CHAPTER VI

THE LUNGS AND PLEURÆ

Owing to the extreme vascularity of the lungs, it is not surprising that inflammation of these organs is a frequent complication of acute general diseases. Thus, inflammation of the lungs is one of the commonest accompaniments of the acute specific fevers and other infective disorders. Overcrowding in badly ventilated rooms increases the risk of infection from persons who have tubercle bacilli in their sputum.

PART A. SYMPTOMATOLOGY.

The **Cardinal Symptoms** of diseases of the lungs are **cough, breathlessness, expectoration,** and sometimes **pain in the chest** and **hæmoptysis.** The general symptoms are pyrexia, sweating, emaciation, and debility. The heart, especially on its right side, suffers sooner or later in all serious or prolonged pulmonary diseases owing to interference with the pulmonary circulation.

§ **101.** Concerning **Cough,** if it is attended by expectoration (as in 1 to 4 below), it points to definite changes either in the lungs, bronchi, or upper respiratory passages. If, without expectoration (as in 5 to 9 below), it may point to simple congestion of the throat or larynx, to the presence of pleurisy, to the early stage of some pulmonary disorder, or to some source of reflex irritation. The *Causes of Cough* are as follows:

1. The commonest form of cough is the recurring WHEEZY cough, attended by expectoration, so typical of bronchitis.

2. PAROXYSMS of coughing followed by vomiting occur in *whooping-cough* and advanced *phthisis*. *Bronchiectasis* may be attended by paroxysmal cough, with purulent expectoration; so also is the rupture into a bronchus of an empyema, or of a lung or liver abscess. Paroxysmal cough, usually without expectoration, may occur with enlarged bronchial glands and other *mediastinal tumours*: also during attacks of asthma. [See also 8 on page 147.]

3. The HAWKING cough of throat affections is very characteristic, and is met with in catarrhal *pharyngitis*, especially in cigarette smokers, also in chronic laryngitis. It also occurs in *nervous* subjects. A similar type of cough may be found in cases of chronic nasal catarrh associated with *infection of the accessory nasal sinuses*, especially the maxillary antrum. In the latter case a constant hawking cough, with occasional severe paroxysms, is often present, and is relieved only by drainage of the antrum.

4. The IRRITABLE cough, most marked in the early morning and on going to bed, is especially associated with *early phthisis*. There may or may not be much expectoration.

5. A NIGHT cough may be due to chronic congestion of the pharynx, which is sometimes associated with a *long uvula.*

6. The long BARKING or nervous cough of *hysteria* is very characteristic. It is unattended by expectoration.

7. The SHORT SUPPRESSED cough associated with *pleurisy* is so characteristic as to be almost diagnostic; it is unattended by expectoration unless pneumonia is present.

8. The GANDER OR BRASSY cough associated with *aneurysm* and other *mediastinal tumours* is typical, and when once heard is readily recognised.

9. The REFLEX cough, due to irritation in the area of the vagus nerve, may be caused by (i.) *gastro-intestinal* disorders, such as dyspepsia, constipation, diarrhœa, or worms in children; (ii.) *pericarditis;* (iii.) *carious teeth;* and (iv.) *ear* troubles, such as impacted wax; (v.) *abdominal* disease with irritation of the diaphragm—*e.g.*, by subphrenic or liver abscess.

The *Diagnosis* of these varieties of cough is important in practice, since they arise from, and may be seen in, affections other than those of the lungs. When a short dry cough is set up by going into the cold, it may be due to pharyngeal congestion or irritation. In simple throat affections the cough comes on in paroxysms, especially after talking. On the other hand, if such a cough comes on in a warm atmosphere, we should suspect phthisis. In chronic irritation of the larynx or trachea the cough is often worst in the early morning, when a paroxysm is induced by the effort to bring up a little glairy mucus. The face is congested, there is difficult inspiration and even vomiting.

The *Treatment* of cough depends upon the cause, but, in general terms, irritable coughs should be treated by Tr. opii camphorata, by a linctus of squills and tolu, by bromides, by heroin $\frac{1}{12}$ to $\frac{1}{6}$ gr., or codeine gr. $\frac{1}{2}$, or by various medicated lozenges, such as the B.P. morphia and ipecacuanha, or krameria and cocaine lozenge. Coughs associated with tenacious sputum, which is difficult to expectorate, should be treated, not by sedatives, but by alkaline mixtures, with or without the addition of small doses, *e.g.*, gr. 3 of potassium iodide, to loosen the sputum.

§ 102. Breathlessness, or dyspnœa, is another symptom of lung affections. The causes of breathlessness are dealt with in more detail in the symptomatology of cardiac disorders (§ 26). The types of breathlessness special to respiratory disorders are:

1. Breathlessness attended by SNIFFING and NASAL BUBBLING is caused by *nasal* or *naso-pharyngeal catarrh.* The obstruction in the nose or mouth in these conditions may cause considerable stertor at night-time.

2. STRIDULOUS respiration, in which the stridor attends both inspiration and expiration, is caused by obstruction in, or *pressure* upon, the trachea or larynx. It is accompanied in severe cases by drawing in of the epigastrium and lower costal cartilages during inspiration (§§ 171, 176 and 177).

3. Dyspnœa attended by considerable WHEEZING or rhonchi in the chest is characteristic of *bronchitis*, accompanied usually by emphysema.

4. CONTINUOUS dyspnœa may be a prominent symptom of gross disease in the chest, such as a large *pleural effusion* : in this the embarrassment of respiration is mainly due to the resulting displacement and pressure on the heart. The dyspnœa associated with extensive *fibrosis of the lung* from any cause, or with *collapse* following *obstruction of the bronchus*, *e.g.* by malignant disease, may be considerable : dyspnœa is often one of the chief symptoms in the last mentioned, and is often disproportionate to the physical signs of disease present. Under this heading comes the expiratory dyspnœa of *emphysema* (§ 142).

5. A rapid respiration with altered PULSE-RESPIRATION RATIO is very suggestive of *pneumonia*. Especially in children there is seen in this disease a characteristic working of the alæ nasi.

6. PAROXYSMAL dyspnœa is present in asthmatic attacks, but is often an indication of *cardiac disorder* (§ 27).

7. The SUDDEN and URGENT dyspnœa of *spontaneous pneumothorax* is a dramatic clinical phenomenon (§ 126).

§ 103. Pain in the Chest is usually present with affections of the pleura but otherwise it is not a constant symptom in pulmonary disorders. The various causes of pain in the chest are enumerated in § 33. The following are the chief types of pain met in diseases of the lungs :

(i.) The SHARP, cutting, stitch-like pain of *pleurisy*, before the effusion separates the inflamed surfaces, is greatly aggravated by drawing a long breath. This is undoubtedly the commonest of the pulmonary causes of pain in the chest, and this symptom in *pneumonia* indicates involvement of the pleura. It must be remembered, however, that in some *sub-diaphragmatic diseases—e.g.*, of the liver, spleen, or colon—pain is also felt on deep inspiration. One of the most intense forms of pain in the chest is due to *diaphragmatic pleurisy*. It is referred along the lower costal margin, occasionally to the tip of the shoulder, and is accompanied by very shallow respirations, chiefly or entirely thoracic ; sometimes there is hiccough. The pain of diaphragmatic pleurisy is occasionally abdominal, when it may cause difficulty in diagnosis, since it may suggest the presence of acute abdominal disease. (ii.) A SORENESS behind the upper part of the sternum attends the onset of *acute tracheitis* and *bronchitis*. (iii.) SUDDEN severe pain, followed by considerable pulmonary distress and general collapse, occurs with the onset of *pneumothorax*. (iv.) SUDDEN pain, attended by hæmoptysis, marks the occurrence of *embolism* of the lung or rupture of an aneurysm into the lung. (v.) *Cancer* of the lung may or may not be accompanied by pain, according to its proximity to the pleura or other sensitive structures. (vi.) *Mediastinal tumours*, including aortic aneurysm (§ 80), give rise sooner or later to pain in the chest. (vii.) Slight pain in the upper intercostal spaces is a frequent symptom in early phthisis : a similar pain is also found in some cases of lung abscess, the site varying according to the position of the abscess.

The presence of **expectoration** or **sputum** is an important sign; its physical appearance may lead to the diagnosis of certain lung diseases. It must be examined by the physician, and it is therefore described in § 111. It must be remembered that children usually swallow sputum; as also do adults with bad habits. Expectoration from the pharynx must not be mistaken for expectoration from the bronchi or lungs. The amount of coughing required to void the sputum may aid diagnosis—*e.g.*, in the early stages of bronchitis much coughing brings up a little tenacious sputum, in the later stages moderate coughing brings up much frothy muco-purulent sputum.

§ **104. Hæmoptysis** means the spitting of blood (αἷμα, blood; πτύω, I spit), but the term is confined to the expectoration of blood from the organs of respiration. A distinction should be drawn between the expectoration of blood-stained sputum, a common phenomenon in many general diseases attended by severe coughing, and the coughing up of actual free blood; a clear understanding on this point is important in diagnosis, and also in treatment.

The *fallacies* with regard to this symptom are very important. When a patient comes with a history of having "brought up blood," it may at first be difficult to determine whether the blood has come from the stomach, from the lungs, or from the upper respiratory passages (nose, throat, etc.). In the so-called hysterical hæmoptysis, small quantities of thin reddish fluid containing red corpuscles are coughed up; its source is usually the gums or the mucous membrane of the cheeks or pharynx. Although in many such cases the patient is suffering from genuine hysteria, the possibility of deliberate malingering must always be borne in mind.

The differentiation of the various forms of blood-spitting is given more fully under Hæmatemesis (§ 272), but the following points are mentioned here as being characteristic of the issue of blood from the lungs: (i.) It is not infrequently preceded and accompanied by a tickling cough (if the blood be large in quantity it may excite retching on touching the pharynx); (ii.) the patient usually continues to cough up blood for some time afterwards; (iii.) the blood has a bright red colour, is alkaline and frothy, (if very profuse, it may be darker in colour, without froth, and clots may be present); (iv.) physical signs of disease of the lungs are often, though not always, present—they may be absent in the hæmoptysis of early phthisis; (v.) the antecedent history of the patient may point to pulmonary tuberculosis or to cardiac disease, these being undoubtedly the most common causes of hæmoptysis. The above details are given for guidance; in actual practice the distinction between hæmoptysis and hæmatemesis seldom presents real difficulty if care is taken to obtain an accurate history; the descriptions of hæmoptysis given by patients themselves are remarkably constant:—"I felt something suddenly come up in my throat and it was blood."—These or similar words constitute a common statement volunteered by patients. The persistence of stained sputum for a day or two after the initial attack affords further strong

presumptive evidence of true hæmoptysis. The amount of blood expectorated at once may be slight, and the bleeding may be protracted or recurrent; or there may be copious bleeding at one time, and the attack may be fatal within a few minutes. The main causes of hæmoptysis are:

I. PHTHISIS. This is by far the commonest cause, at any rate in young adults, and it is a reasonable clinical rule that definite blood spitting in a young adult, though only to the extent of a drachm or even less, should be assumed to be due to pulmonary tuberculosis until it has been proved to originate from some other cause. The hæmoptysis of phthisis may occur either in the early or in the advanced stage of the disease; in either case it may be small or very large in amount. When the disease is advanced, and bleeding occurs from rupture of an eroded vessel, the issue may be rapidly fatal, even within a few minutes, though death seldom occurs with such suddenness as in rupture of an aortic aneurysm (*q.v.*). Tuberculosis of the lungs may be recognised by: (i.) the previous history of the patient; and (ii.) evidence of congestion, consolidation or excavation of the lung. Nevertheless, the most careful physical examination may fail to reveal any obvious signs; sputum tests and especially X-ray examination are often necessary before the presence of tuberculosis can be ruled out.

II. MITRAL STENOSIS. Here the pulmonary blood pressure is raised and the lungs are congested owing to relative stagnation of blood therein. The patient's cardiac condition is usually known before the hæmoptysis, but in some cases hæmoptysis may be the first symptom which occasions a visit to the doctor. An erroneous diagnosis of phthisis has often been made in these circumstances, even in the absence of tubercle bacilli from the sputum, and when characteristic cardiac signs are present.

III. In BRONCHIECTASIS there may be considerable hæmoptysis. As in phthisis, this may occur at any stage of the disease, but is a prominent and frequently severe symptom in the so-called "dry bronchiectasis" in which, between the attacks of hæmorrhage, the patient may be free from symptoms and physical signs, and only X-ray examination (after the introduction of iodised oil into the bronchi) reveals the cause (see § 143).

IV. In PRIMARY MALIGNANT DISEASE of the bronchus there may be hæmoptysis, seldom severe, except in the advanced stages; even then much hæmorrhage is uncommon. Repeated small hæmorrhages in a patient of middle-age, or especially one of more advanced years, should raise the suspicion of malignant disease in some portion of the respiratory tract. *Adenoma* of the bronchus may be the cause of recurrent hæmoptysis.

V. Various PULMONARY DISEASES other than phthisis may be attended by expectoration of blood of varying amount. In *acute tracheo-bronchitis* the sputum may contain streaks of blood from time to time; in *spirochætal bronchitis* the hæmorrhage is likely to be much more definite and pronounced. In *pneumonia* blood in the sputum is a frequent characteristic, the amount varying with the type and bacteriology of the disease. The rusty sputum which is such a diagnostic feature of the pure pneumo-

coccal lobar pneumonia differs from the expectoration of some of the acute broncho-pneumonic conditions following influenza, especially those associated with a hæmolytic streptococcus; in these the whole lung becomes sodden, and almost pure blood may be coughed up in large quantities throughout the acute stage. In *chronic bronchitis* with emphysema the sputum may at times be blood streaked. *Gangrene*, *abscess*, *sporotrichosis* and other *fungi* infections, and *hydatid* disease may cause bleeding. *Pulmonary distomatosis* is the cause of so-called endemic hæmoptysis in Japan.

VI. § **105. Pulmonary Infarction** is commonly caused by embolism of one of the branches of the pulmonary artery, but may also be due to a primary thrombosis occurring in the pulmonary vessels. Some have suggested that it may be caused by hæmorrhage into the alveolar spaces from rupture of the pulmonary blood-vessels, hence the expressions "*hæmorrhagic infarction*" and "*pulmonary apoplexy*." Pulmonary infarction complicates mitral disease, and in fact any form of chronic heart failure. It may occur in malignant endocarditis and also in association with septic venous thrombosis, *i.e.*, as a complication of an infective thrombo-phlebitis in any part of the body. It may be seen after abdominal operations, and especially major pelvic operations, and occasionally after an operation for empyema. It is also known to occur after childbirth. Small emboli may cause few clinical signs, beyond some pain in the chest. Where a large infarct has been formed, hæmoptysis may be considerable, and pain and respiratory distress severe. In such cases it is common to find at a later date the physical signs of localised pulmonary consolidation, and a friction rub is often audible, owing to the development of a localised dry pleurisy.

When large thrombi are dislodged from distant parts and travel to the lung, the patient may die suddenly at the moment of impaction of the clot in a main branch of the pulmonary artery. In such cases there may be no premonitory symptoms, even the existence of a clot being unsuspected. Post-mortem examination may reveal a large embolus plugging the main branches of the pulmonary artery near its bifurcation.

Treatment. Absolute rest must be insisted on. Morphia may be necessary if the patient is very restless, and oxygen if cyanosis is present. Circulatory stimulants may be required.

VII. Rupture of an ANEURYSM into the trachea or bronchus is usually followed by immediate death, the preceding hæmoptysis being of the most dramatic and appalling character, though in some cases there may be a considerable leakage going on for a day or two before the final issue (§ 80). Apart from such instances of fatal hæmoptysis, a slight degree of blood-spitting is common; hence the occurrence of occasional mild hæmoptysis, if associated with a history of sub-sternal pain, should raise suspicion as to the presence of an aneurysm.

VIII. ULCERATION in some part of the upper respiratory tract (throat, larynx, trachea, etc.). In rare circumstances bleeding from this source

may be considerable, but as a general rule hæmoptysis due to local ulceration or nævi of the upper respiratory passages is small in amount, but apt to be recurrent. The diagnosis depends upon thorough and complete investigation of the respiratory tract, and may necessitate bronchoscopic examination. It should not be too readily concluded that hæmoptysis is due to local causes in the throat; the diagnosis of varicose veins of the pharynx is seldom justified or substantiated, and patients who have been told that their blood-spitting originates from enlarged veins at the back of the throat are usually found on subsequent investigation to be suffering from phthisis or some other serious organic disease.

IX. Purpura, erythræmia, Ayerza's disease, hæmophilia, scurvy, leukæmia, and some other BLOOD CONDITIONS may be attended by bleeding from the lungs. These causes are rare, but when present are usually recognised, though at first they may not be obvious. Hæmoptysis has been recorded in some of the eruptive fevers.

X. CARDIO-VASCULAR and RENAL DISEASE. Hæmoptysis occurs in subjects of *arterial* and *renal* disease. Hæmorrhages from bronchial arteries have been described in cases of chronic interstitial nephritis. It is certain that hæmoptysis may occur in patients with a *high systemic blood pressure*, associated with a condition of essential hypertension, in whom no serious disease of the lungs exists. Though this cannot be described as common, it is perhaps less rare than is supposed.

XI. VICARIOUS MENSTRUATION as a cause of hæmoptysis has been alleged by some. It is recognised by its occurrence in association with the menstrual period, usually shortly before this is due, normal menstruation being absent or greatly diminished, and by the absence of evidence of disease in the chest. It must be insisted that vicarious menstruation, though a genuine phenomenon, is a rare cause of hæmoptysis; this diagnosis should not be an excuse for failure to carry out the most complete investigation in cases of hæmoptysis of uncertain origin.

Differential Diagnosis. Although the cause of hæmoptysis may in many instances be obvious on ordinary clinical examination, in certain cases it can only be determined after prolonged, even elaborate, investigation. First, careful inquiry into the history should be made. Next should come examination of the chest by the usual clinical methods, and bacteriological examination of sputum when present. If these measures fail to establish the presence of definite disease of the heart or lungs, X-ray examination must be insisted on; the introduction of iodised oil into the lower respiratory tract may or may not be necessary. If it is still impossible to substantiate the presence of a lesion which will account for the bleeding, the respiratory tract may require further examination by the laryngoscope and/or the bronchoscope. Not until after such complete investigation, and the exclusion of the commoner organic diseases, should it be assumed that the hæmoptysis is due to one of the rarer causes above mentioned in small print.

The *Prognosis* depends upon the cause. Hæmoptysis must be regarded as a serious symptom, at any rate in the first place, and a patient should never be informed that it is of little or no account until complete investigation has elicited not only the cause of the symptom, but the relative severity of the underlying disease. The hæmoptysis of phthisis is of importance chiefly from a diagnostic standpoint; its prognostic signifi-

cance is less obvious, and even in cases of active tuberculosis of the lungs the occurrence of hæmorrhage should not *per se* be taken as necessarily indicating a grave outlook. It may indeed be the first symptom in an early case. It must be regarded as an indication for complete and exhaustive examination by modern methods, the ultimate prognosis depending on the evidence thus acquired as to the patient's condition as a whole.

