the lungs or pleura, hydatid disease, and pyæmia. From tuberculosis

* S. West, *op. cit.*, vol. ii., pp. 844, 845.

the distinction may be difficult. The most important points are the absence of tubercle bacilli and the presence of the characteristic granules in the sputum; the fact that actinomycosis usually begins in the lower portions of the lungs; the tendency of actinomycosis to involve the chest wall. From empyema the distinction can only be made with certainty by the detection of the ray-fungus in the pus withdrawn by aspiration. The simulation of new growth in the lung and of hydatids is somewhat less close. Pyæmia can in many cases be excluded only by detection of the fungus in the pus from an abscess or in the sputum. In general the diagnosis turns mainly on the patient's occupation—viz., such as would be likely to expose him to infection, the coexistence of pulmonary signs with lesions of the jaw, skin, or chest wall, and the discovery of the fungus.

The prognosis is eminently unfavourable, but recovery is not absolutely unknown.

The treatment consists in full doses of iodide of potash, injections of tuberculin, injections of corrosive sublimate, or operative interference. This last measure has hitherto had little success; it consists in opening and scraping the abscesses in the lungs or pleura when these are within reach.

Hydatids of the Lungs.

The lungs and pleura are involved in from 7 to 12 per cent. of cases of hydatid disease, the lungs being much more frequently involved than the pleura. The part most often involved is the right lower lobe. Usually only one cyst is present, but the occurrence of several cysts is not uncommon. The disease is commonest between the twentieth and the fortieth year. The cyst is usually of moderate size, but may be as large as a child's head, or may even occupy a whole lobe. In some cases rupture takes place even before the cyst has attained any considerable size. Suppuration of the cyst and calcification of the cyst sometimes occur.

The symptoms are often for a considerable time latent, the cyst seemingly exercising simply slight pressure, and hardly any irritative effects. Serious symptoms often arise only after the hydatid has perished and the cyst become inflamed; when cough and dyspnœa and pain appear and hæmoptysis is not uncommon. The physical signs are as follows:

(*a*) Dulness on percussion, usually rounded in outline, pronounced at the centre, and shading off towards the periphery.

(*b*) Absence of breath sounds or weak bronchial breathing.

(*c*) Absence of râles.

Signs of a purulent pleural effusion may be present. When rupture occurs sudden symptoms of suffocation may arise, but often the cyst empties itself gradually. With discharge of the contents of the cyst signs of cavity may appear.

The diagnosis may for a time be difficult or impossible, unless the presence of the hydatid membrane can be recognised in the sputum, or in fluid removed by exploratory puncture. The distinction from phthisis depends on the following points:

(*a*) In phthisis the upper, in hydatids the lower lobes are usually the seat of the disease.

(*b*) In phthisis some flattening of one or both infraclavicular regions will probably be present; not so in hydatids.

(*c*) In phthisis signs of excavation will be preceded by those of infiltration and softening; in hydatids signs of excavation will be preceded by those of a limited collection of fluid.

(*d*) In phthisis there is usually more or less cachexia; little or none in hydatids.

(*e*) The character of the sputum—tubercle bacilli present in phthisis, hydatid membrane in hydatids.

Hydatid fluid is usually clear and limpid, contains little or no albumin, and has an excess of chloride of sodium. If the fluid be purulent, a careful search must be made for the characteristic hooklets.

The prognosis is doubtful. Perforation of the cyst into the

pleura or the pericardium may cause death from shock, or it may give rise to complications ultimately leading to a fatal issue. Gangrene of the cyst may lead to general sepsis and death. Many cases remain latent for long periods. Rupture of the cyst into a bronchus and expectoration of its contents is followed by recovery in a majority of cases.

The treatment is mainly surgical, if the cyst is within reach. Paracentesis rarely results in cure, and incision should be preferred. Where for any cause operative interference is impracticable, the inhalation of turpentine or eucalyptol is advised, or myrtol may be given internally. Injections of carbolic acid into the cyst have been advised, but are not free from danger.

Syphilis of the Lungs.

Syphilis rarely attacks the lungs. Many of the cases recorded as syphilitic are probably tubercular. Bronchitis is an occasional feature of secondary syphilis. It possesses no characteristic features or significance.

Virchow, Wagner, and others have taught us to recognise two forms of syphilitic lung—viz., a gummatous form and a form of chronic interstitial indurative pneumonia. The former affects the root of the lung by preference, and in most cases both lungs are involved. Caseation takes place in the centre of the gumma. The tendency to excavation is less pronounced than in tuberculosis. The interstitial form consists of a nodular overgrowth of connective tissue which undergoes induration and contraction. In many cases it seems to have its origin from the peri-bronchial tissue.

The symptoms are not very characteristic. Dyspnœa is somewhat marked; pain may be present; hæmoptysis is not frequent; pyrexia, night-sweating, and emaciation are much less prominent than in tuberculosis; the sputum is sometimes profuse, purulent, and offensive; tubercle bacilli

are, of course, absent, unless tuberculosis is present as a complication.

The physical signs are not characteristic, and cannot be distinguished with certainty from those of phthisis. The chief points in the diagnosis will be: the presence of signs of consolidation or excavation near the root of the lung; absence of tubercle bacilli from the sputum; the presence of tertiary syphilitic lesions in larynx, liver, spleen, or testis; moderate constitutional symptoms; improvement or recovery under specific treatment.

LECTURE XXIII

THE PULMONARY COMPLICATIONS OF OTHER DISEASES

SUMMARY:

Frequency of pulmonary complications in many diseases.

A. The specific fevers, pulmonary complications in:

Typhoid fever; typhus fever; relapsing fever; scarlatina; rötheln; measles; whooping-cough; small-pox; diphtheria; influenza; plague; malaria.

B. Circulatory diseases, pulmonary complications in.

C. Diseases of the digestive system, pulmonary complications in.

D. Diseases of the kidneys, pulmonary complications in.

E. Diseases of the nervous system, pulmonary complications in:

Cerebral hæmorrhage; bulbar paralysis; Landry's paralysis; spinal myelitis; insanity; hysteria—hysterical cough and dyspnœa, hysterical cramp of the diaphragm; asthma.

F. Some general diseases:

Diabetes; gout; rheumatism; rickets; scurvy; the anæmias.

Pulmonary complications are amongst the commonest and most important complications in many diseases. They have much prognostic importance, and are often the determining

cause of death. Watchfulness regarding their development and care in their management are indispensable. I propose to pass in brief review the principal groups of disease, indicating the chief pulmonary complications which they usually present and, when possible, the special features of such complications.

A. The Specific Infective Diseases.

Typhoid Fever.—The pulmonary complications of typhoid fever are numerous and important. Some degree of bronchitis is almost invariable in the early stage of the disease, and this feature may be allowed some diagnostic weight in a doubtful case. Bronchitis often becomes more marked during the third week of the attack. The degree of bronchitis may be slight in favourable cases which are early brought under careful treatment; while in patients who are neglected, or in whom there is marked disturbance of the nervous system, leading to the accumulation of mucus or the presence of particles of food in the air passages, the bronchitis may be severe and general, and may develop into broncho-pneumonia with or without pulmonary collapse. In rare cases the bronchitis takes on the putrid type, and may be followed by gangrene of the lung.

The above complications are relatively more common in corpulent patients, in elderly persons, in the kypho-scoliotic, and in those who have previously suffered from emphysema or heart disease.*

The subjective symptoms in the above complications may be ill-marked. There may be little cough or expectoration, owing to the torpid condition of the patient; but some degree of dyspnœa may be observed, and in the severer cases cyanosis will be present. The physical signs present no special peculiarities. The condition of the alæ nasi should be observed.

* Strümpell, 'A Text-book of Medicine,' English edition, p. 14.

Lobar pneumonia occurs in about 8 per cent. of cases. It may be an early symptom, and in such cases may possibly be due to the direct action of the typhoid bacillus on the pulmonary parenchyma. These cases set in with the ordinary signs and symptoms of pneumonia, and at first there is no suspicion of typhoid fever; but it is observed that the initial headache is marked and prolonged, that the case is somewhat asthenic from the start, that no crisis occurs, and that in the course of the second week spots, or characteristic diarrhœa, or marked enlargement of the spleen, or abdominal distension, makes its appearance, and the true nature of the case becomes evident. These cases are called *pneumo-typhus* by German writers. Only one well-marked example of this type has occurred in my experience.

Lobar pneumonia, depending on the presence of the pneumococcus, is most common in the third and fourth weeks of the disease. The symptoms may be ill-marked, but the physical signs are distinctive. Pleurisy is a not infrequent complication, and the resulting effusion may be purulent. Pneumothorax is very rare. Pulmonary infarction and pulmonary abscess are occasionally present.

Typhus Fever.—The pulmonary complications of typhus are not very frequent or characteristic. Broncho-pneumonia is the most important. It has no special features. Bronchitis is comparatively common in the initial stage. Pleurisy and pulmonary gangrene are occasionally observed.

Relapsing Fever.—The pulmonary complications of relapsing fever are not frequent. Pneumonia is the most important.

Scarlatina.—The pulmonary complications of scarlatina are not frequent or important. Lobular pneumonia occurs sometimes at an early stage, but more often at a later stage, in connection with renal developments. Pleurisy is not uncommon, and the effusion is often purulent.

Rötheln.—The pulmonary complications of rötheln are

unimportant. Pneumonia has been observed in some epidemics.

MEASLES.—The pulmonary complications of measles are numerous and important. In many epidemics the mortality has depended almost wholly on pulmonary involvements, which are especially common and deadly amongst the children of the poorer classes.

A certain degree of bronchitis is practically invariable in measles. In many cases this develops into a severe general bronchitis, which often invades the finer tubes (capillary bronchitis), and areas of more or less definite consolidation may be observed (broncho-pneumonia). Atelectasis often ensues. These grave complications arise usually at the height of the attack or at the commencement of desquamation. They should be suspected if the temperature remains high, the cough becomes hard, painful, and incessant, and the pulse frequent and weak. In feeble children the chief symptoms may be marked movement of the alæ nasi, increasing frequency of the pulse, cyanosis, and progressive exhaustion. Such symptoms are of grave prognostic significance.

Lobar pneumonia is much less frequent and less fatal.

WHOOPING-COUGH. — The pulmonary complications of whooping-cough are frequent and important. They account for the great majority of fatal cases of the disease. According to Osler, capillary bronchitis, lobular and pseudo-lobar pneumonia, account for nine out of ten deaths. Pulmonary collapse may be pronounced. Pleurisy is less frequent. Emphysema may result from strain during coughing, and may be the starting-point of chronic bronchitis. Pulmonary tuberculosis is an occasional sequela of whooping-cough. It may be a question whether the disease opens the way to bacillary infection, or simply stirs up quiescent tubercle to activity.

Hæmoptysis may occur in whooping-cough apart from tuberculosis, but much oftener in association with it.

When a child has a retarded convalescence from whooping-cough, the possibility of a pulmonary cause should always be before our minds. If pyrexia, wasting, and debility are present, the probability of tuberculosis is strong; but the disease may have its seat in the bronchial glands, and a positive diagnosis may for a time be impossible. The preventive treatment of tuberculosis should be adopted in all suspicious cases.

SMALL-POX.—The pulmonary complications of small-pox are common, especially in severe cases. Broncho-pneumonia is the chief, and is a common cause of death. It is often accompanied by pleurisy.

Lobar pneumonia is much less frequent.

DIPHTHERIA.—The pulmonary complications of diphtheria are numerous and important, and are responsible for a large part of the mortality from the disease. A true croupous bronchitis, due to the extension of the membrane to the bronchioles, is present in some cases. Much oftener, however, we find a broncho-pneumonia associated with the presence of pneumococci or streptococci.

Lobar pneumonia is much less frequent.