Treatment. (*a*) For *profuse hæmorrhage* immediate treatment is necessary. It is usually best to allow a semi-recumbent position with the head turned a little to one side to allow more comfortable breathing and greater freedom for coughing and expectoration of the blood. A hypodermic injection of morphine, gr. $\frac{1}{4}$, is the most efficacious remedy for immediate administration in severe cases; it quietens the patient, who is frequently alarmed and restless. Morphine is by no means always necessary, and should not be prescribed as a routine measure. Since the alveoli and bronchioles are usually full of blood, it is wise to give atropine, gr. 1/100 to gr. 1/50, in combination with morphia. The administration of an extract of blood platelets by intramuscular injection (hæmoplastin or coagulen Ciba), in combination with intravenous injection of 20 c.c. of a 10 per cent. solution of calcium gluconate, is a useful measure which appears to check and sometimes to stop the hæmorrhage in serious cases: 10 c.c. of 1·0 per cent. freshly prepared solution of Congo-red (intravenously) has often proved of value—to avoid reactions it must be passed through a fine filter. Where the hæmorrhage is due to extensive tuberculosis of the lung and if it be definitely known that it is coming from one side, collapse of the affected lung by artificial pneumothorax may be the only effective remedy. In these circumstances one must introduce a much larger amount of air (perhaps up to 1,500 c.c. altogether, or even more) than is ordinarily given in the course of pneumothorax therapy; such a procedure has obvious disadvantages, which can only be disregarded in view of the extreme urgency of the situation. (*b*) When hæmoptysis occurs in *small quantity*, calcium gluconate may be tried, the best plan being to give the drug intravenously as in the more severe cases. The oral administration of calcium salts appears to have little appreciable effect. Intramuscular injections of emetine, gr. $\frac{1}{2}$ every six hours, are sometimes useful. Occasionally a large dose of liquid extract of ergot, *e.g.* ℥ 120 diluted with water, is followed by dramatic cessation of the bleeding. The hæmorrhage of congestion due to cardiac disease should not be checked, unless excessive, as it relieves the pulmonary congestion.

PART B. PHYSICAL EXAMINATION.

Examination of a patient suffering from disease referable to the organs of respiration includes: (*a*) an accurate history; (*b*) careful physical examination of the chest; (*c*) examination of the upper respiratory passages; (*d*) X-ray examination of the chest; (*e*) pathological examination of

sputum, blood, pleural fluids, etc.; (*f*) bronchoscopic examination. It is not suggested that X-ray examination and/or instrumental examination of the respiratory tract are necessary in every case, but in view of the development of modern technique and of the increasing part played by radiological and instrumental methods in the diagnosis and treatment of chest disease, it is well that emphasis should be laid on the limitations of unaided physical examination. Physical signs, although valuable as part of the total evidence, can only give information as to relatively gross structural changes. In many cases diagnosis and treatment are possible only after careful synthesis of the facts supplied by all the above-mentioned methods of examination. The history usually gives a fair guide as to the extent of examination required; time spent in obtaining an inclusive account of the symptomatology is not wasted, and often avoids unnecessary multiplication of special tests.

The physical examination of the lungs is carried out by means of Inspection, Palpation, Percussion, and Auscultation.

§ 106. Inspection.—The inspection of the chest must be carried out in a good light, and the patient must be instructed to stand or sit erect, or, if in bed, to lie flat and evenly, and to breathe deeply. After noting the movements from the front, examine the back, then look from behind over the clavicles in order to make out the slighter distortions or inequalities of the chest. By inspection we note (1) the rate and character of the breathing; (2) the position of the apex beat (§ 39); (3) the shape and size of the chest. The chief landmarks of the chest are mentioned in § 38. Posteriorly the chest is divided into the suprascapular, scapular, and infrascapular regions. The scapular region is divided, by the scapular spine, into the infra- and supra-spinous regions. The names sufficiently indicate the positions of the various regions.

(1) *Rate and Character of the Breathing.*—The rate varies normally from 15 to 20 per minute, or one-fourth the rate of the pulse; any change in this proportion, or pulse-respiration ratio, should be observed. Notice whether the breathing is rapid, slow, shallow, or irregular. The respiration should be counted without the patient's knowledge; thus while counting the breathing, it is a good plan to feel the radial artery as if you were examining the pulse. Both sides of the chest should move equally. *Any diminution of movement of any part of the chest points to disuse of the underlying lung from disease*, whether new (pleurisy and pneumonia) or old (fibrosis or collapse). Instead of the normal concavity of the intercostal spaces there is flattening or convexity when pleural fluid is present. Drawing in of the interspaces on both sides during inspiration is indicative of some interference with the free entry of air into the lungs (inspiratory dyspnœa), as in diphtheria or other cause of obstruction of the larynx or trachea. The grunting expiration of broncho-pneumonia in children should be especially noted. It is convenient also at this stage to observe whether the alæ nasi are moving. *Cheyne-Stokes'* breathing is a peculiar rhythmical irregularity of breathing (see § 28). When movement of the

chest causes pain, as in pleurisy, or when the muscles of the chest wall are paralysed, there is abdominal breathing. When the diaphragm is out of action, as in certain abdominal conditions, there is exaggerated heaving of the thorax and noisy respiration.

(2) The *position and character of the apex beat* gives an important clue to many diseases of the lungs. Thus effusion and pneumothorax displace it to the opposite side; fibrosis or collapse draw it over to the same side; emphysema masks it.

(3) *The Shape and Size of the Chest.*—A cross-section of the *healthy* adult chest gives almost the form of an ellipse, the longer diameter being from side to side. In the child it is more circular in shape. The chest should appear symmetrical, although in reality the right side is slightly larger than the left. There should be no marked hollowing anywhere; the clavicle should form only a moderate prominence. The circumference of the chest varies with the height of the individual, but it should average about 34 to 35 inches for a man 5 feet 6 inches in height. With deep inspiration it should expand about $1\frac{1}{2}$ to 2 inches or more.

The categoric association of certain alterations in the shape of the chest as a whole with particular diseases can hardly be maintained with such emphasis as is often found in the older text-books. The long-narrow, so-called "*phthisical*," chest is by no means always indicative of phthisis, which may occur in a chest of normal shape. The "*rachitic*" chest, with its vertical parasternal grooves and horizontal Harrison's sulcus, is now rarely seen. The *barrel* chest is still common, with its upper ribs crowded together, the lower ribs further apart than normal and the epigastric angle unusually wide. This is traditionally attributed to emphysema, but the association between the two conditions is open to dispute. More important than the general shape and cross-section of the chest are any irregular or *asymmetrical abnormalities*, among which the student should look for *hollowing*, *prominence*, or *contraction*.

(*a*) *Localised Hollowing* or "flattening" of the infraclavicular region may indicate tuberculosis of some standing or any other disease in which fibrosis and lung contraction occur.

(*b*) *Undue Prominence* on one side of the ribs anteriorly may be due to: (i.) Scoliosis—*i.e.*, lateral curvature of the spine, the convexity of the chest being in the opposite direction. (ii.) Intrathoracic tumours, including aneurysm, effusion, abscess, or air (pneumothorax) in the chest. (iii.) If the cardiac region be prominent, it may be the result of cardiac disease in early youth, before the ribs were fully developed. (iv.) An enlarged liver or spleen, abdominal tumour, or abscess may also cause a bulging of the lower ribs on the right and left sides respectively. (v.) Subcutaneous emphysema or œdema, a localised deposit of fat or other tumour. (vi.) Localised muscular over-development, as in athletes.

(*c*) *Contraction* of an *entire side* of the chest may be due to (i.) fibrosis of the lung from whatever cause; (ii.) a previous empyema; (iii.) pulmonary collapse.

During inspection of the lungs it is convenient to note at the same time if there is any abnormal pulsation in the aortic region (see aneurysm), also to note the state of the veins in the neck, the presence of cyanosis of the face and hands, or clubbing of the fingers.

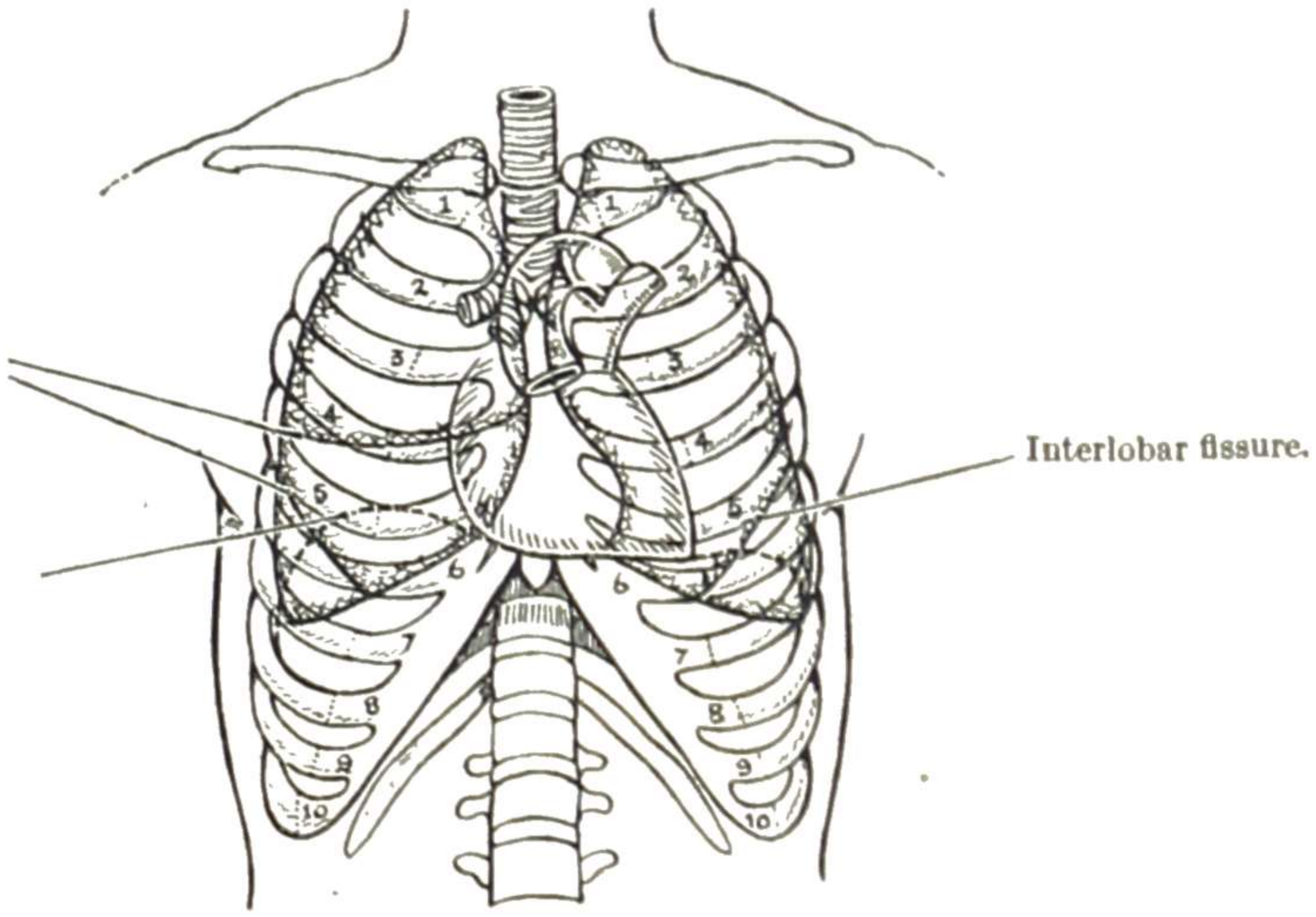
§ **107. Palpation** is the next step in the routine examination of the lungs. The position of the apex beat should be confirmed. The amount of movement with respiration is estimated better by palpation than by inspection. This test is important in the diagnosis of consolidation at one apex, and in the detection of fluid, tumour, or other cause of deficient activity of one lung or part of a lung. By palpation *Vocal Fremitus* (V.F.), or the vibration of the voice, can be felt. It is scarcely appreciable in women or children with high-pitched voices, but is marked in the adult man. The normal V.F. is slightly greater at the right than at the left apex. Towards the bases it is less intense but almost equal. This test is of the greatest value in differentiating solid and fluid. Thus the V.F. is *increased* where there is consolidation of the lung, as in pneumonia or phthisis, whereas it is *diminished* or absent when the lung is separated from the chest wall by fluid or air, or when air is not entering the larger bronchi, as in obstruction of a bronchus.[1] Not only is the V.F. a valuable differential sign, but its degree of diminution is a useful measure of the *amount* of fluid present in cases of pleural effusion. In bronchitis the rhonchi can be felt, *rhonchial fremitus;* and in pleurisy and pericarditis *friction* may be distinctly felt by the hand. A broken rib, a pointing empyema, subcutaneous emphysema, and external tumours are made out by palpation.

§ **108. Percussion** is, after palpation, the next step in the examination of the chest. Begin at the apex and percuss *alternate sides* at exactly corresponding points in order *to compare the healthy and unhealthy sides*, and thus work gradually downwards. Place the first or second finger *firmly* and *flat* against the chest, in a horizontal position. Then strike upon it with the tip of the second finger of the right hand. The blow should come from the wrist, not the elbow, and should be short and sharp. Except for the percussion of deep structures (*e.g.*, the dome of the liver) heavy percussion should not be employed.

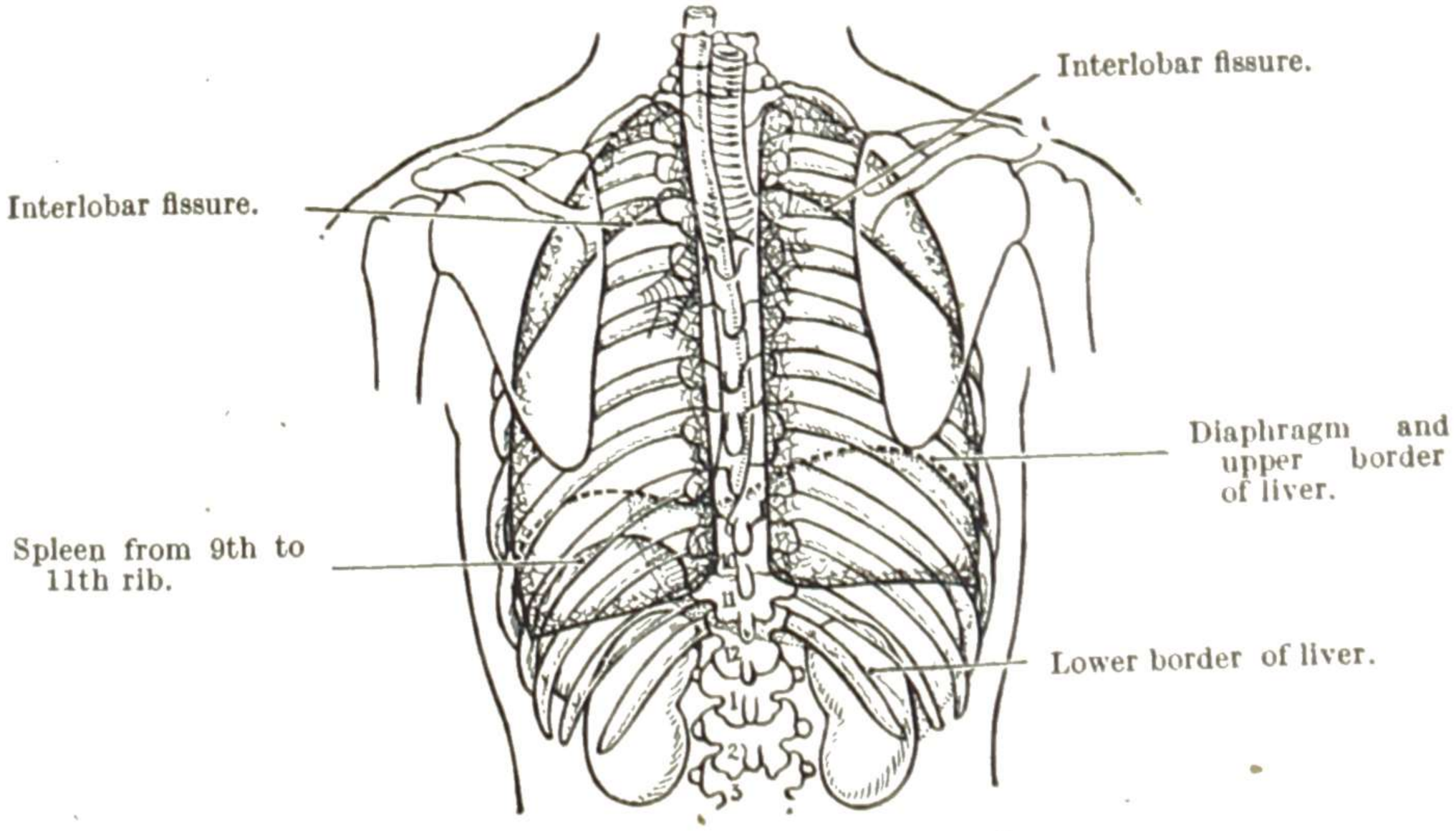
When examining the *back* of the chest (Fig. 41), the patient should be instructed to cross his arms and bend a little forward so that the scapulæ are drawn out of the way. The normal resonance of the lung extends posteriorly to the upper border of the eleventh rib on the right side, and to the lower border of the eleventh rib on the left side. On deep inspiration the resonance extends over an inch lower, and during deep expiration

[1] The absence of vocal resonance on auscultation over a pleural effusion is due to the fact that the lung, being displaced by the fluid, is usually above the level at which the chest-piece of the stethoscope is applied. It is true that water conducts sound better than air when the source of the sound and the auditory reception apparatus are both below the water level. In this case the source of the sound (viz. the larynx) is entirely outside the fluid, and the vibrations are conducted via the trachea and bronchi through the compressed lung to the chest wall.

over an inch higher. Owing to the thickness of the scapular muscles the note over the scapulæ may be markedly impaired in muscular people. For examination of the *sides* of the chest the patient should be told to put his hands on the top of his head.



(*a*) ANTERIOR VIEW OF CHEST.



(*b*) POSTERIOR VIEW OF CHEST.

FIG. 41.

The normal pulmonary note can only be learned by practice and experience, and the student should *frequently practise first on normal chests*, so as to accustom himself to the normal resonance.

The normal percussion note is resonant. It is *dull* or flat when the lung tissue is solid, as in pneumonia; or when the chest contains fluid,

as in pleural effusion, or with a thickened pleura or a tumour. When a note is said to be dull, it means that the pitch is raised and the volume of the note diminished. Between a dull note and one that is resonant there are all stages of *impairment*. The percussion note is *hyper-resonant*, sometimes tympanitic, whenever the lung tissue is unduly open—*i.e.*, too full of air, as in emphysema, or sometimes when there is air in the pleura (pneumothorax). When one part of the lung is floating above a pleural effusion (which compresses the lower part of the lung), the percussion note is unduly resonant. This kind of resonance is called *Skodaic resonance*; and it may be very resonant and tympanitic (drum-like) in character, somewhat resembling the note normally obtained over the stomach.