Lobular pneumonia may persist after the general symptoms of diphtheria have disappeared, and may lead to a fatal issue.

INFLUENZA.—The pulmonary complications of influenza are frequent and important, and are the chief cause of the mortality from the disease. A 'catarrhal' or 'pulmonary' type of the disease is described. A certain degree of bronchial catarrh is present in most cases; pleurisy is sometimes observed, and the effusion may be purulent; but by far the most important of these complications is pneumonia, usually of the lobular type.

Influenzal pneumonia is a very important condition, and generally presents the following features in more or less typical form :

(*a*) It is usually of the lobular type, and may depend on the presence of Pfeiffer's bacillus or of mixed infections.

(*b*) The symptoms are often acute and intense, but there is usually a tendency to asthenia.

(*c*) The cough is often dry and harassing, and the sputum scanty and tenacious; later it is more abundant and looser.

(*d*) The physical signs are often obscure and ill-defined; weak breathing and scanty 'sticky' râles are common; some impairment of resonance is noted rather than distinct dulness; vocal resonance and vocal fremitus are sometimes reduced; patches of bronchial breathing may be present.

(*e*) The physical signs show a marked tendency to shift about, a fresh area developing as another area clears up.

(*f*) The course of the case is sometimes prolonged and indefinite.

PLAGUE.—In the ordinary type of bubonic plague pulmonary complications are somewhat rare, but a special 'pneumonic' or 'pulmonary' type of the disease is recognised, of which the chief characters are the severity of the symptoms, the brief course, and the high mortality—viz., about 96 per cent.

The usual physical signs of pneumonia are present. The sputum is sometimes bloody, and contains large numbers of the plague bacillus.

MALARIA.—The pulmonary complications of malaria are not very characteristic. Bronchitis and pneumonia are occasionally observed.

In many other infective diseases, not included in the above list, pulmonary complications play a part in the clinical history, and often have a marked influence upon the mortality; but it cannot be said that they present any distinctive characters. Broncho-pneumonia in particular may occur in almost any infective disease, and often is in relation to the amount of prostration or asthenia present. Febrile, bed-ridden patients

are specially prone to this affection, and it is important to bear in mind that in many of this group of diseases broncho-pneumonia may be ill-marked as regards both symptoms and physical signs, but yet very fatal. The general treatment of the pulmonary complications of infective diseases requires to be tonic and stimulant, rather than of an antiphlogistic character. All depressing measures must be avoided.

B. Circulatory Diseases.

Pulmonary complications are amongst the commonest of complications in connection with diseases of the circulatory system. They take the form usually of passive congestion and œdema, but broncho-pneumonia is not uncommon, and pulmonary infarction occasionally occurs. Hydrothorax is frequent. These conditions usually depend upon disease of the mitral valve, and for obvious mechanical reasons the stress of the lesion falls upon the lungs to a more marked degree in mitral stenosis than in mitral regurgitation. Pulmonary tuberculosis, formerly thought to be excessively rare in cardiac disease, is now known to be not uncommon in mitral stenosis and in congenital obstruction of the pulmonary valve.

The lungs in chronic and advanced mitral disease are found to be engorged with dark blood; their fibrous tissue is denser than usual; the lung is tough; the capillaries of the alveoli are dilated and varicose, and their walls are thickened; there is a marked increase of pigments; the bronchial mucous membrane is congested; hæmorrhagic extravasations and infarctions of the pulmonary artery may be present. In hypostatic congestion, as in prolonged debilitating diseases, the posterior parts of the lungs are dark in colour, and engorged with serum and blood. Portions of the lungs may sink in water.

Embolism of a branch of the pulmonary artery may give rise to a symptom-complex not always easy of distinction

from phthisis.* The chief diagnostic guides in such cases are the following: The physical signs consequent upon pulmonary embolism develop suddenly; they are rarely apical in seat; they are accompanied by local pain and dyspnœa; the sputum is bloody and mucoid, but does not contain any elastic fibres or tubercle bacilli. The history is one of rheumatism and endocarditis, not of tuberculosis.

The diagnostic problems which arise in connection with the pulmonary complications of circulatory disease are, upon the whole, not very difficult. The careful practitioner will always watch the lungs sedulously in cardiac cases; he will attach due weight to such a symptom as hæmoptysis, recollecting, on the one hand, that it is often an evidence of somewhat intense pulmonary congestion or infarction, and, on the other hand, that it exercises a certain derivative effect, and may tend to the relief of the patient. On the whole, it is a serious symptom, but rarely the source of immediate danger. He will be watchful for the appearance of hydrothorax, and will know when to relieve it by a timely aspiration. He will recollect the tendency to pulmonary tuberculosis in children who are the subjects of congenital obstruction of the pulmonary artery. He will carefully estimate the presence and amount of pulmonary œdema, and will mark whether it tends to increase or subside. He will recognise the signs and symptoms of brown induration of the lungs.

Hæmoptysis from the rupture of an aneurysm of a branch of the pulmonary artery has been already considered.†

An aortic aneurysm may compress the lungs and give rise to the so-called 'aneurysmal phthisis.'

C. Diseases of the Digestive System.

The pulmonary complications of diseases of the digestive organs are, upon the whole, of minor importance.

* See p. 339. † See p. 446.

The entrance of particles of food into the air passages may give rise to an irritative bronchitis, broncho-pneumonia, pneumonia, or pulmonary gangrene. The same results may follow from the penetration into the air passages of particles of a malignant growth in the mouth, pharynx, or œsophagus. Blood regurgitated from the stomach may find its way into the air passages, and cause difficulty in the differential diagnosis of gastric ulcer and pulmonary tuberculosis.* Tuberculosis of the tonsils may be a precursor of, or associated with, pulmonary tuberculosis. In rare cases rupture of the œsophagus has led to inflammatory affections of the pleura and lungs. One case of this kind has occurred in my experience. Gastric ulcer may be followed by the formation of a subphrenic abscess, which may rupture through the diaphragm, and become the starting-point of empyema and pyo-pneumothorax. Of this course of events I have seen one example. Jaundice is a not uncommon accompaniment of pneumonia. Cancer of the liver may give rise to pleural effusion.