Increased Resistance is another quality which can be observed in the process of percussion as above described. It is greatest over fluid, but is present also in consolidation. This sign is used especially by those whose auditory appreciation is imperfect. Subtle differences cannot be appreciated by this means.

§ **109. Auscultation.**—In auscultation there are four things to be observed: (*a*) The intensity and the quality of the respiratory murmur (R.M.); (*b*) the relative length of inspiration and expiration; (*c*) the presence of adventitious sounds within or outside the lungs; and (*d*) the voice sounds or vocal resonance (V.R.).

(*a*) The normal CHARACTER OF THE BREATH SOUNDS—*i.e.*, the vesicular or "respiratory murmur" (R.M.), should be listened to in healthy chests as often as possible. Away from the apex and larger bronchi it has a soft whiffing character. The important feature of vesicular breathing is the absence of any appreciable pause between the inspiratory and the expiratory phases. The R.M. is normally very loud in children, and when a loud R.M. is met with *in adults*, it is called "*puerile* breathing." The breath sounds audible over the right apex are more pronounced than on the left side because of the presence of the eparterial bronchus on this side (cf. p. 162, fallacies no. 9). The difference varies with the age and the build of the patient. The breath sounds are bronchial [1] when the lung is solid, as in tuberculosis, pneumonia, or collapse from any cause. In this condition the sound produced in the glottis is conveyed down the bronchi and smaller tubes direct to the ear, owing to the increased conductivity of the solid tissue. In pleural effusion bronchial breathing is sometimes heard, especially near the angle of the scapula; the effusion causes a collapse of the lung so that the bronchial quality present in the nearest bronchus is well conducted to the surface (Fig. 49). *Bronchial* breathing can be heard *normally* by listening over the upper segment of the sternum, or near the fourth dorsal vertebra at the back. It has two main features: (i.) inspiration and expiration are of approximately equal length

[1] The terms "bronchial" and "tubular" are sometimes taught as synonymous; it is more accurate to say that there are three kinds of bronchial breathing—high-pitched, or tubular; medium-pitched, or true bronchial breathing; and low-pitched, or cavernous breathing.

and character, or the expiratory phase may be obviously prolonged; (ii.) there is an appreciable interval between the inspiratory and the expiratory phases. One important thing to realise about bronchial breathing is that its recognition depends on quality rather than quantity of sound—there is usually less sound with a diseased lung; the R.M. also is weak over thickened pleura or fluid. *Cavernous* respiration is low-pitched bronchial breathing, and is heard when the sound produced in a dilated bronchus or cavity is conveyed to the surface. Cavernous respiration is normally heard over the trachea. *Amphoric* breathing is a sound like air entering a bell-jar, and is sometimes heard over pneumothorax or a very large cavity.

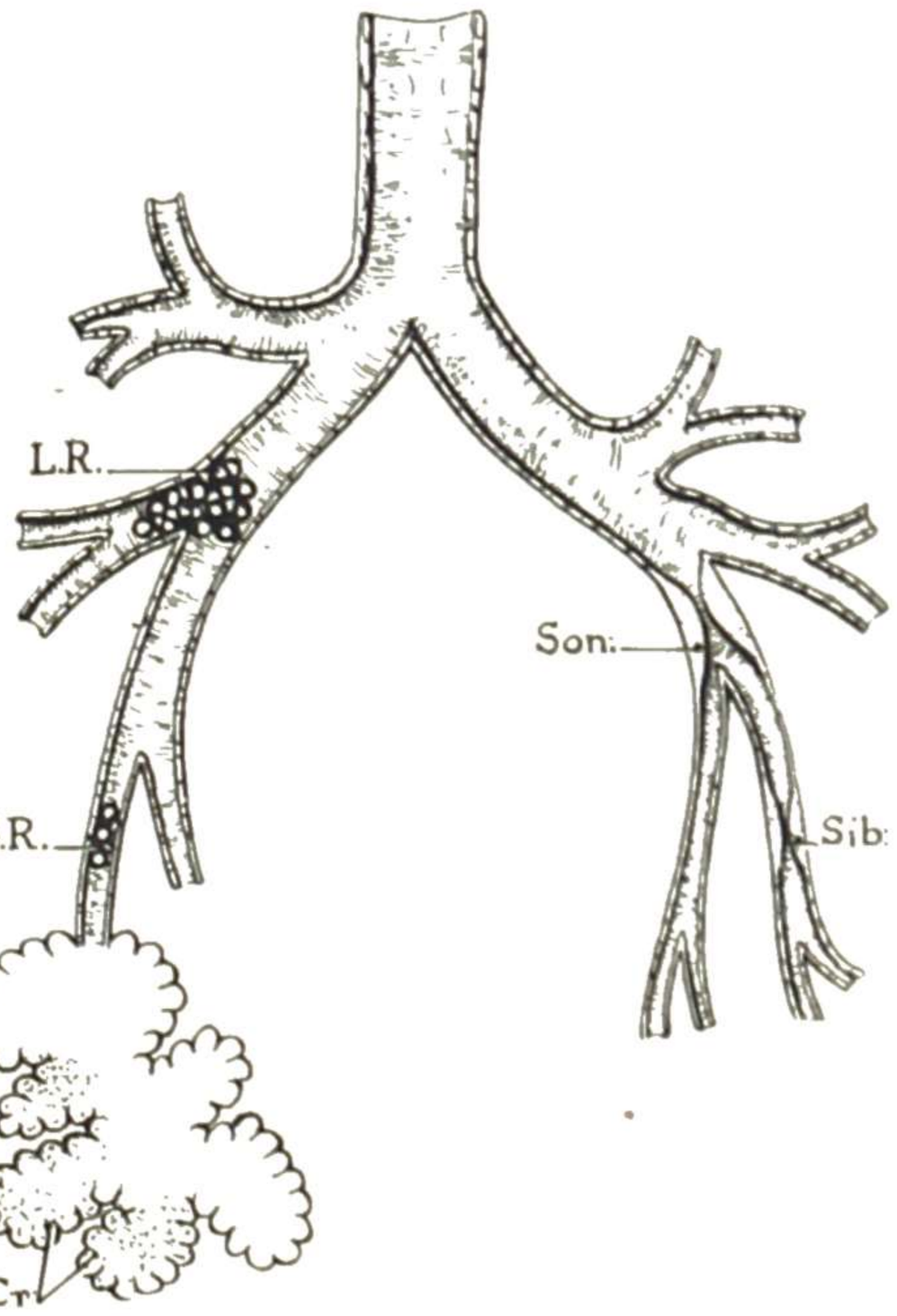


FIG. 42.—Diagram to show the production of **râles** (moist sounds) on left, by mucus in bronchial tubes, and **rhonchi** (dry sounds) on right, by narrowing of the tubes. **Moist sounds, Crepitations,** may occur in vesicles of lungs near base.

L.R. Large bubbling râles, S.R. Small râles, Cr. Fine râles (crepitations), Son. Sonorous rhonchi, Sib. Sibilant rhonchi.

(*b*) Heard through the stethoscope, the inspiratory sound is normally three times as long as the expiratory sound, which follows it without a pause. The *process* of expiration is much longer than inspiration, but through the stethoscope most of the former is unheard because the velocity of the air-current is low. *Expiration is prolonged* in any disease which involves a loss of elasticity of the lung tissue, such as emphysema, or an increase in conductivity, as in consolidation.

(*c*) The presence or absence of ADVENTITIOUS SOUNDS has next to be noted. These may be either dry or moist.

DRY SOUNDS.—(i.) *Pleural friction* is produced by the two inflamed

and roughened surfaces of the pleura rubbing together. The sound has been likened to the creaking of leather. It is generally heard both in inspiration and in expiration, is often intensified by pressure with the stethoscope and by deep breathing, and is not abolished by coughing. (ii.) Within the respiratory passages *rhonchi* may be added to the respiratory murmur. These are continuous sounds due to narrowing of the bronchial lumen, either by swelling of the mucosa, *e.g.* in bronchitis, or by spasm of the bronchial muscle, as in asthma. When low-pitched they are described as *sonorous* (produced in the larger tubes), when high-pitched as *sibilant* or *whistling* (when the smaller tubes are concerned).

Moist Sounds.—These are known as *râles*, and are due to the presence of mucus or other fluid in the bronchial tubes. Râles are of three main varieties,[1] according to the size of the tubes involved and the amount of fluid present, viz. large or bubbling, medium, and fine râles. When the finest tubes are affected the alveoli do not escape, and the finest râles, often known as *crepitations*, are due to the opening up during inspiration of alveoli, the walls of which have been kept in apposition by a thin layer of moist secretion. They have been likened to the sound produced by the rustling of tissue-paper near the ear. When these added sounds are few and difficult to detect, they may become clearer when the patient draws a deep inspiration immediately after a slight cough (post-tussic râles). Crepitations sometimes resemble friction sounds, but are distinguished by being audible only during inspiration, and by being altered by coughing.

(*d*) The voice sounds, or vocal resonance (V.R.). (i.) When the patient speaks, the vocal resonance is increased (*bronchophony*) if the conductivity of the lung substance is rendered greater by consolidation, such as that produced by tuberculosis or pneumonia. If this be so great that even whispered words are distinctly conducted, it is known as *whispering pectoriloquy*. (ii.) The vocal resonance is diminished when a layer of fluid or air intervenes between the lung and the chest wall (*e.g.*, in pleural effusion and pneumothorax), or when there is a thickened pleura. Nevertheless, in a pleural effusion, at the upper level of the fluid, the higher tones of the voice sounds are sometimes conducted, and have been likened to the bleating of a goat (hence called *Ægophony*). Transference of the voice sounds depends upon patency of the bronchi; in any condition therefore in which there is gross obstruction of the bronchus or its main divisions, *e.g.*, by a growth, the vocal resonance is diminished or lost.

The Coin or Bell sound is a sign of some value. To elicit this, a large coin is laid flat on the chest and is tapped by another coin; the mouthpiece of the stethoscope is placed some distance away, but not on

[1] Various terms have been used to distinguish different types of râles, *e.g.* consonating, clicking, crepitant, and so forth. These are a matter of the personal factor of the individual auscultator, and are apt to confuse the student. The essentials of classification are contained in the comparatively simple division just described.

the same rib or over the stomach. A bell sound is pathognomonic of pneumothorax.

Clinically, all the diseases of the lungs may be conveniently divided into those with **dulness on percussion,** those in which the percussion note is **normal,** and those in which it is **hyper-resonant.** Those with **dulness** may be subdivided into two groups—those in which the dulness is due to CONSOLIDATION, and those in which it is due to FLUID. The clinical features by which solidification of the lung is distinguished from fluid in the chest are so important that they are given in tabular form.

TABLE V.—DIFFERENTIATION OF SOLID LUNG FROM FLUID IN THE CHEST.

	Consolidation of Lung.	Pleural Effusion.
INSPECTION.	Movement impaired. May be flattening over the part (if infraclavicular region).	Movement impaired. May be bulging (of intercostal spaces).
PALPATION.	V.F. INCREASED.	V.F. DIMINISHED or absent.
PERCUSSION.	Resonance impaired.	Absolutely dull over liquid.
AUSCULTATION.	BREATHING BRONCHIAL.	R.M. ABSENT OR WEAK.
	V.R. INCREASED.	V.R. DIMINISHED.
HEART	In normal position (pneumonia), or pulled towards affected side (fibrosis or collapse).	Displaced to the opposite side.

Fallacies in Diagnosis of Diseases of the Chest.—This list includes the most important fallacies, but it is impossible to make it exhaustive.

1. When the chest wall is very thin the sounds heard on auscultation are proportionately loud. The percussion note is also more resonant, and it is consequently easy to fall into the error of supposing that emphysema is present. In children the breath sounds are always more distinct than in adults, and are, moreover, more readily conducted, so that adventitious sounds having their origin on one side may even be heard quite plainly on the other.

2. A chest wall with excess of muscular development, subcutaneous fat or œdema will give rise to error if it be not borne in mind that the sounds on auscultation and percussion are alike deadened and indistinct. The sounds heard over the scapular region are always less distinct than those heard elsewhere. When a patient does not breathe deeply, owing to enfeeblement or pain on movement of the chest, or when the chest wall is very fat, the breath sounds may be almost inaudible.

3. The presence of much hair on the chest, as it is rubbed by the stethoscope, gives rise to sounds like fine crepitations.

4. The sounds in the subcutaneous and fascial tissues round the shoulder joint often lead to mistaken diagnosis of pleurisy at the apex (scapular creak).

5. Care should also be taken to hold the chest piece firmly and flat on the skin.

6. It is well to remember that dulness on percussion does not necessarily mean that there is fluid or consolidation present. It may also be caused by thickened pleura and by a tumour. The latter may be outside the chest, but pushing up into the thorax—*e.g.*, hepatic or splenic enlargement, subdiaphragmatic abscess.

7. Tumours of the chest wall will sometimes lead to the impression that there is some difference in the size of the two sides of the thorax, and this difference may be referred to some morbid condition of the chest contents. The swelling caused by subcutaneous emphysema or blood clot, both of which may follow an accident, gives

rise to a faint crepitation which may be easily mistaken for the signs of injury to the lung beneath.

8. When one lung has been long out of action, as in fibroid phthisis, the other undergoes compensatory enlargement and encroaches on the affected side of the chest. The hypertrophied lung gives rise to sounds identical with those of emphysema.

9. The breath sounds are better heard and the percussion note is higher at the right than at the left apex, owing to the presence of the eparterial bronchus on the right side. The area over which the bronchial quality can be detected is also larger.

10. Atrophy of the muscular tissues about one shoulder leads to an apparent flattening on that side very like that seen in phthisis.

11. Dextro-cardia is very rare, but it is necessary to be on one's guard lest it be rashly supposed that the heart is displaced by effusion or by some tumour.

12. Finally, it is well to remember that the presence of lung signs usually found in association with acute disease must always be interpreted with due regard to the constitutional condition and co-existing signs of disease in other organs.

13. Distension of the abdominal organs, as in meteorism, may extend high up into the chest and simulate hyper-resonance of the lungs. This is especially probable when the lungs have been drawn up with adhesions or fibroid contraction or when one half of the diaphragm is paralysed.

14. **Hernia of the Diaphragm** is rare and often unsuspected. Usually congenital in origin, it occurs chiefly through the left half of the diaphragm, so that the stomach, small intestine, colon, omentum or spleen may become intrathoracic.

The chief *symptoms* are pain, often in the left hypochondrium or left shoulder-tip, coming on during or after a meal, with a feeling of extreme distention which is relieved by vomiting or by eructation. Other symptoms include dyspnœa, palpitation, dysphagia, and those of intestinal obstruction. The *physical signs* include hyper-resonance in the lower third of the left chest (often mistaken for a pneumothorax), cardiac displacement and cyanosis. Symptoms and signs may be entirely absent. The true state of affairs can only be diagnosed by X-ray screening, and with a barium meal or barium enema.

§ 110. Radiology of the Chest.—X-ray examination often reveals the presence of disease in the lungs when other methods of examination give a negative result. Its value is, perhaps, best exemplified in the case of early tuberculosis, indubitable evidence of which may be furnished by a radiogram when the most careful and competent clinician has been unable to detect any abnormal physical signs in the chest. Good radiological work is essential for the diagnosis of many cases of bronchiectasis and of new growths of the lungs and bronchi. Not only in diagnosis is X-ray important; in treatment, and especially in thoracic surgery, it is a *sine qua non*. For ideal work in a difficult case the investigation should be carried out by physician, surgeon and radiologist co-operating in a team. For practical purposes the physician must often interpret his own radiograms. Examination with the fluorescent screen is an important preliminary; by this means it is possible to determine any abnormality in the movements of the diaphragm and to observe various other features which cannot be demonstrated by radiography alone. The ordinary straightforward X-ray film may suffice to show the variations from the normal which characterise the commoner forms of intrathoracic disease; many conditions cannot be adequately shown without the use of iodised oil. When iodised oil (a preparation of 40% iodine suspended in poppy-seed oil, with which it is in loose chemical combination), is introduced into the

bronchial tree, the latter is rendered opaque to X-rays and can be outlined in a radiogram (bronchogram). Detailed information is thus afforded, *e.g.*, as to the existence of cylindrical or saccular dilatation of the bronchial tubes (bronchiectasis), as to the exact size and position of cavities, sinuses, etc., in the lung, and as to bronchial obstruction due to new growths. A *Barium Meal* examination may be useful for the investigation of new growths pressing on the œsophagus, or in cases of œsophago-bronchial fistula.

Mass Miniature Radiography has materially advanced the preventive treatment of chest disease. With a 35-mm. ciné film in a specially designed apparatus, the radiologist can examine a large number of individuals in rapid succession : these miniature films are subsequently projected on a screen, and in the enlarged image it is possible to detect definite or suspicious abnormalities. Any individual revealing abnormalities in the miniature film is subsequently X-rayed on a standard apparatus with a 15″ × 12″ film, and submitted to such clinical, bacteriological and other investigations as are necessary to establish a diagnosis :—this should not be made from the miniature film alone. In very large numbers of ostensibly healthy individuals thus examined in numerous surveys, a small but definite proportion have shown lesions of the lungs, heart or mediastinum (and see § 131).

Tomography. In a special form of X-ray apparatus, recently introduced, the tube, instead of being fixed, moves across an arc during the exposure ; a corresponding synchronous movement of the cassette containing the film takes place in the opposite direction. As a result certain of the rays pass always through one point on the film, the remainder passing through different points and giving an image which is blurred or even invisible while the fixed point image is distinct. For this reason, and as the focal point of the tube can be altered so as to centre the fixed rays at different depths of the chest, it is possible, by taking a series of radiographs, accurately to delineate various details which are not adumbrated in the ordinary flat radiograph, and to obtain more exact localisation of cavities, etc., in the lung. The tomograph has a limited sphere of application, but within that sphere its value is inestimable.

§ 111. Examination of the Sputum.[1]—Much may be learned from an examination of the sputum. First, as regards its APPEARANCE. Watery sputum is expectorated in large quantity in œdema of the lungs. If the disease be confined to a moderate catarrhal process of the bronchial tubes (*e.g.*, bronchitis), the sputum is white, clear, and frothy (" mucous expectoration "). If the process be more severe and suppurative, or if the lung tissue be breaking down, then pus is present, and the sputum is yellowish (mucopurulent). In phthisis, when the lung is breaking down, the sputum is often voided in thick purulent masses like coins, hence called *nummular*. In cases of pulmonary abscesses, tuberculous cavities, and of empyema bursting into the lung, large quantities of almost *pure pus* are expectorated from time to time. Extremely fœtid expectoration is voided in gangrene of the lungs and in bronchiectasis. In *pneumonia* the sputum is very characteristic, being (i.) almost airless and extremely viscid, so that the vessel containing it may be inverted without spilling it, and (ii.) frequently tinged with blood, thus having the colour of rust. In severe cases, and in new growth of the lung, the sputum becomes thinner, frothy, and dark red, the " prune-juice " sputum. *Casts* of the bronchial tubes, which can

[1] It is important to ensure that the specimen examined is really sputum and not merely saliva or nasal secretion.