D. Diseases of the Kidneys.

The pulmonary complications of renal disease are frequent and important. The chief of these are the following :

(*a*) Œdema of the lungs.

(*b*) Pleural effusion, either as part of a general renal dropsy or of inflammatory origin.

(*c*) Pneumonia, either croupous or catarrhal.

(*d*) Bronchitis.

(*e*) Cheyne-Stokes breathing.

As a rule, these conditions do not present serious diagnostic difficulties. Œdema of the lungs sometimes sets in quite suddenly, and apparently without obvious cause. It may in a few hours put in imminent peril a patient who seemed previously in a fairly satisfactory state. This possibility

* See *ante*, p. 444.

should not be overlooked in giving a prognosis in renal cases. The prognosis in pulmonary œdema thus arising is, according to my experience, extremely grave. It is still a question how far pulmonary œdema depends upon failure of the left heart, and how far upon local changes in the walls of the pulmonary vessels.

Strümpell is of opinion that the pneumonia of renal cases often 'stands midway between a catarrhal and a croupous inflammation.'* The prognosis in these cases is unfavourable.

Cheyne-Stokes breathing generally portends a rapidly fatal issue, but there are exceptions to this rule.

Hæmoptysis is, upon the whole, somewhat rare in renal cases.

E. Diseases of the Nervous System.

The pulmonary complications of diseases of the nervous system are numerous, but not specially characteristic. They are conditioned chiefly by the following considerations:

(*a*) The severity and duration of shock—*e.g.*, in cerebral hæmorrhage.

(*b*) The degree of helplessness of the patient—*e.g.*, in many cases of hemiplegia, spinal myelitis, locomotor ataxia.

(*c*) The mental state of the patient—*e.g.*, in insanity, hysteria.

(*d*) Irritation or paralysis of the muscles concerned in respiration.

(*e*) The aspiration of particles of food, or of secretions from the mouth, nose, or pharynx into the air passages, most often seen in cases of coma or mental alienation.

We do not often get hints of value in the diagnosis of nervous disease from signs or symptoms referable to the lungs; often enough we get prognostic indications of great

* A. Strümpell, *op. cit.*, p. 611.

importance. In states of exhaustion the result of chronic nerve disease, pulmonary complications are often the determining cause of death.

In cerebral hæmorrhage the bases of the lungs are often deeply engorged, and on section the lungs drip with blood and serum. They usually continue to contain air. These changes are sometimes more marked on the hemiplegic side. The breathing in such cases is often deep and laboured. In bulbar paralysis, owing to loss of control of the mechanism of deglutition, aspiration-pneumonia is common. In Landry's paralysis the determining cause of death may be paralysis of the muscles of respiration.

Spinal diseases have, in general, no characteristic pulmonary complications; but passive congestion and œdema of the lungs are common in bed-ridden and helpless patients. When myelitis invades the cervical region of the cord, it may lead to attacks of dyspnœa, and, in some cases, to death. Paralysis of the diaphragm may occur in these cases. Insane patients are prone to pulmonary complications, especially tuberculosis, which is the cause of death in about two-thirds of these cases.

Hysteria presents some interesting respiratory phenomena. Hysterical dyspnœa may take on protean forms. Often the breathing is simply excessively rapid (80 to 100 respirations per minute), without any apparent distress. The patient may take deep 'catching' breaths, or make sucking sounds with the lips and tongue. However rapid the respirations, there is seldom any cyanosis or genuine air-hunger. Much more rarely the breathing is abnormally slow (8 to 10 respirations per minute), and may be accompanied by audible wheezing in inspiration and expiration. Hiccough is common. The hysterical cough is well known ; it is a dry, barking, or croaking sound, unaccompanied, as a rule, by any expectoration; but the patient may contrive to manufacture a false hæmoptysis, usually by sucking her gums.

Hysterical cramp of the diaphragm is occasionally observed. The physical signs of this rare condition are as follows: Prominence of the epigastrium, cessation of the diaphragmatic movements, cessation of the abdominal respirator movements, excessive action of the upper parts of the thorax, extension of the limits of pulmonary resonance to a lower point than usual. Stuttering speech may be part of the respiratory phenomena of hysteria.

How far asthma is at bottom a neurosis of the vagus nerve it is impossible to determine. This view is identical with that which recognises spasm of the bronchioles as the fundamental fact in this affection. It does not, however, account satisfactorily for the frequency of bronchial catarrh in asthma, or for the presence of Curschmann's spirals and Charcot-Leyden crystals in the sputum.

A form of cough supposed to be due to irritation of the trigeminal fibres distributed to the nose, pharynx, and external auditory meatus has been described by Schadewald and Wille.*

F. Some General Diseases.

Diabetes.—The pulmonary complications of diabetes are frequent and important. Many patients die of acute pneumonia. Gangrene is not uncommon. Nearly half of all diebetic patients ultimately die of pulmonary tuberculosis.

Gout.—Gouty patients are very liable to attacks of bronchitis, which often take on the form of the 'catarrh sec' of Laennec. Cough is severe, there is much wheezing, sibilant rhonchi are heard, and the sputum is scanty and of the glutinous or 'pearly' type. In some cases the attacks are more severe in character, and are accompanied by the physical signs of pulmonary congestion.

Most sufferers from chronic gout present evidence of the presence of emphysema.

* L. Hirt, 'The Diseases of the Nervous System,' English edition, p. 76.

Rheumatism.—There are no characteristic pulmonary involvements in rheumatism. Many writers recognise the existence of a 'rheumatic' pleurisy.

The existence of an 'arthritic' type of hæmoptysis, as held by the late Sir Andrew Clark, must be considered doubtful.