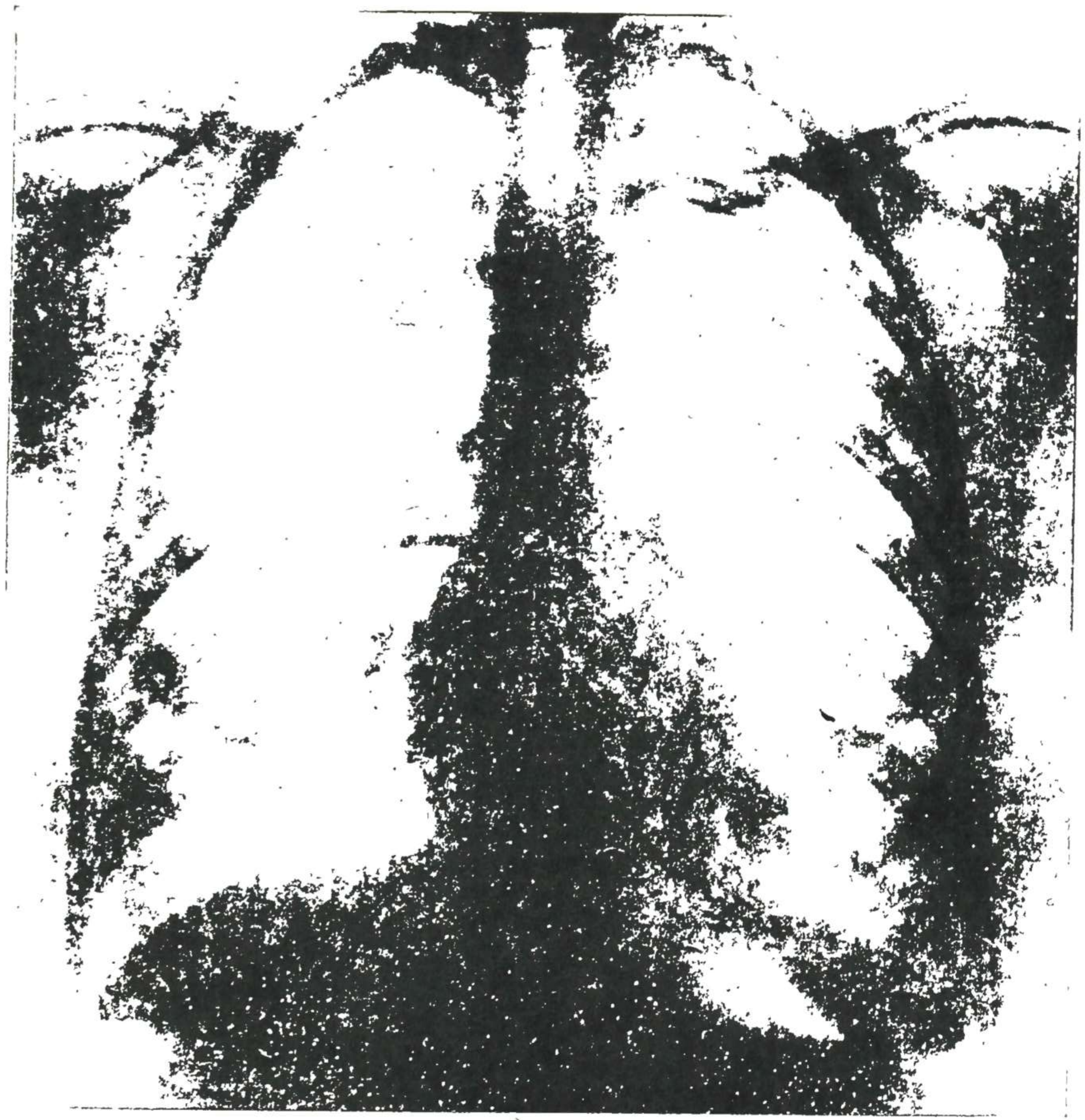


FIG. 43.—RADIOGRAM OF A NORMAL CHEST (FEMALE). The relative want of translucency in the lower zones of the lung fields is due to opacity caused by the mammary glands.

FIG. 44.—RADIOGRAM FROM A CASE OF BILATERAL BRONCHIECTASIS. The dilated bronchi on the left side have been outlined by iodised oil.

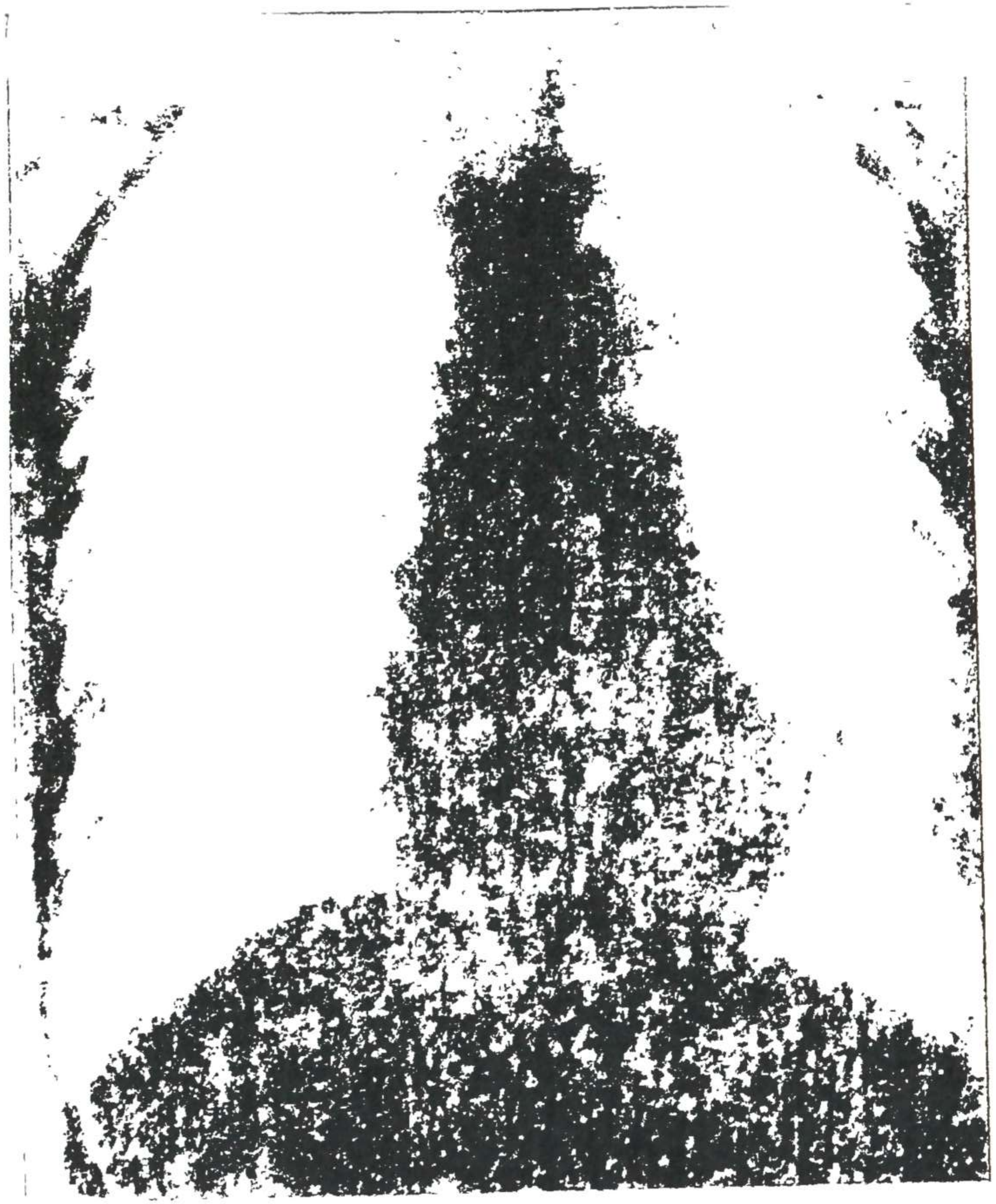


FIG. 45.—RADIOGRAM FROM A CASE OF PULMONARY TUBERCULOSIS, showing a fairly large cavity in the left upper lobe.

FIG. 46.—RADIOGRAM OF CHRONIC PULMONARY TUBERCULOSIS. Infiltration and fibrosis at both apices. A few areas of calcification in each upper lobe—especially on the left side.

FIG. 47.—RADIOGRAM FROM A CASE OF PRIMARY BRONCHIAL CARCINOMA, showing a dense opacity in the right upper zone due to collapse of the upper lobe from obstruction of the eparterial bronchus by growth.

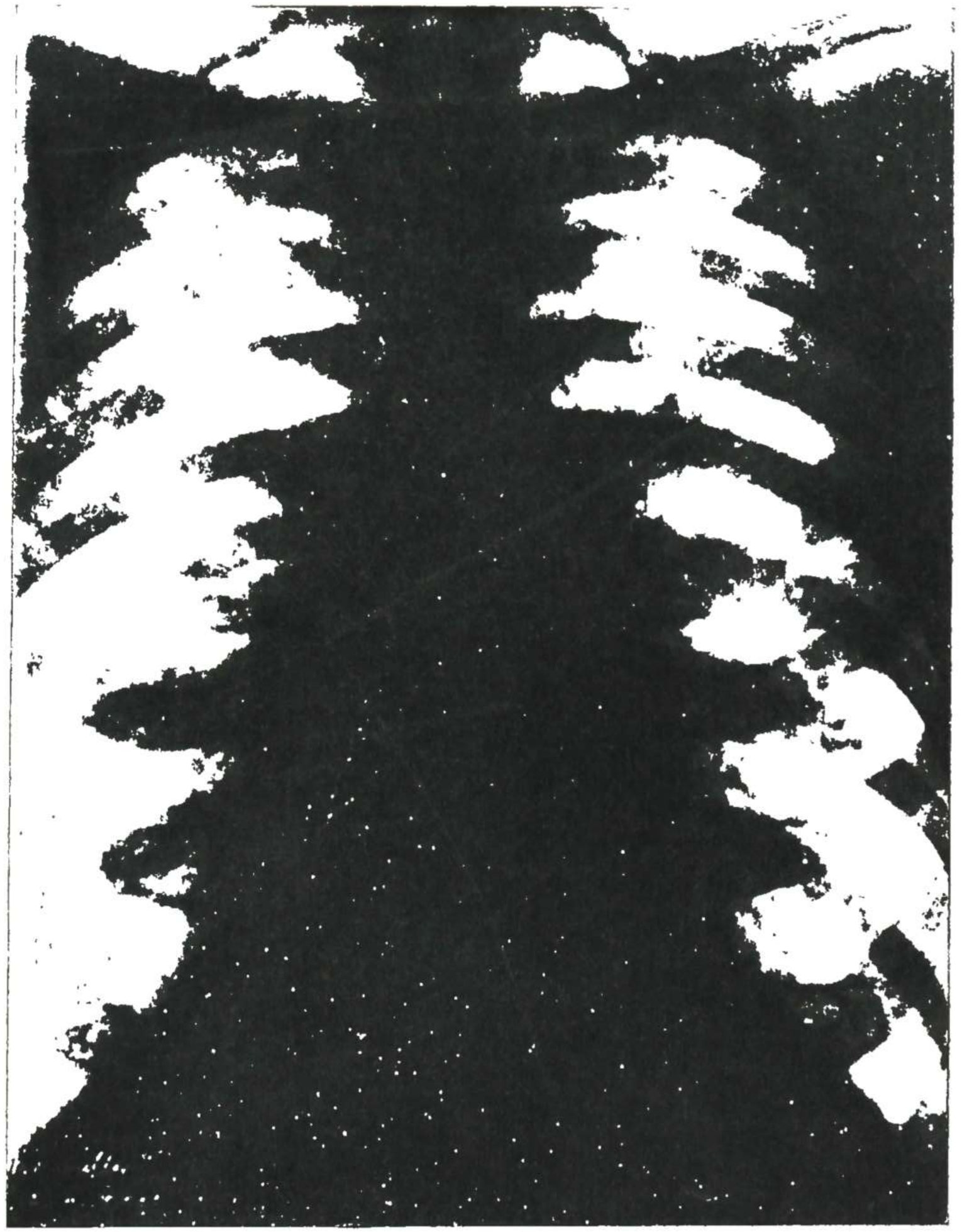


FIG. 48.—RADIOGRAM FROM A CASE OF SILICOSIS in a coal-miner, showing general reticulation of both lung fields with early nodulation.

be seen by the naked eye, are expectorated in plastic bronchitis, and occasionally in bronchial pneumonia, and shreds of membrane in diphtheria. Hydatid cysts, resembling empty gooseberry-skins, are expectorated in that rare condition hydatid disease of the lungs, or when hydatid of the liver ruptures into them. In town dwellers, and those with dusty occupations, the sputum is dark, or even black, from the presence of carbonaceous and other particles. "Anchovy sauce" coloured sputum is characteristic of abscess of the liver which has burst into the lung (§ 336).

Microscopic Examination of the Sputum.—Various *bacteria* and *fungi* (*e.g.*, tubercle, pneumococcus, influenza, pyogenic cocci, anthrax, glanders, plague, spirochætes, the fungi of actinomycosis, blastomycosis, and aspergillosis) may be found in the sputum. Various *parasites* (streptothrix, echinococcus, Distoma pulmonale, etc.) are sometimes found in the sputum. Sarcinæ and Oïdium albicans come usually from the mouth. The method of detecting these is described in Chapter XXI.

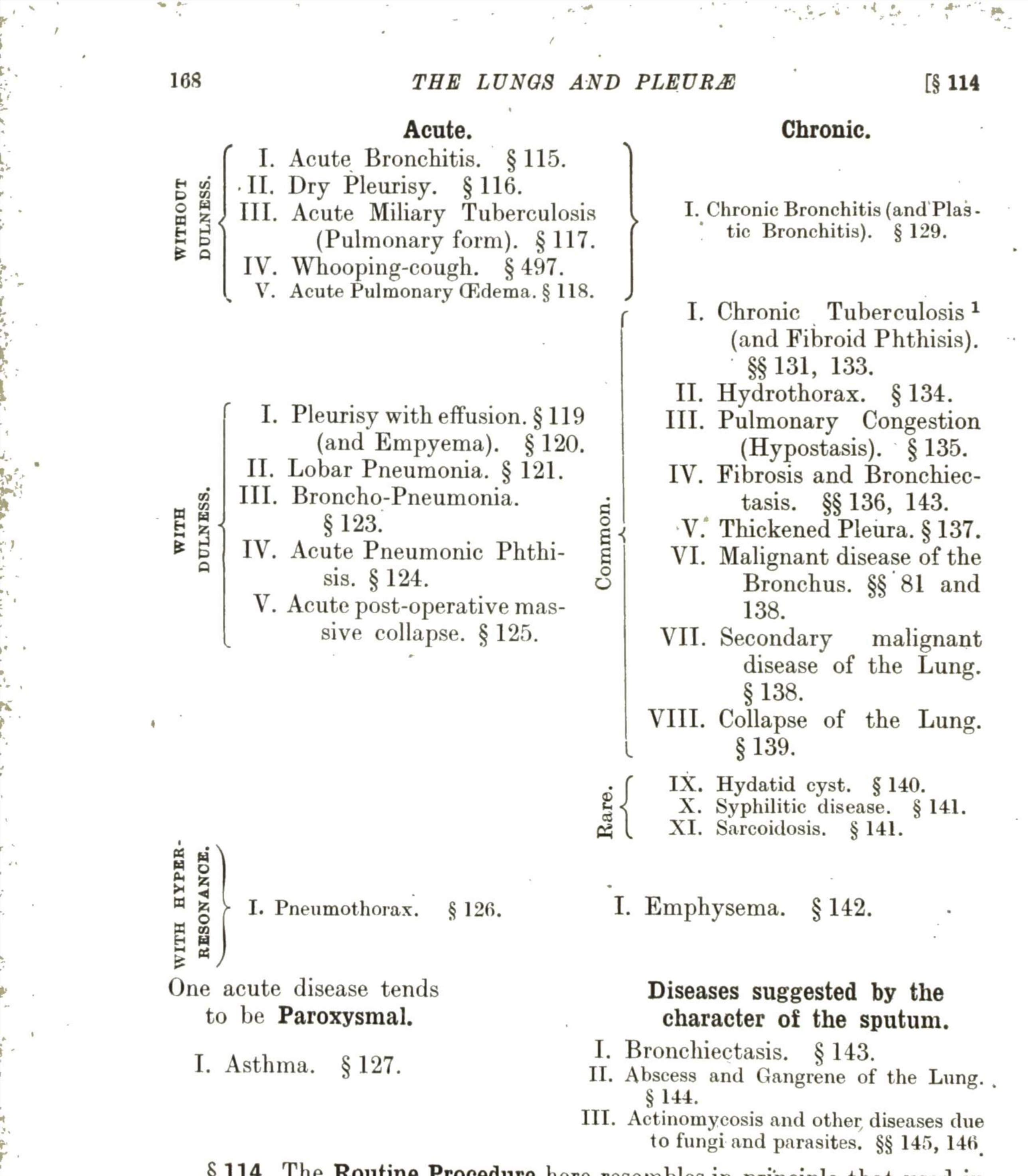
In all destructive diseases of the lung, especially gangrene and abscess, fragments of pulmonary tissue are present—*i.e.*, epithelial cells and connective tissue. The most characteristic is *elastic tissue*. Elastic fibres are best revealed by taking a small portion of the sputum and boiling it with liquor potassæ, which breaks up and renders clear all the other elements, but leaves the elastic fibres unattacked. These sink to the bottom of the test-tube, and may be withdrawn by a pipette (precautions, see Urinary Deposits) for examination under the microscope. They appear as wavy, highly refractile fibres, of uniform thickness, with square-cut ends, and are typically arranged as if surrounding an air cell.

In cases of primary new-growth of the bronchus, recent improvements in technique, introduced by Dudgeon, have rendered easier the detection of carcinoma cells in the sputum and in pleural effusions.