Rickets. — Pulmonary complications are common in rickets. They are predisposed to by the low state of nutrition of rickety patients, and by the imperfect osseous and muscular development of the thorax in these cases. Bronchitis is frequent; broncho-pneumonia is also common, and may be the determining cause of death. Rickety children require special care, both as regards the prevention and the treatment of pulmonary disease. Rickety children do not often become tubercular.

Scurvy.—Pulmonary complications are rare in scurvy. Hæmoptysis occurs, but is decidedly infrequent. Hæmorrhagic infarction of the lungs has been occasionally observed.

The Anæmias.—There are no characteristic pulmonary complications in the anæmias, either of the chlorotic or pernicious type. The notion held by Trousseau that chlorotic girls, if cured of their chlorosis, are apt to become tubercular is opposed to all experience, at all events in this country. I should say that the very opposite rule is much nearer the truth.

LECTURE XXIV

PULMONARY DISEASE IN CHILDREN

SUMMARY:

Anatomical considerations.
Physiological considerations.
Pathological considerations.
Considerations arising out of the state of the sensorium in the young child.
Difficulties of diagnosis in the pulmonary diseases of children.
Physical examination of young children. Methods, difficulties, and peculiarities of the physical signs.
Bronchitis, acute and chronic.
Broncho-pneumonia.
Croupous pneumonia.
Pleurisy.
Phthisis.

We shall devote the present lecture to a consideration of those points in which some of the principal pulmonary diseases in children present material differences from the same affections as found in the adult. Those differences are important, and are conditioned by the following considerations:

(*a*) ANATOMICAL CONSIDERATIONS.—The general shape of the thorax in the infant is cylindrical, rather than conical or dome-shaped, the antero-posterior and transverse diameters of the chest being nearly equal until the third year of life;

the thoracic walls are relatively thinner than in the adult, they are elastic and yielding, and their musculature is relatively ill-developed; the capacity of the thorax is encroached upon by the high position of the diaphragm, the large size of the thymus gland, and the frequency with which flatulent distension of the stomach and bowels is present;* the trachea and bronchi, though relatively larger in the infant than in the adult, are so small that slight causes produce grave impediment to the free entry and exit of air; the air cells are smaller than in the adult, hence irritants excite inflammatory effects more easily; the interstitial tissue is more abundant.

It is evident from the above considerations that the young child is not only more likely to contract pulmonary disease, but that it is at a disadvantage in contending with such disease. Its small air passages are easily obstructed, and its soft and yielding thorax, with its ill-developed muscles, is ill adapted for those additional efforts which respiratory difficulties demand.

(*b*) Physiological Considerations.—The metabolism of the child is more rapid than that of the adult; its normal rate of respiration is greater. Hence, these processes are more unstable than in the adult and more readily thrown into disorder.

(*c*) Pathological Considerations.—Catarrhal processes tend to be active in the young child; they are readily excited by slight causes of irritation; they are more prone to extend their area than similar processes in the adult.

(*d*) Considerations arising out of the Condition of the Sensorium in the Young Child.—Cough is a purely reflex act in the infant, in whom there is little or no voluntary effort towards the expulsion of secretions or irritating material from the air passages; the sputum is usually swallowed.

* L. E. Holt, 'The Diseases of Infancy and Childhood,' third edition, p. 510.

The above considerations easily explain the frequency and the grave character of pulmonary affections in the young child. The child readily becomes the subject of catarrhal processes; the narrow air passages are easily obstructed, the soft yielding thorax easily sinks in from atmospheric pressure when obstructive dyspnœa is present; the delicate air vesicles easily become the seat of emphysema; the tendency to pulmonary collapse is marked; the accessory muscles of respiration do not respond well to the additional calls upon them made by the respiratory needs of the organism; the small air cells are readily excited to inflame; catarrhal processes are prone to extend.

Difficulties of Diagnosis.—The difficulties of diagnosis in the pulmonary affections of young children are often considerable. The child does not assist the efforts of the diagnostician: it may be alarmed by the sight of a strange face or of a stethoscope; it does not always breathe well; it is prone to cry; its examination requires patience and time. Further, the physical signs, both physiological and pathological, present certain differences which are not always borne in mind, and which easily become the source of erroneous inferences. Puerile breathing is mistaken for bronchial breathing; small areas of consolidation are overlooked because the percussion stroke is too heavy, or because the yielding walls of the infantile thorax afford a more resonant note under certain conditions than might be expected; the chest is not fully explored because the child is restless or fretful, and important physical signs are thus missed; the fact that bronchial breathing is common in the pleural effusion of the child is not sufficiently borne in mind; an apical pneumonia is overlooked; the absence of cough and expectoration is sometimes puzzling; the prominence of gastric or of cerebral symptoms in the pulmonary affections of childhood may mislead the unwary practitioner.

PHYSICAL EXAMINATION OF YOUNG CHILDREN.—This requires time, patience, and tact, but does not present any insuperable difficulties. Care should be taken that the room is comfortably warm; that the hands of the examiner are not too cold; that nothing is done at the outset of the examination to startle or alarm the child; that the child is allowed to get accustomed to the sight of a stethoscope or other instrument before they are applied to the chest; that percussion is gentle; that the diagnostic importance of the cry is borne in mind.

Inspection.—Among the points which may call for special attention in the case of children are the following: Abnormal shape and development of the thorax; inequalities in the expansion of the two sides, or portions of the two sides, of the thorax; recession of the lower ribs and intercostal spaces.

Palpation.—The vocal fremitus can often be determined from the cry. The apex-beat of the heart will usually be found in the fourth intercostal space, in the left mammary line, or a little exterior to this line.

Percussion.—This must be gentle. Normal resonance is more marked in the infant than in the adult. The cracked-pot sound is sometimes elicited on percussing the back of a crying baby under normal conditions. Care must be taken not to overlook small areas of consolidation by too vigorous percussion. Dulness due to the thymus gland may be detected over the sternum as low as the third rib. A 'boxy' note may be heard in the early stage of pneumonia or in acute congestion of the lungs.

Auscultation.—The naked ear can be used for auscultating a baby's back, but is not convenient for exploring the apical and axillary regions. With a little patience a child will usually permit the application of a stethoscope.