§ 112. Bronchoscopy is playing an increasingly important part in the diagnosis and treatment of chest diseases: the bronchoscope is now recognised as an indispensable instrument for the complete investigation of pulmonary disorders. Thus apart from those cases in which the presence of a foreign body is known or suspected, it may give vital information in an obscure case of hæmoptysis. In bronchial neoplasms the diagnosis can often be confirmed, and a biopsy undertaken. When no intra-bronchial mass is visible, broncho-stenosis due to extrinsic pressure from a neoplasm may be recognised; and a widened angle of bifurcation of the trachea due to a large lymph node infiltrated by secondary growth may decide between exploratory thoracotomy and palliative X-radiation. As a means of treatment, it may be possible via the bronchoscope to extract inspissated mucus plugging a bronchus in a patient with post-operative pulmonary collapse; and even to remove an innocent intrabronchial neoplasm (*e.g.*, adenoma).

PART C. DISEASES OF THE LUNGS AND PLEURÆ: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 113. Classification.—For practical purposes, diseases of the lungs and pleuræ may be divided into Acute and Chronic, and each of these may be subdivided into those without dulness, those with dulness, and those with hyper-resonance.



§ 114. The **Routine Procedure** here resembles in principle that used in diseases of the heart. First, *What is the patient's leading symptom?* If suffering from lung disease, his cardinal symptom will be one of those mentioned in section A: cough and breathlessness are the most common.

Secondly, follow this up with a few questions to ascertain the *history of his illness*, and especially whether *the disease be acute or chronic*. Other important points are whether the patient has been exposed to a "chill," and whether there is any "lung disease" in the family. Do not use the word "consumption"; it may frighten your patient unnecessarily.

[1] There is no dulness in the early stages of the disease.

[2] Spontaneous pneumothorax is often an acute incident in a chronic disease—tuberculosis.

Thirdly, proceed to the PHYSICAL EXAMINATION OF THE LUNGS. The routine method is as follows :

1. Ascertain whether there is any increased rate or other modification in the breathing, or any alteration in the shape of the chest (by *inspection*, and, if necessary, by measurement). Note whether any part of the chest shows decreased movement. Observe also the alæ nasi, the colour of the face and lips, and look for clubbing of the fingers.

2. Find the position of the apex beat.

3. Test the vocal fremitus by *palpation*.

4. Ascertain if there be any dulness or hyper-resonance (by *percussion*).

5. Listen to the breath and voice sounds, and then to any adventitious sounds which may be present.

6. The sputum should be inspected, and, if necessary, examined by the pathologist in further detail.

7. X-ray examination must be insisted on if necessary ; in the diagnosis of many chest diseases the physician is more and more dependent on radiological evidence.

The chest should always be stripped, and it is more convenient to examine the patient in a standing or sitting posture, if he be not too ill.

If the illness developed gradually, and is of some standing, and unattended by obvious constitutional disturbance, then turn to **Chronic Pulmonary Disorders** (§ 128).

If the illness came on recently and suddenly, accompanied by fever, quickened respiration, coated tongue, and with marked malaise, then the case is one of the **Acute Pulmonary Diseases,** below.

There is one disease of the lungs, ASTHMA, which comes on in sudden acute attacks from time to time ; it is **chronic,** with **acute exacerbations.**

Although it is convenient for the sake of classification to include asthma among the pulmonary diseases, since it manifests itself by grave respiratory distress and is often associated with bronchitis, it must be remembered that true spasmodic asthma is not properly speaking a disease of the lungs, but belongs to the group of so-called allergic diseases of which the characteristic respiratory syndrome is but one manifestation. See also under § 127 (Asthma) and § 521 (Allergy).

ACUTE DISEASES.

We now proceed to percuss the chest.

The acute diseases without alteration in the percussion note, *i.e.*, **without dulness**—excluding WHOOPING-COUGH, which is an infective disorder and has no physical signs in the lungs peculiar to it, and ASTHMA,[1] which is of a paroxysmal character—are : ACUTE BRONCHITIS ; DRY PLEURISY ; ACUTE MILIARY TUBERCULOSIS [2] ; and ACUTE PULMONARY ŒDEMA.

I. *The patient complains of a* **cough,** *with frothy expectoration, and his*

[1] Bronchitis is commonly associated with asthma.

[2] In the early phase of this malady there is no alteration of the percussion note, but as the disease progresses dulness appears.

temperature is slightly elevated; there is **no alteration** *in the percussion note but on auscultating the chest, loud* RHONCHI *are heard. The disease is* ACUTE BRONCHITIS.

§ **115. Acute Bronchitis,** or inflammation of the bronchial tubes, is certainly the most common acute disease of the lungs in this climate.

Symptoms.—The disease commences gradually in the course of one or two days, with a feeling of tightness of the chest, of soreness behind the sternum, shortness of breath, frequent cough, and slight rise of temperature, 100° to 101° F. The inflammatory process lasts from ten days to three weeks, and gradually subsides. The sputum is viscid and scanty during the first few days, and then becomes thinner, muco-purulent, and more easily coughed up.

TABLE VI.—DIAGNOSIS OF COMMON ACUTE DISEASES OF THE LUNGS AND PLEURÆ.

	Percussion Note.	Auscultation.
Acute Bronchitis . . .	Normal	R.M. and V.R. normal; Loud râles and rhonchi.
Dry Pleurisy	Normal	V.R. normal. R.M. normal, or may be diminished in intensity owing to restricted inspiratory movements due to pain; Pleural friction.
Acute Pulmonary Tuberculosis	Normal, or scattered areas of impaired note	R.M. diminished; crepitations later.
Pleurisy with effusion .	Dull	R.M., V.R. and V.F. diminished; Pleural friction at early and late stage.
Lobar Pneumonia . . .	Dull	V.R. and V.F. increased: Bronchial breathing.
Broncho-Pneumonia . .	Scattered areas of impaired note	Fine crepitations and scattered areas of bronchial breathing.

Physical Signs.—The percussion note is unaltered unless, as so frequently happens, emphysema be present also, in which case the chest is unduly resonant. On auscultation the vesicular murmur is obscured over the whole chest on both sides by loud rhonchi and moist râles (see Fig. 42) which are variable and altered by coughing. On palpation rhonchial fremitus can frequently be felt.

Etiology.—Bronchitis, which is of course microbic in origin, is generally attributed to: (i.) A chill; that is to say, sudden exposure to cold. (ii.) Sometimes, however, it is caused by spreading from laryngitis. (iii.) It frequently accompanies many of the specific fevers, especially measles, whooping-cough, and typhoid. It is so frequently present with the first and last as to constitute an aid to the diagnosis of those diseases. (iv.) Certain occupations which expose people to irritating vapours and small particles of dust predispose to acute bronchitis. Thus the cotton-mill hands and chemical workers frequently suffer from bronchitis. It is also common amongst cabmen, mariners and others who are exposed to all weathers. (v.) It is a common accompaniment of many other pulmonary diseases, though it may be a subordinate feature. (vi.) It is most frequent in childhood and old age. (vii.) A rare form of bronchitis, due to a fluke, is met with in the East (pp. 219 and 374, Table XIX).

The *Diagnosis* is not difficult in most cases, but *acute miliary tuberculosis* is at first apt to be regarded as acute bronchitis. The diagnosis of tuberculosis is aided by the greater elevation of the pyrexia in the former, and by the subsequent course of the disease. The "*capillary bronchitis*" of children is really a *broncho-pneumonia* (*q.v.*); the constitutional symptoms and dyspnœa are much more marked, there may or may not be some dulness, and the differentiation from simple acute bronchitis is not always easy.

The *Prognosis* is favourable in adolescence and adult life, and the disease usually clears up in one to three or four weeks, though it has a special liability to return, and ultimately to become chronic. It is dangerous in infancy and old age, where the resisting powers are feeble. It is one of the commonest causes of death in old age. If an attack of acute bronchitis does not begin to clear up in two or three weeks, pulmonary tuberculosis should be suspected, especially if the patient be young.

Treatment.—The indications are: (i.) During the first stage, to promote secretion; (ii.) during convalescence, to improve the general condition so as to enable the patient to throw off his liability to bronchitic attacks. The patient must be kept in a warm room at 65° F., without draughts but with adequate ventilation. At the onset give an aperient and a diaphoretic mixture, with perhaps a few grains of Dover's powder to soothe the pain. To promote the flow of secretion, warm alkaline drinks and expectorants such as ipecacuanha and antimony, together with liq. ammon. acet., are especially useful. A good mixture consists of: Sod. bicarb. gr. 10, Sod. chloride gr. 3, Spt. chlorof. ℥ 5, Aq. anisi ad fl. oz. 1. The dose should be followed by a *very hot* drink. When the secretion is free it is sometimes helpful to stop the antimony and administer expectorants, such as ammonium carbonate, syrup of tolu, senega, and squills (Formula 57). If the sputum is very tenacious, add potassium iodide to the expectorant mixtures. The patient must be confined to bed, and will derive great benefit from the inhalation of steam. In childhood this is best done by a bed canopy and a steam kettle beside it; in adults a steam-kettle on the fire will suffice. Linseed-meal poultices, cataplasma kaolini, or a turpentine stupe to the chest relieve the distressing tightness of the chest (see also Formulæ 30 and 68). The importance of chemotherapy and of oxygen therapy must not be forgotten (cf. § 121). During the stage of recovery tonics and cod-liver oil are called for.

II. *The patient complains of sharp* PAIN *in the chest on inspiration; he has a short dry cough, and his temperature is moderately elevated; on auscultation,* FRICTION *sounds are heard. The disease is* DRY PLEURISY.

§ **116. Dry Pleurisy** is inflammation of the pleura without effusion. In this disease there is a fibrinous exudation on the visceral and parietal layers of the pleura, and a tendency to the formation of adhesions, and to the effusion of fluid.

Symptoms.—The disease in some cases comes on quite suddenly with a stabbing pain in the chest, accompanied by a short dry cough. The

constitutional disturbance is seldom very great, and the patient may not necessarily take to his bed. The temperature may rise to 100° or 101° F., rarely higher. The most obvious symptom in this disease is pain in the chest, usually affecting one side, and characterised by being greatly increased on deep inspiration and by coughing. The pain is caused by the contact of the inflamed pleural surfaces, and is usually, though not necessarily, located over the diseased part. For the distribution of pain in diaphragmatic pleurisy, see § 103.

Physical Signs.—On inspection one side of the chest is seen to be limited in movement. Percussion reveals nothing abnormal. On auscultation, the respiratory murmur may be found to be normal, or it may be lessened, as the patient endeavours to restrain the movements of the chest on account of the pain so caused. At a very early stage a pleural rub is heard over one side, often most marked near the angle of the scapula (compare § 109). Sometimes the inflammation undergoes resolution, sometimes it goes on to effusion. As effusion takes place, the pain and pleural friction disappear.

Etiology.—(i.) It may occur as a complication of some acute infective disease, such as measles, scarlatina or influenza. (ii.) Inflammation may extend from disease of the underlying lung, such as pneumonia, tuberculosis, cancer, and infarction, or from adjacent organs, such as the liver or spleen. (iii.) Undoubtedly a large number of cases of pleurisy are tuberculous in origin, especially if recurrent; this fact should always be remembered. Acute pleurisy, with or without effusion, occurring in a young adult, should be regarded as tuberculous in origin, unless it can be definitely proved to be due to some other infective agent.

The *Diagnosis* from *fibrositis* (pleurodynia) may be difficult. Local injection of the muscles with 2 per cent. procaine solution will decide the point. In *intercostal neuralgia* there are tender points along the course of the nerve, and the pain is not aggravated by deep inspiration. Pleural friction is distinguished from the rhonchi heard in *bronchitis* by there being in nearly every case of pleurisy a distinct interval between the inspiratory and the expiratory rub. A radiogram must be taken to see if there is any other evidence of tuberculosis or other intra-pulmonary disease.

Prognosis.—It is not in itself a serious malady, and readily yields to treatment; but sometimes effusion occurs (Pleural Effusion, § 119). When this effusion becomes purulent (§ 120) the prognosis is graver. A frequent result of no great importance is thickening and adhesions of the pleura.

Treatment.—Considerable relief is derived by simply strapping the affected side of the chest, so as to limit the costal movements of respiration. For pain, the greatest relief is undoubtedly given by a linseed-meal or a kaolin poultice. Aspirin is of great service and should be used in preference to opium, though the latter may be necessary. A good combination, in cases with severe pain, is aspirin gr. 10 and Dover's powder gr. 10. Diuretics and diaphoretics are useful. In more chronic cases liniments may be helpful; infra-red radiation may be invaluable. If the con-

dition does not resolve in a few weeks, we must suspect some other cause for the mischief, such as those mentioned under pleurisy with effusion. The treatment of tuberculosis is discussed later (§ 131).

III. *The patient exhibits the signs of subacute bronchitis; but he has* SEVERE MALAISE *and a high* TEMPERATURE. *The disease is* ACUTE MILIARY TUBERCULOSIS.

§ 117. Acute Miliary Tuberculosis (acute phthisis, galloping consumption) is often part of a tuberculous process infecting the whole body, and is therefore sometimes described as the pulmonary form of acute general tuberculosis (see Fig. 121, a chart showing the typical course of the temperature). The type of fever exhibited varies in different cases; in some there is considerable remission in the mornings, in others the chart shows continuous pyrexia with little remission.

Symptoms.—The malady is of most insidious onset: there may be progressive weakness and emaciation. Some weeks before physical signs have appeared the thermometer may show the typical intermittent pyrexia so characteristic of tubercle—an evening elevation of 101° to 103° F., and a morning normal temperature. As the disease progresses the remissions are likely to be less, the fever being more of the continuous type. In some cases the inverse type is present, when the temperature is higher in the morning than in the evening. Night-sweats and cough are present, with muco-purulent expectoration. Dyspnœa, and sometimes cyanosis, develop out of proportion to the physical signs; the cyanosis may be extreme, and of itself is a very characteristic feature. Great weakness ensues, and in the third or fourth week the patient may develop the symptoms of the typhoid state or of meningitis.

The *Physical Signs* referable to the lungs are indefinite, or resemble at first those of bronchitis. At first there is no alteration in the percussion note, but by and by careful percussion discovers scattered patches of impaired resonance. Auscultation at first may give little help, but in a week or so it reveals rhonchi and fine râles over certain areas, which do not shift from place to place, as in bronchitis. Later on the râles are coarse and bubbling, and areas of tubular breathing may be found.

The *Diagnosis* in the first stage from bronchitis and broncho-pneumonia is extremely difficult. We have to rely upon the disproportionate emaciation and cyanosis, the character of the temperature, and the patchy distribution of the physical signs in tuberculosis. In other cases the malady is almost indistinguishable from typhoid fever except for the marked predominance of the pulmonary signs and the absence of the roseola; the Widal test is negative. In all stages the detection of tubercle bacilli in the sputum is one certain means of diagnosis, though their absence does not exclude acute pulmonary tuberculosis. X-ray examination of the lungs may reveal the characteristic "*snowstorm*" appearance of miliary tuberculosis.

Etiology.—The disease may occur at any age, but is commonest in infants, in young adults, and in those with a tuberculous family history. Acute general tuberculosis may originate from a primary focus, such as a tuberculous joint which had been considered cured. The disease may follow measles or whooping-cough in children.

Prognosis.—The disease is almost uniformly fatal in a few months, but occasionally the course is prolonged, even up to two years, and recovery has been recorded (cf. § 132*a*, chronic miliary tuberculosis). *Treatment* is almost entirely symptomatic.

IV. *The patient, a child, has* PAROXYSMS *of coughing which terminate in a* WHOOP, *and frequently in* VOMITING; *there is some fever, but the only signs in the lungs are those of a little bronchial catarrh. The disease is* WHOOPING-COUGH.

Whooping-cough (Pertussis) is an acute infectious disease, and it is described among the microbic disorders (§ 497).

V. *The patient is suddenly seized with acute dyspnœa and copious frothy sputum flows from the mouth and nose. The disease is* ACUTE PULMONARY ŒDEMA.

§ 118. **Acute Pulmonary Œdema.** *Symptoms.* The sudden onset of acute dyspnœa, with copious, often blood-stained (rose-coloured) and albuminous sputum, are characteristic. The face is pale and cyanosed, the expression is one of intense anxiety; there may be a cold sweat. The pulse is feeble, and there may be pain or a feeling of oppression in the chest. The disease usually depends on failure of the left ventricle, allowing fluid to accumulate in the lungs. It may arise in the course of heart disease, especially aortic disease, arterio-sclerosis, pregnancy, epilepsy, giant urticaria, acute infections, or renal disease. It is an occasional complication of lung operations, *e.g.*, thoracoplasty or lobectomy. The *physical signs* consist of râles and crepitations which are heard all over the chest.

Treatment.—Sometimes the disease is so rapidly fatal that no treatment is of avail. The best emergency treatment is immediate blood-letting to 20 ounces. Atropine has an almost specific action; $\frac{1}{100}$ gr. should be given hypodermically at the earliest possible opportunity, and in severe cases, with copious frothy sputum, should be repeated frequently: even $\frac{1}{50}$ gr. of the drug may be given every two hours until the full pharmacological effects are obtained. Oxygen, nikethamide B.P. (coramine) or other stimulants may be necessary. Mersalyl (2 c.c.) has been used with beneficial effects. The recurrence of attacks cannot be prevented except in those cases when the patient is able to foretell their coming, when atropine given in time may ward off or mitigate the attack. The only prophylactic treatment is that directed to the underlying disease. The disease may never recur, but some patients may relapse at variable intervals for years.

We now turn to the **Acute Diseases with Dulness on Percussion.**—I. PLEURISY WITH EFFUSION (Serous or Purulent); II. LOBAR PNEUMONIA; III. BRONCHO-PNEUMONIA; IV. ACUTE PNEUMONIC PHTHISIS; and V. ACUTE POST-OPERATIVE MASSIVE COLLAPSE.

I. *The patient has a* DRY COUGH, *with moderate fever and other constitutional symptoms. The lower part of the chest is* DULL *on one side, and over this area the* VOCAL FREMITUS *and* RESONANCE *are* DIMINISHED *or* ABSENT. *The heart is displaced towards the healthy side. The disease is* PLEURISY WITH EFFUSION.

§ 119. **Acute Pleurisy with Effusion.**—When describing acute Dry Pleurisy (§ 116) it was pointed out that the disease may undergo resolution or result in adhesions. It may also go on to effusion.

Symptoms.—There is usually a history of a more or less acute onset with pain (§ 116), but as the disease progresses, and the surfaces of the pleura are separated by fluid, pain becomes less and less marked. Occasionally the onset is insidious, and a considerable amount of fluid accumulates in the pleural cavity without any history of initial pain. The patient suffers from general malaise, and finds it difficult to lie on the sound side, because the action of the healthy lung is thereby impeded. A degree of breathlessness may be present, but even with a large amount of fluid this is not invariably a prominent feature.