The breathing of the infant is what is called *puerile*—*i.e.*, harsher, louder, and more superficial than the breathing of

the adult. These characters are due to the fact that the sounds have not to be conveyed through so thick a layer of lung and chest-wall as in the case of the adult. Puerile breathing is often mistaken for bronchial breathing, but this mistake is easily avoided by attention to the fact that bronchial breathing is always well marked during expiration, while puerile breathing is not. The breathing under the right clavicle in children with thin chest-walls is often bronchial in character, owing to the proximity of the relalatively large right bronchus. The auscultator must be on his guard on this point, and must be careful to correct the conclusions which he draws from auscultation of this region by attention to the other modes of examination, more especially percussion. The breathing of the infant is often jerky and unequal, and it is important that the auscultator should assure himself that such conditions are physiological, and not pathological. The difficulty of distinguishing bronchial râles from friction sounds is commonly greater in the case of the child than in that of the adult.*

As the child will not always pronounce words at command, the auscultator should accustom himself to estimate the conduction of the cough. The bronchophonic quality of the cough in cases of consolidation of the lung is readily appreciated.

Absence of breath sounds at one base from blocking of the bronchi with secretion is common in young children. After a fit of crying or cough the breathing in such cases often re-appears.

Bronchial breathing, usually weak and 'distant' in quality, is common in the pleural effusions of infancy and childhood. Definite signs of excavation are relatively rare in young children.

The 'fine crepitation' of commencing pneumonia is not often heard in young children.

* See p. 143.

Bronchitis.—Bronchial affections are extremely frequent in young children, and are apt to assume a grave form, owing to the small calibre of the air passages, the imperfect muscular and osseous development of the thorax, the readiness with which the bronchial tubes become blocked with secretion, the tendency to pulmonary collapse, the proneness of bronchitis to invade the smaller bronchi and the air cells. Cases of mild bronchitis limited to the larger tubes are, however, quite common in young children.

The symptoms are similar to those of the same affection in the adult, but in many cases more severe. The temperature rises higher, the respirations are more accelerated, the movements of the alæ nasi are marked, the increased frequency of the pulse is more marked. Cough is more variable than in the adult; it may be constant and severe, or slight or even absent, especially in the worst cases. The sputum is usually swallowed. Diarrhœa is frequent.

When bronchitis extends to the finer tubes, all the symptoms become intensified, and soon in severe cases evidence of deficient aeration of the blood appears. The child becomes cyanotic; there is dulness, apathy, or stupor; the cough and cry cease; the pulse is very rapid and weak.

The physical signs are those of generalized bronchial catarrh. Signs of pulmonary collapse and of acute emphysema are relatively frequent. When the inflammatory process invades the finer tubes, we almost always get the signs of limited patches of consolidation of the lungs—in other words, of broncho-pneumonia. When decided dulness is present, the possibility of fluid, especially pus, should always be considered. The difficulty of distinguishing the broncho-pneumonia of children from miliary tuberculosis has already been considered.*

The prognosis in the bronchitis of children depends on the

* See p. 341.

age of the patient, being worse in infants than in older children; on the height of the temperature; the extent of tubes involved; and on the cause of the bronchitis. If the affection is associated with whooping-cough, measles, scarlatina, nephritis, or rickets, the prospect is less favourable than in a primary bronchitis.

Chronic bronchitis does not present any characteristic features in children. It is, on the whole, somewhat rare. It is found in connection with chronic cardiac disease, tuberculosis, hereditary syphilis, rickets, general malnutrition, and as a consequence of the acute affection. The cough sometimes is spasmodic in character, and the distinction from whooping-cough may present some difficulty. A certain somewhat small proportion of cases of chronic bronchitis in children develop bronchial asthma. The best treatment for chronic bronchitis in children is change of climate, and the adoption of as tonic and fortifying a regimen as possible. Sedatives and lowering measures generally should, as far as possible, be eschewed.

Broncho-Pneumonia.—In the first three years of life broncho-pneumonia is decidedly more frequent than croupous pneumonia. It is the special scourge of infancy, and throughout childhood the great majority of cases of secondary pneumonia are of this type. According to Holt, broncho-pneumonia is very infrequent as a primary affection after the fourth year, but is seen throughout childhood as a complication of the infectious diseases.*

The broncho-pneumonia of childhood cannot be marked off in any definite way from the same affection in the adult. The following points may, however, be indicated as offering a certain contrast: Broncho-pneumonia is more frequent and more deadly in the child than in the adult; the temperature is usually higher; gastro-intestinal symptoms are relatively prominent and severe; the bronchial glands are

* L. E. Holt, *op. cit.*, p. 531.

oftener involved; the danger of sequential tuberculosis is greater.

PNEUMONIA.—Croupous pneumonia is essentially the same disease in children as in adults, but some points of contrast may be noted:

(*a*) *Mode of Invasion.*—This is as abrupt in the child as in the adult, but in the former a rigor is exceptional, while vomiting and convulsions are relatively frequent. Baginsky found vomiting in 66 per cent., rigor in only 6 per cent., of his cases.* Holt found convulsions in 5 per cent. of cases.†

(*b*) *Character of the Symptoms.*—The temperature may be higher in the child, the pulse very rapid—viz., 120 to 140 or 160 beats per minute; the respirations are usually more frequent—viz., 50, 60, 80, or even more, per minute, and a marked frequency of the pulse and respirations has not the same grave significance in the child which it possesses in the adult; the cough may be suppressed; sputum is usually absent; herpes labialis is relatively frequent; severe cerebral symptoms—viz., convulsions, delirium, unconsciousness, coma—are relatively frequent; symptoms of meningeal irritation, such as retraction of the head, are occasionally seen; defervescence by lysis is more frequent than in the adult.

The pulse has less prognostic significance in the child than in the adult; even a high degree of frequency has comparatively little significance, but irregularity of the pulse should always lead to inquiry for cerebral involvements.