Physical Signs (see Fig. 49).—(i.) Inspection may show diminished movement, fulness or bulging of the chest wall. (ii.) On palpation, the vocal fremitus is found to be diminished or absent over the fluid, and there may be bulging of the intercostal spaces. (iii.) Percussion reveals

absolute dulness over the areas of the fluid. Above the level of the fluid, if the lung be otherwise healthy, there is a hyper-resonant note (Skodaic resonance). (iv.) On auscultation over the fluid, the breath sounds are absent; the vocal resonance is greatly impaired or lost. Bronchial breathing may occur.[1] At the upper margin of the fluid posteriorly—perhaps just about the angle of the scapula—only certain overtones of the voice are transmitted, and they produce, therefore, a sound like the bleating of a goat (ægophony). (v.) When the effusion is large it causes displacement of the heart, which may be very considerable. The amount of fluid present may be estimated by the degree of cardiac displacement, and of respiratory distress.

The *diagnosis* of pleurisy in its earlier stages is referred to under Dry Pleurisy. The differentiation of the physical signs of fluid in the chest,

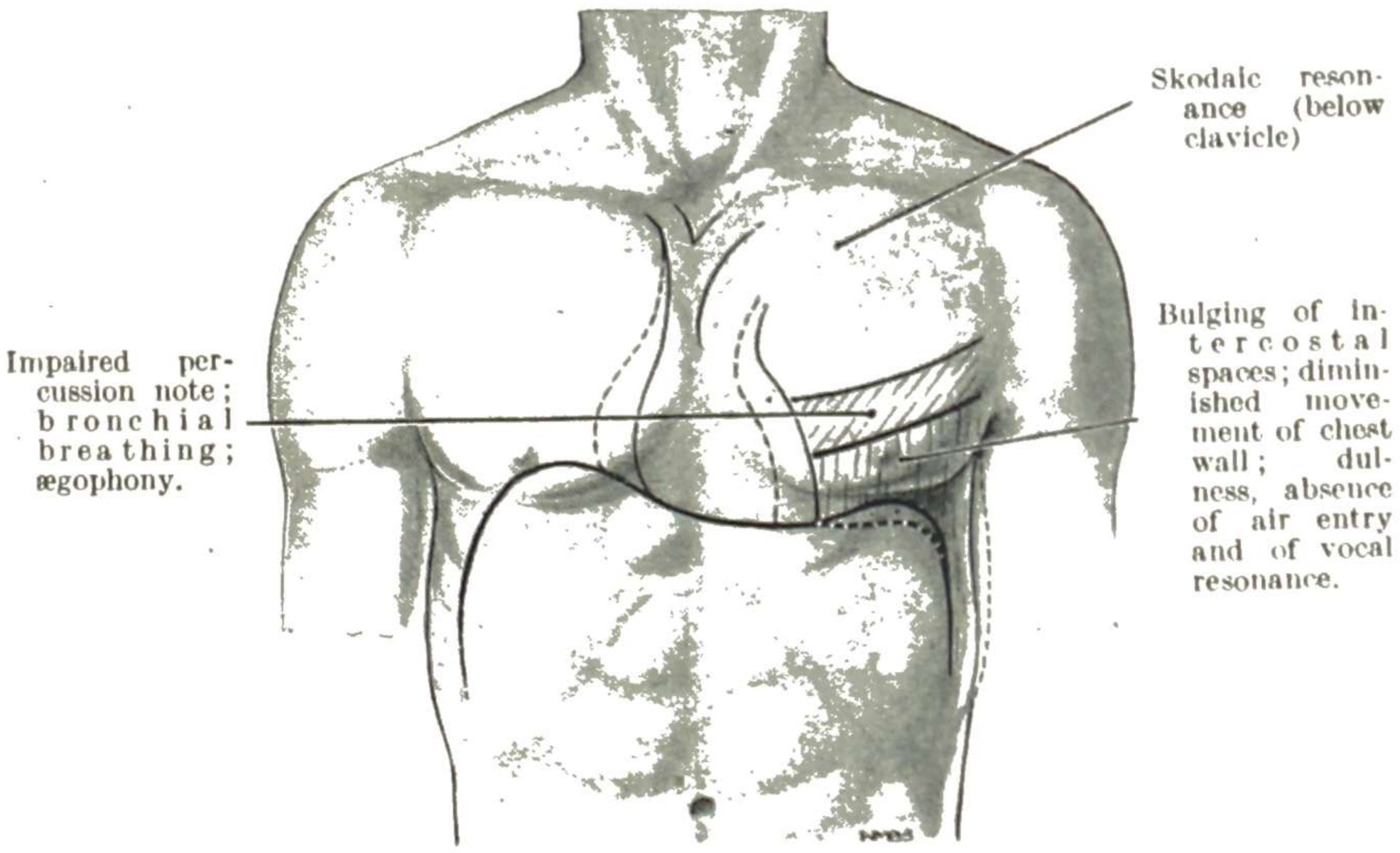


FIG. 49.—Physical signs of Pleurisy with effusion.

as compared with those of consolidation of the lung, is so important that it is given in Table V (§ 109). In case of doubt, exploratory puncture is essential to determine both the presence and the character of fluid in the pleural cavity.

Etiology.—Tuberculosis is by far the commonest cause of a sudden pleural effusion in a young adult. The fluid in such cases is commonly clear and straw-coloured: the cells are predominantly lymphocytes, though occasionally some polymorphs may be present. Young patients with a serous lymphocytic effusion should be regarded as tuberculous unless some other cause can be proved; culture on special media and/or guinea-pig inoculation of the deposit often reveals tubercle bacilli (p. 1121).

[1] High-pitched bronchial breathing is commonly heard over the compressed lung above the level of the fluid. Occasionally, however, breath sounds are still audible over a much larger area, in spite of the presence of a considerable effusion. This is especially the case in children (*e.g.*, where empyema is present).

The occurrence of a pleural effusion in a middle-aged or elderly patient should raise a suspicion of malignant disease: in such cases further investigation by bronchoscopy, etc., is indicated. In a few instances of acute tuberculous effusions the fluid is blood-stained; in the majority of cases a sanguineous pleural effusion is pathognomonic of new growth. (For other causes of serous pleural effusion, see § 134 Hydrothorax.)

Prognosis.—This depends on the cause and on the treatment available. Most tuberculous effusions will absorb if left alone. In the early stages there is often an irregular pyrexia; this usually settles in the course of a few weeks; the fluid shows signs of diminution, the vocal fremitus and resonance return and the breath sounds become more audible. After absorption, the adjacent pleural surfaces usually become adherent. If the effusion lasts a long time and re-collects after repeated aspiration, a tuberculous empyema should be suspected.

Obliterative Pleurisy.—Occasionally, after gradual absorption of a pleural effusion, the formation of multiple adhesions results in a partial or complete obliteration of the pleural cavity. This is a common sequel of effusion occurring during a course of artificial pneumothorax therapy, and arrests this treatment. In such cases, provided no uncollapsed cavities remain in the lung, an obliterative pleurisy may sometimes be an entirely beneficial end-result. The physical signs of this condition are those of thickened pleura. (See § 137.)

Treatment.—In the first place patients must be kept strictly in bed: they usually prefer to sit up, supported by pillows. No treatment other than a diagnostic puncture of the chest is necessary, but if after a few weeks' trial these measures fail, the question of aspiration should be considered. It must be emphasised that the mere presence of fluid in the pleural cavity is not an indication for its immediate removal. There is seldom any urgency, unless it is found to be purulent (empyema), or unless the amount present causes grave discomfort to the patient, who may be suffering from cardiac embarrassment. When the fluid has persisted for many weeks without showing any signs of absorption, it may then be advisable to remove part of it (*e.g.*, 10 to 20 ounces), after which the remainder often becomes absorbed in a reasonably short time. As a general rule it is inadvisable to delay paracentesis and aspiration under the following conditions: (i.) a large effusion (*e.g.*, with dulness extending upward as far as the third rib); (ii.) cardiac embarrassment, as evidenced by cyanosis, palpitation, and a rapid pulse; (iii.) respiratory embarrassment, shown by urgent dyspnœa and paroxysmal attacks of coughing; (iv.) effusion in the other pleura, or œdema of the other lung; (v.) if the fluid is not sterile.

Paracentesis thoracis.—When possible the patient should be sat up, well supported with pillows; the hand on the affected side is placed on the opposite shoulder. The usual site of puncture is the 8th space in the post-scapular line—or at a site where localised dulness is maximal. An intradermal injection of 2 per cent. procain is made at the intended site of puncture with a hypodermic syringe and needle,

The needle is withdrawn, then thrust through this now anæsthetic skin area, and on slowly through the tissues of the intercostal space until the pleura is reached; the piston of the syringe is pushed down as the needle advances. Thus the track of the needle is anæsthetised with a fine stream of procain. The exploring needle, attached to 20 c.c. glass record syringe, is pushed vertically through the anæsthetised area, gentle suction being maintained all the time. If there is fluid in the pleural cavity, it will enter the barrel as soon as the needle reaches it. Perforation of the lung is indicated if air or frothy bright red blood is sucked up into the syringe; in such a case the needle is withdrawn, the blood driven out and the process repeated, with the needle inserted in another direction.

Aspiration of a pleural effusion is best performed with a Potain's or Dieulafoy's aspirator. It is better to avoid the large trocar and cannula usually supplied with Potain's aspirator: a smaller one, such as Rivière's initial pneumothorax needle, or even the ordinary exploring needle, is usually large enough and can be fitted by means of an adapter to the tube of the aspirator. The smaller needles are easier to introduce, and the patient is spared much discomfort. Passage of the needle is facilitated by making a small incision in the skin with the point of a fine scalpel. The side tube of the needle is connected with a large glass bottle, the air in which can be exhausted by means of a pump. After withdrawing the plunger, and turning the tap to connect the pleural cavity with the interior of the bottle, the fluid from the pleural cavity is sucked into it by the partial vacuum. The aspiration can be continued until the patient is conscious of slight discomfort. If cough or pain occur, it is wiser to cease; it is unwise to remove more than 20 fl. oz. at a time unless it is accompanied by air-replacement, which obviates the cough and discomfort due to the sudden re-expansion of the lung. At the conclusion, after withdrawing the needle or cannula, cover the puncture wound with gauze soaked in collodion.

Siphonage can be used for the removal of the fluid, but is seldom so efficacious. When the condition of the underlying lung is such that collapse therapy is necessary, the fluid may be replaced by air: but this should never be the routine treatment of a pleural effusion.

Ia. *The physical signs are those of pleurisy with* EFFUSION, *but it does not clear up in due course, and the patient continues to have* SWEATINGS, SHIVERINGS, *and an* INTERMITTENT HIGH TEMPERATURE. *The disease is probably* EMPYEMA.

§ 120. Empyema is a collection of purulent or sero-purulent fluid within the pleural cavity. It often follows a serous effusion, but it may be purulent from the beginning. The pneumococcus is the organism most commonly found.

The *Symptoms* and *Physical Signs* are similar to those of serous effusion (*q.v.*, *supra*), with certain others in addition—viz.: (1) It may be found that the fluid *does not clear up* as a serous effusion should do, and thus the presence of pus may be suspected. (2) Whenever pus forms, either in the pleura or elsewhere, it is marked by the occurrence of sweatings, shiverings, and an intermittent pyrexia. (3) Œdema of the integument, the pointing of an abscess in an intercostal space, over the clavicle, or even in the groin, or copious expectoration of pus, may occur if an empyema is overlooked. The modern knowledge of the radiograph and the wider use of the exploring needle have made these accidents less frequent. (4) Clubbing of the fingers, especially in children, is a valuable sign which

may come on very rapidly in empyema. (5) The history commonly reveals one of the following *causes* of empyema:

(i.) Lobar pneumonia is by far the commonest cause, especially in children; (ii.) septic conditions of the pericardium, mediastinum, or respiratory tract; (iii.) tuberculosis in any form in the thorax; (iv.) the acute specific fevers; (v.) abscess of the lung, abscess of the liver or spine bursting towards the pleura, or peri-hepatic abscess resulting from appendicitis, leaking gastric or duodenal ulcer; (vi.) pyæmia; (vii.) careless paracentesis, or any wound from without, permitting the introduction of organisms.

(6) In doubtful cases a leucocyte count should always be made, since in the absence of acute lobar pneumonia more than 20,000 leucocytes per cu.mm. would strongly favour the diagnosis of empyema. (7) Diagnostic puncture is essential when pus is suspected, though there are two fallacies in this method: first, in rare cases the fluid may be too thick to come through the needle; or, again, the pus may be encysted between the lobes of the lung. In any case, a pathological examination of the material at the point of the needle will assist the diagnosis.

Prognosis.—Empyema is always serious, and may run a somewhat prolonged course of some months. Cases of pure pneumococcal empyema are much more favourable than those due to streptococci or staphylococci, either alone or with the tubercle bacillus. Operation, adequate drainage, and strict aseptic precautions, both at the operation and at the subsequent dressings, are the points in treatment which most favourably influence prognosis. If left to itself, the results vary: sometimes there is compression and destruction of the lung; sometimes, as above mentioned, the pus opens into the lung, burrows in various directions, or opens through the chest wall; or the condition may lead to pyæmia.

Treatment.—A pneumococcal empyema is usually drained as soon as the diagnosis is made. In streptococcal empyema drainage by rib-resection is not performed until the fluid is frankly purulent, because operation in the sero-purulent stage is attended by very high mortality. In the early stages, the introduction of penicillin daily into the pleural cavity (120,000 units in 10 c.c. of physiological saline) after withdrawal of the fluid is very valuable. In tuberculous cases open operation is avoided whenever possible.

The after-treatment of empyema is designed to promote expansion of the lung and is most important. The establishment of air-tight drainage at operation is very helpful. The tube leading from the wound is connected with an under-water drain. In the convalescent stage breathing exercises are of the utmost value. To obtain the best results these should be supervised by an expert, who will ensure the maximum degree of movement of the affected side, movements of the contralateral side being restricted by the masseuse.

II. *The patient has been* TAKEN ILL SUDDENLY; *the temperature is high, the dyspnœa considerable, and cyanosis is present; the expectoration*

soon becomes rusty; there are SIGNS OF CONSOLIDATION *at the base of one lung. The disease is* ACUTE LOBAR PNEUMONIA.

§ 121. Pneumonia—*i.e.*, inflammation of the pulmonary tissue proper, or parenchymatous inflammation—occurs in two forms. The *first* and more acute is, from its area of distribution, termed "Lobar Pneumonia." The *second* is termed "Broncho-Pneumonia," because it affects the bronchi, and spreads to the lungs; see § 123.

Acute Lobar Pneumonia commences suddenly, with well-marked constitutional symptoms, such as headache, backache, rigor, and, in children, vomiting or convulsions. The temperature during the rigor rises to 103° or 104° F., and it remains at this point for about a week (Fig. 50). The aspect of a pneumonia patient is very characteristic (§ 7). The face is flushed and cyanosed, and herpes often appears near the mouth. There is pain in the affected side due to involvement of the pleura, short cough, shallow rapid breathing, and tenacious mucoid sputum which becomes rust-coloured on the third or fourth day. The pulse-respiration ratio is 3 to 1, or $2\frac{1}{2}$ to 1, instead of the normal 4 to 1. The urine is scanty and high-coloured. The patient shows more and more distress, and in a short time there may be delirium, with signs pointing to failure of the cardio-vascular system. About the *seventh* or *eighth* day the fever, as also the pulse and respiration rate, in favourable cases, terminates by crisis, falling to normal in the course of a few hours. This is accompanied by marked general improvement; the pulse-respiration ratio returns to normal, and a critical sweating or diarrhœa may occur. Pseudo-crises occasionally occur, but these are distinguished from true crises by the fact that the pulse and respiration do not return to normal. In some cases the temperature falls by lysis. The whole illness lasts about two or three weeks. If it lasts longer, *tuberculosis should be suspected* (§ 131), or more commonly some complication such as empyema (§ 120).

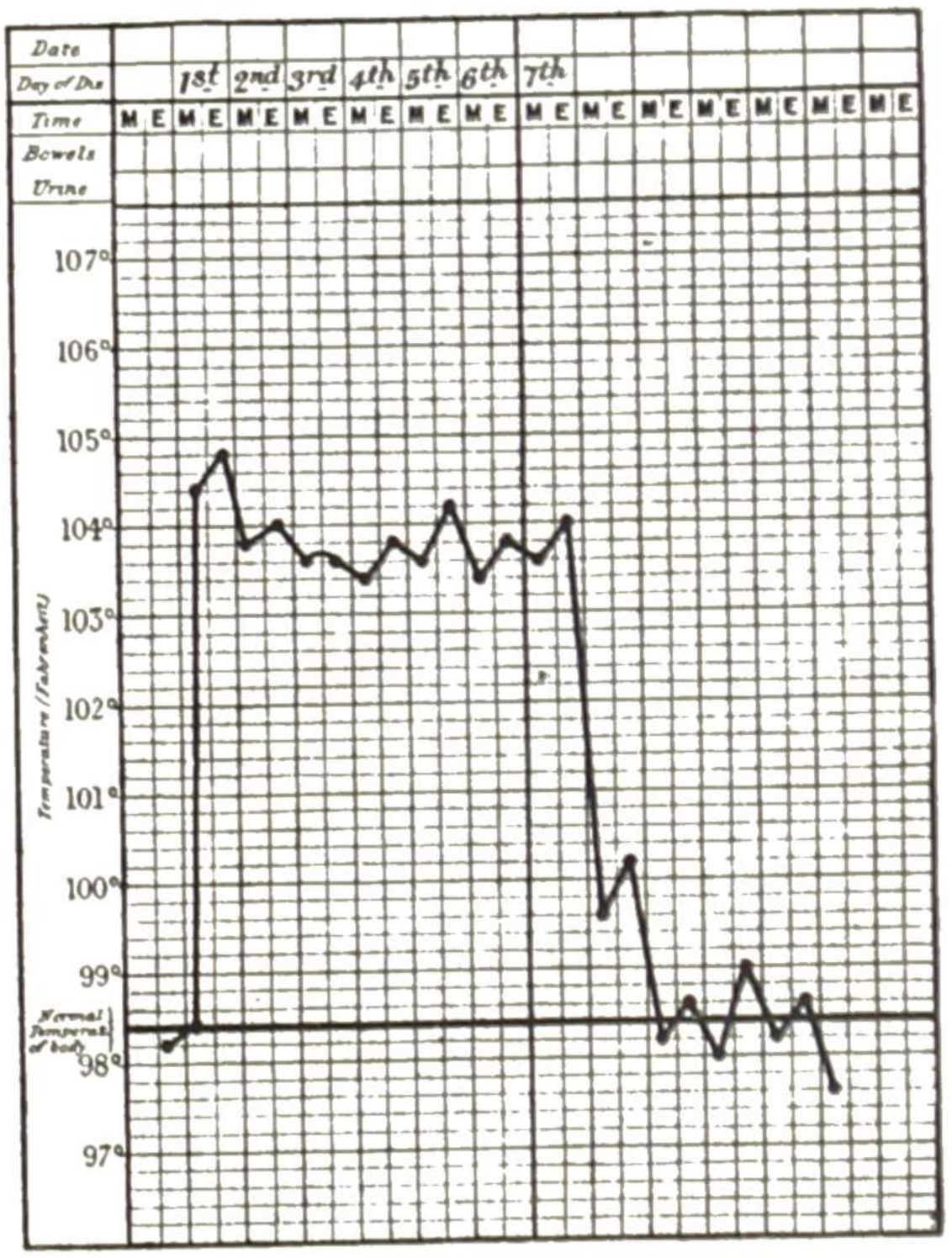


FIG. 50.—ACUTE LOBAR PNEUMONIA, showing typical crisis on the seventh day. George H., aged thirty-five, was taken ill very suddenly with shivering and acute pain in the side. (No chemotherapy was given.)

The *Physical Signs* are limited to one lung or one lobe, usually the right lower lobe. It is only in rare cases that both lungs are affected. At the onset percussion may, for the first day or two, reveal no dulness,

but, as a rule, there is elicited early in the disease slight impairment of the percussion note, which soon becomes dull. On auscultation the breath sounds are weak, and fine rustling crepitations are heard which have been compared to the rustling of hair or tissue-paper against the ears. The weak respiratory murmur is noticeable many hours before the bronchial breathing. The latter, when it does come, may be feeble; the significance, however, is the same. As the inflammatory exudation increases, the lung tissue becomes solid, and over the dull area we get all the *signs of consolidation* (§ 109). When the fever abates, coarse moist râles (redux crepitations) are heard, and the normal percussion resonance and breath sounds gradually return.

Central Pneumonia. Cases of acute pneumonia begin with the typical onset described at the beginning of this section, but physical signs of consolidation do not appear until very late (*e.g.*, a week or ten days). In other cases the fever abates suddenly, after two or three days, with a crisis similar to that of an ordinary case, the physical signs of lobar consolidation being entirely absent. Such phenomena are due to the fact that the actual lesion in the lung is deep-seated, and only spreads to the surface much later, if at all. X-ray examination reveals the condition.

Etiology.—Pneumonia occurs at all ages and in both sexes, but is commonest in adult males. It is a bacteriæmia, the specific cause being a diplococcus, the pneumococcus of Fraenkel. Debilitating influences, such as exposure, are said to predispose to the disease; but it is surprising how often strong, apparently healthy men are attacked, and these not infrequently succumb. A blow on the chest may determine an attack (traumatic pneumonia). Like other local inflammatory diseases, it may arise as a complication of a constitutional malady; the acute specific fevers in particular rendering a person vulnerable to the pneumococcus. The termination of chronic nephritis, etc., by pulmonary complications is usually a question of so-called hypostatic pneumonia. This should not be included under Lobar Pneumonia proper. When pneumonia runs an atypical course we should always bear in mind the possibility of the lung affection being only a complication of a constitutional disease such as typhoid fever.

Diagnosis.—Pneumonia is diagnosed from acute *pleurisy with effusion* (in which the lungs are often affected) by means of the data given in the table of diagnosis between consolidation of the lungs and fluid in the pleura (§ 109). *Broncho-pneumonia* runs a different course, and the signs are scattered over both lungs (see table below). The sudden onset of acute pneumonia resembles that of *scarlet fever*, *erysipelas*, and *small-pox*, but the absence of rusty sputum and altered pulse-respiration ratio distinguishes them. There is a pneumonic form of *acute pulmonary tuberculosis* which has to be borne in mind (§ 124), also various *aberrant forms of pneumonia* (§ 122) and acute exacerbations in bronchiectasis. Pneumonia may, especially in children, at its onset simulate *abdominal inflammation*, pain being referred to the abdomen, and lung signs being absent (§§ 238,

248). It is in such cases that a diminished respiratory murmur on one side of the chest and an increased respiratory rate are signs of great value.

Prognosis.—The case mortality of all ages combined used to vary between 20 and 40 per cent., but the advent of the sulphonamides and subsequently of penicillin treatment has reduced this to 5 per cent. or less. Instead of lasting on an average 7–8 days, treatment with these drugs causes the temperature to drop to within one degree of normal within 48 hours, with corresponding improvement in the patient's clinical condition. The prognosis is worse when the natural resistance of the patient is low or the invading organism particularly virulent: pneumonia is more serious in elderly persons, in young children, and in alcoholics, diabetics and in others suffering from debilitating conditions. Children from 3 to 10 years nearly always recover, but robust men in the prime of life often succumb, although the prognosis is generally stated to be good in healthy adults. The type III pneumococcus produces a higher mortality than any of the other types. Unfavourable features include the extensive involvement of lung (the outlook being worse when both lungs are involved), marked cyanosis, considerable delirium or a typhoid state, an unduly low temperature, marked tachycardia and particularly absence of the usual leucocytic response. Jaundice, meteorism and auricular fibrillation may occur. Complications of serious import include pneumococcal septicæmia, meningitis or endocarditis, but even these often come under control with adequate and early chemotherapy. Delayed resolution, lasting one to three months, is uncommon; empyema, abscess, and gangrene may supervene in weakly subjects. By far the commonest complication is empyema.

Treatment.—Chemotherapy with the sulphonamide drugs, and/or penicillin should be started immediately the diagnosis is made. In pneumococcal infections sulphadiazine, sulphamethazine or sulphamerazine are usually chosen, though in staphylococcal infections sulphathiazole and in streptococcal infections sulphapyridine are regarded by some as more effective (see Tables XXVIII, XXIX and § 515). For an adult, the usual initial dose is 4 G. followed by 1 G. 4-hourly for 48 hours, after which smaller amounts are given until the temperature has been normal for at least 48 hours. The total amount given usually varies between 40 G. and 60 G. (For doses in children see Table XXVIII.) An adequate fluid intake of at least 6 pints in 24 hours is essential: it is advisable also to give large amounts of alkali simultaneously, *e.g.*, sodium bicarbonate 6 G. initially followed by 2·5 G. every 4 hours with the sulphonamide tablets, which should be crushed before administration.

Penicillin may be used instead of, or in addition to, sulphonamide therapy: the usual dose is 20,000–30,000 units in sterile water subcutaneously every 3 hours day and night for periods up to 5 days. The necessary concentration in the blood can be maintained by giving as much as 500,000 units 8-hourly, thus avoiding disturbing the patient's sleep.

General treatment.—The administration of sulphonamides and/or

penicillin cannot be regarded as a substitute for the general measures which must be observed in all cases of pneumonia. The patient's strength must be maintained by rest in bed, good nursing, adequate sleep, and regular visits by the doctor. Fresh air is essential: patients treated near an open window have less dyspnœa and cyanosis, and do better than those treated in a vitiated atmosphere. They should be kept sufficiently warm by blankets and, if necessary, by hot bottles. The bowel should be cleared with an initial dose of calomel. The *diet* must be fluid with 2 to 3 pints of boiled milk in addition to fruit-drinks, lemonade or barley water, with glucose. Raw eggs, broths and jellies can be added later. If acute dilatation of the stomach or intestinal paresis sets in, with vomiting and abdominal distension, pituitary extract, prostigmin and lavage will often avert a fatal issue. For meteorism give turpentine fl. oz. $\frac{1}{2}$ to a large enema. *Sleep* is of such paramount importance that no patient should be allowed to spend a restless night. The cause of the restlessness should be sought for and treated. For extreme restlessness with delirium, give chloral and potassium bromide, in doses of 30 to 40 grs. of each. When fever over 103° is the cause of sleeplessness, it may be reduced by tepid sponging, a measure which, next to the relief of pain and engorgement of the right heart, is the most satisfactory means of procuring sleep. Hypnotics such as paraldehyde may be given, or morphia when pain is severe. An initial dose of $\frac{1}{4}$ grain of morphia at the commencement of the disease often benefits, by ensuring sleep: in the later stages it is usually wiser to avoid morphia—it should never be given if the patient is cyanosed. Frequently *pain* is the disturbing factor. Apart from the use of morphia, this may be relieved by the local application of ice, fomentations, poultices, a blister, or leeches. Another cause of sleeplessness is *engorgement of the right heart*: in every case of pneumonia careful watch should be kept for this. If, in the early stage of the disease, the patient is blue and restless, the cardiac dulness increased considerably to the right, the liver enlarged, and the veins of the neck full, we should immediately relieve the right heart, by venesection (5 to 10 ounces) or by applying six leeches to the skin over the liver. Opium (10 gr. Dover's powder) may be used in the early stages of the disease, and is often of the greatest value, but if the right heart shows signs of engorgement, it is better to give morphia with atropine. Small doses of potassium iodide loosen tenacious sputum.

Stimulants.—The value of oxygen inhalations is well proved, especially in cases with cyanosis; they should be begun early. The most convenient method of administration is by a nasal catheter or by one of the recognised forms of mask (*e.g.*, the B.L.B. mask), at the rate of five to seven litres per minute, which is as fast a flow as can be comfortably borne. The value of the oxygen tent in severe cases, and especially in children, is beyond doubt. The modern apparatus is easy to manipulate. In order to obtain the necessary concentration (30 to 60 per cent.), oxygen is introduced at a rate of five to ten litres per minute, the patient being continually in the tent, except when nursing attention demands a temporary interruption. The

administration of oxygen by other (traditional) methods (subcutaneous injection and a wide-mouthed glass funnel attached to the tube from a gas cylinder and held in front of the patient's face) is inadequate and of no practical use. Dry oxygen is an irritant. It should therefore be moistened by being passed through warm water before reaching the patient. Nikethamide B.P. (coramine) and other vaso-motor stimulants can be used as required. Concerning alcohol, there is much difference of opinion. It is particularly indicated in alcoholic patients, for whom it should be used freely (4 to 12 ounces whisky in twenty-four hours), and especially in conditions of collapse near the crisis, when it may tide the patient over so that he is out of danger before the subsequent depressing effect of the drug becomes manifest. Pneumococcal pneumonia is an infectious disease. Preventive treatment comprises care of the mouth, teeth, and nasopharynx, and the use of pneumococcal and influenzal vaccines.

Treatment by *specific serum* has largely been replaced by chemotherapy. If toxæmia is severe before chemotherapy can be started, Felton's serum (which is polyvalent for Types I and II pneumococci) may be used (and see § 521).

§ 122. Aberrant Acute Pneumonias.—We have seen that in pleurisy, acute lobar pneumonia, and in other inflammatory diseases of the lungs, the course of the malady is fairly definite, and the physical signs in the lungs are characteristic. But it is important to remember that these same conditions may occur secondary to, or as part of, some general disorder. In these circumstances some of the symptoms or physical signs may be wanting or irregular, and it may not be possible to arrive at a diagnosis, except by passing in review the whole history of the case, and by making a thorough and systematic examination of all the other organs. Instances of this eccentric group of pneumonias are met with in acute glanders, tuberculosis, plague, anthrax, syphilis of the lung, distoma infection, actinomycosis, and psittacosis.

The practical outcome of this is that when a case of pneumonia, or other apparently local inflammatory condition, is *atypical* in its physical signs or its clinical history, we probably have to do with a manifestation of one of the conditions just mentioned, or some general disease, such as typhoid fever, influenza, scarlatina, pyæmia, or other general infective disorder.

The term *pneumonitis*, introduced originally in the United States of America, was intended to cover a wide range of conditions characterised by the presence of an area or areas of localised consolidation in the lung. Such conditions, although inflammatory in origin, are not examples of pneumonia in the ordinary sense of the term, and do not give any typical clinical picture with a definite clinical course. The term is a useful one to denote the nature of the underlying pathological process, but it should not be supposed that pneumonitis indicates a clearly defined clinical entity.

The so-called *primary virus pneumonia*, frequently referred to by writers in the U.S.A. as primary atypical pneumonia, comes into this group. Usually mild in type, with few physical signs in the chest, there is no leucocytosis or response to chemotherapy: in consequence the temperature may be slow in settling to normal.

III. *The illness has come on* LESS SUDDENLY *than in lobar pneumonia; there is cough, with frothy expectoration; the physical signs of* CONSOLIDATION *are* MORE PATCHY *and accompanied by signs of bronchitis. The disease is probably* BRONCHO-PNEUMONIA.

§ 123. Acute Broncho-Pneumonia is also an acute parenchymatous inflammation of the lungs, but it runs a very different course to that of acute lobar pneumonia. The inflammatory process occurs in small patches,

scattered unequally throughout both lungs, and it is accompanied by bronchitis: hence the name.

The *Constitutional Symptoms* usually come on more gradually in this disease. The temperature is remittent, about 100° F. in the mornings and 101° to 103° F. in the evenings, accompanied by cough, dyspnœa and muco-purulent sputum. The pulse is rapid, but the pulse-respiration ratio is not altered to anything like the extent of that in lobar pneumonia, and the face is generally pale instead of flushed. Without chemotherapy fever is likely to persist for 2 or 3 weeks or more.

Physical Signs.—When the patches of consolidation are small, there may be no dulness on percussion, but only tubular breathing; when they are of moderate size, signs of consolidation (§ 109) can be made out. The chief auscultatory signs in children consist of *intensely loud*, "consonating" râles, and rhonchi.

Etiology.—Broncho-pneumonia occurs at all ages, but is *especially frequent in young children.* The cases fall into two groups, primary and secondary. Primary broncho-pneumonia, due to the pneumococcus, arises in much the same way as lobar pneumonia. Secondary forms arise: (i.) Complicating acute infections, such as measles, whooping-cough, diphtheria, small-pox, influenza, typhoid and scarlet fevers; (ii.) complicating chronic debilitating conditions, such as chronic renal disease, chronic cardiac disease, or bed-lying, as from fracture of the femur in old people; (iii.) *aspiration* or *deglutition* (*septic*) *pneumonia*, such as occurs after operations on the tongue, mouth, or nose, in quinsy, cancer of the œsophagus communicating with the air-passages, bronchiectasis, and following hæmoptysis or the passage of food down the trachea, as in post-diphtheritic paralysis and in bulbar palsy. In operations on the throat and nose under general anæsthesia, attention must be paid to the drainage of blood and the removal of all solid particles of tissue. (iv.) A common but more chronic varicty is of tuberculous origin.

Diagnosis.—Tuberculous broncho-pneumonia is discussed in another section. The pulmonary signs of *measles*, *whooping-cough*, *bronchitis* and *psittacosis* resemble broncho-pneumonia in its early stages, and it may not be easy to diagnose these several diseases until the rash of the one or the whoop of the other appears. The constitutional symptoms in acute bronchitis are much less severe. The diagnosis from *lobar pneumonia* is given in tabular form on page 185.

The clinical picture of acute pneumonia appears to have altered of late years, and the hard and fast line of demarcation between these types is to-day less easy to recognise. The very severe pneumonia seen in conjunction with influenza, and associated with a virulent streptococcal infection, is a comparatively recent phenomenon, attention having been dramatically focussed upon it by the great epidemic towards the close of the War of 1914–18.

In many cases of acute streptococcal pneumonia the onset is similar in its suddenness to that of the pure pneumococcal (lobar) type, but the subsequent picture often differs considerably from that above described under lobar pneumonia, two characteristic features being the greater intensity of the toxæmia in the very early stages and the tendency to end by lysis rather than by crisis. The toxic state of the patient, mental

TABLE VII.—DIFFERENTIATION BETWEEN A TYPICAL CASE (untreated by chemotherapy) OF

	LOBAR PNEUMONIA	*and* LOBULAR OR BRONCHO-PNEUMONIA.
Onset	Sudden, with rigors ..	Gradual, and preceded by bronchitis.
Course of Temperature	Continuous	Remittent.
Defervescence ..	Crisis usually by seventh day[1]	By lysis in two to four weeks.
Percussion	Dulness in one lung, usually the base.	Scattered patches of dulness in both lungs.
Auscultation ..	(i.) Fine crepitations .. (ii.) Consolidation signs in a day or two.	Fine crepitations and consolidation signs over dull areas, though obscured by rhonchi and bronchitic râles.
Sputum	Rusty	Frothy and muco-purulent.
Respiration ..	Pulse-respiration ratio 3 : 1 or $2\frac{1}{2}$: 1.	Less marked difference of pulse-respiration ratio.

apathy, pallor, often cyanosis, are early features, sometimes apparent before the occurrence of appreciable physical signs in the chest. Recent work on the pathology of the pneumonias, especially in children, shows that it is difficult to insist upon the exact division into the lobar and bronchial types described in most text-books; many cases exhibit certain of the features of both, and the traditional distinction may be impossible to maintain. The important point is to differentiate the acute pneumonias according to the nature of the infecting organisms as a guide to accurate chemotherapy.

Prognosis.—Prior to the advent of drugs of the sulphonamide group the case mortality in children under five varied from 30 to 50 per cent.; the younger the child the more fatal was the disease, and under the age of 6 months 90 per cent. of cases were lethal. The age of the patient, the virulence of the infection, and the degree of response to chemotherapy are leading factors in the prognosis. The longer the disease persists the more likely it is to have a fatal termination. Broncho-pneumonia is nearly always secondary, and a factor of importance in the prognosis is the nature of the antecedent disease. When a child weakened by a *prolonged* fever is attacked, the prognosis is very grave; nevertheless, children often recover in apparently hopeless cases. Pulmonary fibrosis is a well-recognised sequela of broncho-pneumonia occurring in whooping-cough and measles. The aspiration and deglutition pneumonias are usually fatal.

Treatment resembles that of lobar pneumonia (*q.v.*) Children should be placed in an oxygen tent. Adults may need Dover's powder or linctus heroin if their nights are rendered sleepless. If the symptoms of cough and dyspnœa are distressing, alkaline expectorants with small doses of potassium iodide (gr. 3) will often give relief. For the reduction of excessive fever tepid sponging may be invaluable.

[1] In many epidemics lysis is more common than crisis.

§ 124. IV. **Acute Pneumonic Phthisis** (synonym: Acute Caseous Pneumonia) is not uncommon. The symptoms resemble those of pneumonia, and may start suddenly with a rapid rise of temperature and pain in the side. The temperature may continue high for a week or so. The physical signs also resemble those of pneumonia. It differs from this disease, however, in the presence of tubercle bacilli in the sputum, and the temperature, instead of falling abruptly by crisis about the seventh day, gradually becomes intermittent, and the *course of the disease* becomes indefinitely prolonged for weeks. This is followed by physical signs of breaking down in the lung, purulent expectoration, night sweats, and in some cases death in five to twelve weeks from exhaustion, hæmoptysis, or complications, such as pneumothorax (§ 126).

§ 125. V. **Acute post-operative massive collapse** of the lung is an important condition. Opinion inclines to the view that most of the so-called post-operative pneumonias are due to massive collapse. There are probably several causal factors; obstruction of a bronchus by viscid secretion is believed to be the most important. If a main bronchus is obstructed, constitutional disturbance may be severe, simulating that of pneumonia. The physical signs and radiological appearances are like those of pneumonia, but the displacement of the heart towards the affected side should make the diagnosis clear. *Treatment* consists in giving inhalations of 7 per cent. carbon dioxide and 93 per cent. oxygen several times a day, for 10 to 15 minutes at a time. An alkaline expectorant mixture containing a small dose (*e.g.*, gr. 3) of potassium iodide, thrice daily, helps to increase secretion and to liquefy any viscid mucus present in the bronchi. Removal of the obstructing plug of sputum, mucus, etc., by means of the bronchoscope, is often necessary.