The blood changes are marked in the pneumonia of children—viz., increased density of the blood, which may later give place to diminished density; increase in the amount of hæmoglobin and in the number of white cor-

* A. Baginsky, 'Lehrbuch der Kinderkrankheiten,' seventh edition, p. 681.

† L. E. Holt, *op. cit.*, p. 567.

puscles, especially of the polynuclear leucocytes, while the eosinophile leucocytes are diminished or absent.*

Signs of heart failure, and especially signs of over-distension of the right heart, are less frequent in the child than in the adult.

The temperature in the pneumonia of children has the following characters: The initial rise is usually marked and the fastigium high until the crisis, but in children under three years of age a more or less remittent type of pyrexia may be present from the first; the general range of the temperature is sometimes higher than in adults; a pre-critical fall of temperature is relatively common; a post-critical rise, due to extension of the pneumonic process to fresh areas of lung, to purulent pleural effusion, and more rarely to meningitis, pericarditis, gastro-enteritis, or malaria, is not infrequent; defervescence by lysis is comparatively common. Holt found defervescence by lysis in 44 out of 93 cases, while in 522 cases collected by him, 396 ended by crisis and 126 by lysis.†

The migratory type of pneumonia is not uncommon in children. In one of my cases the temperature remained high until the twenty-first day, owing to progressive involvements of fresh areas of lung.

Gastric and intestinal symptoms are commonly well marked in children. Vomiting and diarrhœa are frequent initial phenomena. These symptoms, as a rule, soon abate.

Nervous symptoms are frequent in children, and are a common source of error in diagnosis. Convulsions, delirium, drowsiness, stupor, opisthotonos, dilated or contracted pupils, irregular pulse, retracted abdomen, incontinence of urine or fæces, may all be present. In such cases a diagnosis of meningitis is often made. This unfortunate error can be avoided by attention to the following points—viz., the sud-

* A. Baginsky, *op. cit.*, p. 683.

† L. E. Holt, *op. cit.*, p. 569.

denness of the onset (much more marked in pneumonia than in meningitis); the rapidity of the breathing; the height of the temperature; the results of physical examination of the thorax.

Urinary symptoms are seldom prominent in children, and albuminuria is less frequent than in the case of adults.

(*c*) *Character of the Physical Signs.*—As a rule there are no distinguishing peculiarities in the physical signs present in the pneumonia of children. The observer must be careful, however, not to confuse the physiological peculiarities of the physical signs in the chest of the child—*e.g.*, puerile breathing —with the results of disease. The compensatory changes in the unaffected lung are often marked in children, and this harsh and intense breathing may be mistaken for bronchial breathing. The mistake is, however, easily avoided by attention to the character of *the expiration*, which in bronchial breathing has its peculiar quality well marked, while in harsh 'compensatory' breathing the expiration is uncharacteristic.

Delay in the development of the physical signs seems somewhat frequent in children. The commonest difficulty in connection with the diagnosis of pneumonia in children is the differentiation of the disease from pleurisy. This is sometimes difficult, as bronchial breathing is often well marked over the affected area in the pleural effusions of childhood. Attention to the state of the vocal fremitus when it can be obtained, the outline and limits of the dull area, the position of the apex-beat of the heart, and the history and general symptoms, will usually prevent error.

(*d*) *Prognosis and Mortality.*—Croupous pneumonia is a much more benign disease in the child than in the adult. Baginsky observed 11 deaths in 246 cases (4·5 per cent.). Holt collected 1,295 cases with a mortality of 39—*i.e.*, 3 per cent. Death from pneumonia in children is generally due to some complication, or to extensive involvement of the lungs.

PLEURISY.—'Pleurisy in children,' says Henoch, 'differs in no essential particular from the same disease in later life.' Yet some points require special attention in the child. The difficulties of diagnosis are greater in the child than in the adult, and the symptoms and physical signs are occasionally misleading. Let us consider the following points:

(*a*) *Causation.*—Pleurisy is more often associated with pneumonia or one of the infective fevers—scarlet fever, typhoid fever, measles, or influenza—in the child than in the adult, while the connection of pleurisy with tuberculosis is probably less close in the early years of life than it is during adolescence.

(*b*) *Mode of Onset.*—An acute mode of onset with considerable pyrexia and severe symptoms is relatively frequent in the child, but, on the other hand, an insidious onset is also common in the early years of life.

(*c*) *Nature of the Symptoms.*—Vomiting, epileptiform convulsions, and other symptoms of cerebral irritation are relatively frequent in children.

(*d*) *Nature of the Physical Signs.*—Bronchial breathing and bronchophony over the area of the fluid are the rule in children, but in most cases the bronchial breathing is weak and 'distant' in quality. It may, however, be loud and insistent.

Percussion should be lighter in the case of the child than in the adult. Deep percussion may bring out a comparatively resonant note in the effusions of childhood. Sometimes the breathing has the normal character, but is simply a little weak. 'Good, though deficient, vesicular murmur,' says Goodheart, 'may be present all over the side which is full of fluid, and unless this is remembered there is likely to be a mistake in diagnosis.' The exaggeration of the breath sounds on the healthy side is marked.

(*e*) *Character of the Effusion.*—Pus is much commoner in the child than in the adult. Goodheart found pus in 78 out

of 149 cases of pleural effusion. Loculation of the fluid is common. Serous effusions are often less abundant than purulent effusions.

(*f*) *Course and Event.*—The general course of the disease does not differ much from what is found in the adult. Pus, if promptly diagnosed and thoroughly treated, admits generally of a good prognosis in the child. Considerable deformity of the chest is a not uncommon result of pleural effusion in childhood. These deformities often undergo considerable subsequent amelioration.

PHTHISIS.—Tuberculosis of the lungs is more frequent in infancy than in later childhood. On the whole, the disease is not very frequent in childhood. Baginsky gives the following figures: Of 3,575 deaths from phthisis in Berlin in the course of one year the proportion amongst children was as follows:*

From birth to 1 year	...	...	95 cases.
1st to 2nd ,, ...	...	...	89 ,,
2nd to 5th ,, ...	...	...	91 ,,
5th to 10th ,, ...	...	...	38 ,,

It will thus be seen not only that the general death-rate from tuberculosis of the lungs is relatively low in children, but that the rate rapidly declines after the second year.

In infants and young children the invasion of the bacilli is generally by way of the bronchial glands. Meningitis is frequent after the second year. Beginning with the third year, tuberculosis of the bones, cervical and mesenteric lymph nodes, peritoneum and intestines, becomes more frequent, and in any of these organs it may occur as the principal lesion, although at autopsy the lungs, even at this age, are rarely found free of infection.'†

In the first few years of life the pulmonary lesions are

* A. Baginsky, *op. cit.*, p. 699.

† L. E. Holt, *op. cit.*, p. 1076.

more widely diffused throughout the lungs, and the limitation of the tubercular process to the apical regions is less marked than in later life. The tendency to miliary tuberculosis is greater in the child, but the most usual form of pulmonary tuberculosis in the early years of life is the broncho-pneumonic, while the tendency to excavation is relatively less marked. The typical fibro-caseous excavating type of phthisis, which is the rule in the adult, becomes common in childhood only after the second dentition. 'Tuberculous broncho-pneumonia is the most frequent and most characteristic form of tuberculosis in infants and young children, and it is the one which at this age usually causes death. In this form of disease there are produced in the lung caseous nodules, or larger caseous areas, some of which have usually undergone softening by the time the case comes to autopsy. The process usually runs a somewhat subacute course. With the lesions mentioned there are always associated those of simple broncho-pneumonia. The pleura is involved in nearly every case. There may be simply dense connective-tissue adhesions which bind the lung firmly to the chest wall, or the pleura may be greatly thickened and contain caseous deposits. Occasionally empyema is seen, but it is almost always sacculated and small. Both lungs are usually affected, but one to a much greater degree than the other. There are found large areas of consolidation, which sometimes involve an entire lobe, but more often areas are seen in several lobes. These portions of the lung appear much firmer and harder than in ordinary pneumonia. The upper lobes are more often affected than the lower, and especially that part of the lobe which is near the root of the lung, on account of its frequent association with tuberculosis of the bronchial glands. The disease very often extends forward from this point to the middle lobe of the right, or the corresponding part of the left, lung. . . . Areas of excavation large enough to deserve the name of cavities were

present in thirty-five of seventy-two autopsies upon tuberculous patients two years old and under. They are found in the great majority of the cases in which continuous pulmonary symptoms have been present till death.'*

The following summary of the characteristics of pulmonary tuberculosis in the first few years of life may be given:

(*a*) *Pathological Characters.*—The process is broncho-pneumonic in type and subacute in character; the lesions are usually widely diffused throughout the lungs, and both lungs are early involved; limitation of the process to the apical region is less frequent than in the adult; the portions of the lungs in the neighbourhood of the roots are frequently involved; the tendency to excavation, although marked, is less pronounced than in the case of the adult; fibroid change is less frequent than in the adult; the bronchial glands are much involved; extension of the tubercular process to the meninges of the brain is relatively common; involvement of the pleura is relatively common; secondary affections of the intestines are less frequent in infancy than in older children; involvement of the bones is common; enlargement of the spleen is not infrequent.

(*b*) *Character of the Symptoms.*—In general the symptoms of pulmonary tuberculosis in the child are more marked and more rapidly progressive than in the adult. Fever, wasting, and debility are usually pronounced. Loss of appetite, sweatings, diarrhœa, acceleration of the pulse and respirations, are commonly pronounced. Hæmoptysis is rare. Sputum is often absent. The temperature less often takes on the typical hectic type in the child than in the adult, and, on the whole, is less characteristic and less trustworthy as a guide to diagnosis. The chronic, stationary, non-febrile type of disease, which is common in the adult, is uncommon in the child.

(*c*) *Character of the Physical Signs.*—The physical signs are

* L. E. Holt, *op. cit.*, pp. 1077, 1078.

such as the morbid anatomy of the disease would lead us to expect. Areas of moist consonating or crepitant râles are present, usually in the upper lobes or about the roots of the lungs. Dulness is not usually very pronounced. Definite signs of excavation are rarely present in children under three years of age, though cavities are often found at the autopsy on such cases. The cracked-pot sound is not a trustworthy sign of cavity in children. Compensatory harsh breathing may be pronounced, and must not be confused with bronchial breathing.

(*d*) *Course and Termination of the Disease.*—The course of pulmonary tuberculosis is almost uniformly unfavourable in young children, when once the disease has become definitely recognisable. The more acute cases run a course of a few weeks; the more chronic cases may last from six to twelve months, but not often longer.

Diagnosis of Pulmonary Tuberculosis in Young Children.—This is often difficult for various reasons: the distinction from broncho-pneumonia may be for a time impossible;* sputum, which helps us so decisively in the case of the adult, is often absent in the child; hæmoptysis, which is the most significant of all the symptoms, is usually wanting in the tuberculosis of children; the physical signs are often equivocal, and definite clinical evidence of excavation is, in many cases, absent. Hence much caution in diagnosis is advisable. The following rules may be found useful:

(*a*) In a case of prolonged debility with fever and wasting occurring in a young child, enquire carefully for other possible explanations, especially the presence of pus—*e.g.*, in empyema, for bone or joint disease, rickets, glandular disease, Hodgkin's disease, hereditary syphilis.

(*b*) Attach due weight to family and personal history, and to the mode of onset of the disease.

(*c*) Note carefully the precise localization of the physical

* See p. 342.

signs, and whether they tend to advance in any characteristic manner or to recede.

(*d*) If possible, obtain and examine the sputum. In quite young children who do not spit, a plan has been adopted with success of passing the stomach-tube, and examining the bronchial mucus which commonly adheres to it on withdrawal.

(*e*) Attach much weight to the general progress of the case, and the response, or absence of response, to treatment.

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THE END

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