We now turn to the **acute disease with hyper-resonance on percussion**—viz., Pneumothorax. Bear in mind that an acute disease may supervene upon a chronic condition accompanied by hyper-resonance—*e.g.*, when acute bronchitis supervenes on emphysema (see Table VIII, § 142).

The patient is in MARKED DISTRESS, *which has come on* SUDDENLY; *there is cyanosis, often hyper-resonance and absence of breath sounds, or faint bronchial breathing over one lung. The disease is* PNEUMOTHORAX.

§ 126. Pneumothorax is a term used to denote the presence of air in the pleural cavity, the air having gained admission by perforation of the pleura, either from within or from without. Effusion may form (hydropneumothorax) which, after a time, may become infected. The condition is then known as pyopneumothorax.

The *Symptoms* of the onset of the condition differ according to the condition of the lung—*i.e.*, whether it is fairly healthy or is widely diseased. (*a*) When pneumothorax occurs in the less affected of the two lungs—the other side being extensively diseased—the symptoms are very urgent, and consist of severe pain in the side, attended by great dyspnœa, shallow, quick breathing, cyanosis, and some degree of collapse, with sweating,

lividity, and a weak pulse. (*b*) In other cases, where pneumothorax comes on in a lung which is already much diseased, the onset and the physical signs may be hardly noticeable. (*c*) The occurrence of spontaneous pneumothorax in the apparently healthy, though uncommon, is not so rare as has been supposed. The onset of symptoms is sudden, often apparently a result of strenuous physical effort, *e.g.*, during a game of football, the degree of disability and distress depending on the amount of air which gets into the pleural cavity and the resulting degree of displacement of the heart and mediastinum.

The *Physical Signs* in the chest consist of: (i.) Diminished movement on the affected side; (ii.) diminished tactile vocal fremitus; (iii.) hyper-resonance on percussion; (iv.) on auscultation the respiratory murmur is reduced or absent; amphoric breathing may be heard over the lower half of the chest behind; the vocal resonance is usually diminished, but pectoriloquy and bronchophony are sometimes present; (v.) often displacement of the heart to the opposite side. The *bell sound* may be elicited on tapping the chest with two coins in one position, and listening with a stethoscope in another. When fluid is also present (hydropneumothorax), and this is usual, metallic tinkling is heard. The *succussion splash* is the most characteristic sign of hydropneumothorax—a fact which was well known to Hippocrates. It may be heard by placing one's ear against the chest whilst moving the patient's body from side to side.

Etiology.—(i.) A common cause is phthisis, when the ulcerated portion of the lung or an emphysematous bulla bursts into the pleura. (ii.) Spontaneous pneumothorax in young and apparently healthy adults is believed to be due to the rupture of an emphysematous bulla in the sub-pleural region of the lung. Such emphysematous bullæ may be associated with an old primary tuberculous focus, although there may be no evidence of tuberculosis in the past or subsequent history. (iii.) A fractured rib may lead to perforation of the lung. (iv.) Less common causes are gangrene of the lung, or an abscess connected with the spine or liver bursting into the pleural cavity.

Prognosis.—The prognosis in pneumothorax is often grave, but depends upon the cause. The heart and mediastinum may be pushed right over by the accumulation of air at a raised pressure in the pleural cavity: this, if unrelieved, may be fatal. The *immediate* risk depends upon the urgency of the dyspnœa and cyanosis, the state of the other lung, the patient's general health, and the cause of the condition. As regards the *cause*, the pneumothorax that results from late phthisis or gangrene of the lung is fatal; but that which occasionally complicates whooping-cough, pneumonia, early phthisis, and injury, usually results in recovery. Certain it is that the longer the patient lives after the onset of the pneumothorax, the better is the prognosis for ultimate recovery (§ 131). Death usually occurs from shock and cardiac failure, associated with gross displacement of the mediastinum, unless this can be prevented by artificial removal of air from the pleural cavity.

Treatment.—The usual remedies for shock are indicated. The patient must be kept as still as possible, preferably semi-recumbent, though sufficiently propped up to avoid increasing respiratory distress. Warmth should be applied to the extremities and stimulants may be necessary. Oxygen may aid if there is marked cyanosis. If there is severe pain, or if the patient is very restless and in great alarm, small doses of morphia may be indicated. Air must be removed from the pleural cavity when great distension is present, as indicated by marked displacement of organs, extreme pain and discomfort, but the relief is often only temporary. In these circumstances continuous aspiration of air must be carried out until the hole in the lung becomes sealed off. If the lung is seriously diseased, it may be kept collapsed (see § 131, Artificial Pneumothorax). When pus is present, it should be aspirated.

There is one disease of the lungs which belongs neither to the acute nor to the chronic category, but is **paroxysmal**, *occurring in attacks of sudden onset, usually* WITHOUT ELEVATION OF TEMPERATURE—ASTHMA.

§ **127. Asthma** is characterised by paroxysmal attacks of dyspnœa, the inspiratory effort being short, the expiratory prolonged. In severe cases there may be much cyanosis and distress. Chronic bronchitis is liable to complicate asthma, but it is important here to draw attention to the frequent error which is made by regarding exacerbations of chronic bronchitis as paroxysms of asthma.

Symptoms and Clinical History.—The leading characteristic of this disease is its paroxysmal nature. A person who is subject to asthma may be perfectly well one minute, and half an hour later may be seized with the most violent dyspnœa. An attack often commences in the early morning, the patient awakening with a feeling of tightness of the chest; he coughs and gasps for breath and wheezes, and clings to surrounding objects in order to bring into play the accessory muscles of respiration. An attack is often associated with a paroxysmal cough, and a plug of mucoid sputum is ultimately expectorated with corresponding relief. Each attack lasts from a few minutes to a few days, and then, without apparent reason, the patient rapidly recovers his normal health.

There are many curious features in connection with this malady, one of the most interesting being the tendency to skin eruptions (urticaria, prurigo and eczema), and another the fact that these eruptions may alternate with the attacks of dyspnœa. Hay fever, migraine, and even attacks of epilepsy, may alternate in the same way. The paroxysms of asthma are occasionally preceded, ushered in, or terminated by violent attacks of sneezing, or by itching; large quantities of urine may be passed as an attack subsides.

Physical Signs.—On inspection the chest is seen to be maintained in a position of inspiration, undergoing but little expansion with the short inspirations. The percussion note may be unaltered, but, after many attacks, emphysema supervenes, with consequent hyper-resonance. On auscultation the short inspiratory effort is feeble and scarcely audible;

expiration is prolonged. Loud rhonchi and often coarse râles replace the normal vesicular murmur.

Etiology.—The central fact, which alone explains all the symptoms, is a narrowing of the bronchial tubes, due to spasm of the involuntary bronchial muscles, with hyperæmia of the submucosa and swelling of the mucous membrane. Asthma is to be regarded as a manifestation of allergy (§§ 521, 609). The association of the attacks with the presence of certain animals, especially the horse and cat, has long been recognised. It is now claimed that about half the asthmatics tested intradermally with foreign proteins obtained from animals, feathers, foods, etc., show increased sensitivity to some of these substances. In certain cases, indeed, it has been possible to "desensitise" patients so that they no longer have attacks of asthma. Although this recent treatment has often proved disappointing, skin testing is of value since it may reveal a hypersensitiveness to substances which can readily be avoided. The cases which are of allergic origin usually start in early life, often before the age of 10 years. Bronchial spasm may not commence until much later in life, and may suddenly supervene during an acute attack of bronchitis: such cases often give no family or personal history of allergy, and intradermal skin tests are usually negative.

Among the *predisposing* causes we find: (i.) A family history of allergy. Careful inquiry may reveal asthma, hay-fever, urticaria, Besnier's prurigo and infantile eczema. (ii.) Asthma may occur at any *age*, but frequently makes its first appearance soon after the age of puberty. (iii.) Any previous lung disease, especially chronic bronchitis, may predispose to asthma.

Among the *exciting* causes of an attack may be mentioned: (i.) Certain atmospheric conditions which are little understood, and often appear to be most contradictory. Thus I know one patient who is free from asthma in London, but develops an attack immediately she seeks a high altitude. Another develops an attack when she enters London. Some find that the sea relieves them, others that it determines their attacks. (ii.) Reflex causes, such as derangement of the alimentary canal, dietetic indiscretions, or a large evening meal; (iii.) conditions of the nasal passages, such as sinusitis, hypertrophic rhinitis, or polypi; (iv.) dust and irritating particles; (v.) conditions as in hay fever, *e.g.*, proximity to horses, or certain plants; (vi.) emotional causes.

Diagnosis.—The diagnosis usually presents no difficulty. The paroxysmal occurrence of the disease is quite characteristic. Paroxysms of dyspnœa coming on at night occur in the course of chronic nephritis and cardiac disease, and have been loosely called asthma. The actual substances producing asthma may often be determined by performing a series of intradermal inoculations with solutions of the proteins of different substances, *e.g.*, rye, wheat, eggs, feathers, grass, etc., and thus certain definite indications for treatment may be obtained.

Prognosis.—The disease of itself rarely causes death during an attack;

it tends to produce emphysema, bronchitis, and increasing embarrassment of the right heart. Children may grow out of the disease; adults never lose it completely. The severity, frequency, and response of the attacks to treatment are our best guides to prognosis.

Treatment.—(*a*) *During the Attack.*—A subcutaneous injection of adrenalin (1 in 1,000) often aborts an attack. The earlier the drug is given, the smaller the dose; even one to two minims may abort an attack. Inhalation of an adrenalin spray with oxygen, as with the Apneu apparatus, is often efficacious. Pituitrin may be combined with adrenalin, and may be more effective than either drug given separately. Ephedrine tablets are valuable, and in certain cases remove the need for adrenalin injections. Cardophylin (a purin derivative) is a valuable antispasmodic of which 0·24 G. in 10 c.c. of sterile water may be given intravenously or 0·48 G. in 2 c.c. of sterile water by intramuscular injection. The proprietary preparation Riddobron may be effective. Benzyl-benzoate, stramonium, lobelia, belladonna and hyoscyamus may be tried. Atropin and cocaine sprays also relieve. Various inhalations are useful for the prevention or relief of an attack—*e.g.*, the vapour from a teaspoonful of turpentine and chloroform (chloroform may be pushed to anæsthesia), or the fumes of paper prepared with a strong solution of nitrate of potash, or amyl nitrite. A mixture containing equal parts of the leaves of stramonium, lobelia, black tea, and potassium nitrate is burnt in a tin plate, and the fumes inhaled; relief is thus sometimes afforded. Various other preparations, in the form of cigarettes of stramonium, potassium nitrate, and belladonna, are used, but should be discouraged, as their frequent use tends to produce chronic bronchitis. Morphia is helpful, but the risk of addiction is considerable.

(*b*) *Between the Attacks.*—The effect of locality on the disease can only be ascertained by experience. As a rule, though with many exceptions, town air and fogs are detrimental. It is better for patients to live in the upper storey than on the ground floor of a dwelling. The patient's general health must be especially considered, and anxiety, worry, and other psychological factors must receive particular attention. Reflex causes should be eliminated as far as possible: avoid large bulky meals at any time and solid meals after 2 p.m. The nose should be examined for polypi, etc., and all other sources of reflex irritation must be sought for and treated. Ephedrine, gr. $\frac{1}{2}$, at bedtime, together with a simple hypnotic such as aspirin and phenacetin or chloral and bromide, or a capsule of theamine (gr. 3) will often avert nocturnal attacks. The patient should avoid the substances to which he is particularly sensitive, but specific desensitisation has on the whole been disappointing. Injections of normal horse serum (given every fourth day in doses of 4 minims increasing to 2 c.c.) may cure the asthma due to the proximity of horses. Non-specific desensitisation may be tried by injecting Armour's No. 2 peptone intramuscularly (7$\frac{1}{2}$ per cent. solution), or intravenously (5 per cent. solution), starting with 0·3 c.c. and increasing to 2·5 c.c., each third or fourth day.

Weekly intramuscular injections of 10 c.c. of the patient's own blood have often resulted in considerable benefit, without the occurrence of untoward reactions. When the asthma is mainly associated with acute bronchitis, the treatment is that of the primary disease. Potassium iodide is especially useful in liquefying the sputum, and if small doses (3 grains) are not sufficient, much larger doses may be employed. The antispasmodic drugs are again very useful. In some cases benefit may result from the use of an autogenous vaccine. In children, regular doses of glucose, and avoidance of undue excitement or mental strain of any kind, are advisable.

Pulmonary Acariasis.—Asthma due to infestation with mites has been described in India and Ceylon: it is characterised by marked eosinophilia in the blood.

CHRONIC DISEASES OF THE LUNGS AND PLEURÆ

128. Classification.—Chronic disorders of the lungs and pleuræ may follow an acute attack of the conditions described in the previous sections, as when chronic bronchitis and emphysema succeed attacks of acute bronchitis. But many of the chronic diseases of the lungs, such as pulmonary tuberculosis, start insidiously, and attention may not be directed to the lungs for a considerable time.

The chronic diseases, like the acute, may be classified, *for clinical purposes*, according to the results of percussion. It is convenient in actual practice, although unscientific from the point of view of classification, to make a subsidiary group in which the sputum is highly offensive or has some other characteristic feature.

(A) **Chronic Disease** in which the **Percussion Note is unaltered :**

I. Chronic bronchitis .. .. § 129

(B) **Chronic Diseases** attended by **Impaired** or **Dull Percussion Note**

(*a*) The *commoner* diseases presenting dulness, *usually in regular and defined areas either at base or apex*, are—

I. Chronic tuberculosis of the lung .. .. § 131
II. Hydrothorax .. .. § 134
III. Pulmonary congestion (hypostasis or œdema of the lungs) .. § 135

(*b*) The diseases presenting dulness, *usually not* in regular and *defined areas at base and apex*, are—

Common.		Less Common.	
IV. Pulmonary fibrosis (with or without bronchiectasis)	§ 136	VIII. Collapse of the lung	§ 139
V. Thickened pleura ..	§ 137	IX. Hydatid cysts ..	§ 140
VI. Malignant disease of the bronchus	§§ 81 and 138	X. Syphilis of the lung ..	§ 141
VII. Secondary malignant disease of the lung	§ 138	XI. Sarcoidosis	§ 141*a*
		XII. Diseases due to fungi and parasites	§§ 145, 146

(C) **Chronic Diseases** attended by **Hyper-resonance :**

I. Emphysema .. .. § 142
II. Pneumothorax [1] and various other conditions in which the hyper-resonance is not the leading or constant feature (*e.g.*, Skodaic resonance) .. .. .. § 126

(D) **Diseases** suggested by the **Character of the Sputum :**

I. Bronchiectasis §§ 136 and 143
II. Abscess and Gangrene of the lung .. .. § 144
III. Actinomycosis and other diseases due to fungi §§ 145, 146

GROUP A.—The patient's symptoms point to **chronic disease of the lungs,** and on examining the chest there is **no alteration in the percussion note.**

I. *The patient has a chronic cough ; there is no elevation of temperature, and on auscultation* RHONCHI *and* RÂLES *are heard over the chest. The disease is* CHRONIC BRONCHITIS.

§ **129. Chronic Bronchitis** is a chronic inflammation of the bronchial tubes. It usually supervenes on repeated attacks of the acute disorder, but may be chronic from the beginning.

Symptoms.—A patient with chronic bronchitis and—its usual sequel—dilated right heart, often presents a typical appearance. Stout in build, with short, thick neck, of florid, slightly cyanosed complexion, short of breath, wheezy respiration, and pulsating jugular veins, he presents an aspect which can be recognised at once. The clinical history extends over many years, with alternate diminution and aggravation of the symptoms. The cough is usually present during the winter, and improves as the weather gets warmer. The constant coughing and straining to bring up the secretion results sooner or later in generalised emphysema. In later stages the cough continues all the year round, and finally an attack of bronchopneumonia, œdema of the lung, or some intercurrent malady, throws a little extra strain upon the overburdened right heart, and death ensues. There are, as a rule, no febrile or constitutional symptoms.

The *Physical Signs* vary with the amount of secretion present, the extent of the complicating emphysema (§ 142), and the degree of accompanying bronchial spasm. In cases of long duration the chest is barrel-shaped (emphysematous, § 106). It moves poorly. Rhonchial fremitus may be felt on palpation. On percussion there is never any dulness, and the note is hyper-resonant in proportion to the emphysema present. On auscultation sibilant and sonorous rhonchi and bubbling râles can be heard ; crepitations at the bases, due to œdema, may be present.

There are five recognised varieties of this disease : (i.) *Bronchitis* with *winter cough*, attended by slight or abundant expectoration, mucous or muco-purulent, sometimes fibrinous, sometimes containing streaks of blood. (ii.) *Dry Bronchitis* (*catarrhe-sec* of Laennec) is attended by a

[1] Pneumothorax usually comes on acutely, but it may be part of a chronic disease.

frequent cough and soreness of the chest, but little or no secretion; it is of a very obstinate character, and occurs mostly in elderly people of a gouty diathesis. (iii.) *Purulent* and/or *fœtid bronchitis* characterised by expectoration of large quantities of purulent and offensive sputum; associated with bronchial dilatation (*cf.* bronchiectasis). (iv.) *Bronchorrhœa* signifies expectoration of very large amounts of sputum, often of a thin clear nature or else thick and ropy. It is a symptom rather than a disease entity, and is often associated with bronchiectasis (*q.v.*). (v.) *Plastic Bronchitis*, described in § 130.

The *Diagnosis* of chronic bronchitis is usually easy. It is diagnosed from *chronic phthisis* partly by the absence of the tubercle bacillus from the sputum, but chiefly by the absence of radiological evidence of infiltration of the soft tissues of the lung. It is important to remember that bronchitis and emphysema may mask chronic tuberculosis. Such patients may be afebrile and bear no other clinical sign of the more important disease. As they are sources of danger to others, the sputum should always be examined for Koch's bacilli.

Etiology.—Chronic bronchitis may occur at any age, but is more common in elderly people. Sometimes, as before stated, it follows repeated attacks of acute bronchitis, but it may be chronic from the beginning. It often affects plethoric subjects, especially those of a gouty habit, and it is a recognised complication of chronic nephritis. It is a frequent sequel to cardiac valvular disease, more especially disease of the mitral orifice. It may complicate other diseases of the lungs, especially phthisis, and may follow the acute specific fevers, especially measles and whooping cough.

Prognosis.—Adults with chronic bronchitis seldom entirely recover, though they may live for a great many years; and if the heart is fairly healthy and care be taken to avoid exposure, life is not very materially shortened. The co-existence of gout, chronic nephritis and cardio-vascular degeneration make the prognosis somewhat less favourable. The condition of the lungs is not so much a guide to prognosis as the condition of the heart. This, indeed, is the point around which the prognosis centres, and the untoward symptoms which render the prognosis grave are thus referable to the heart—viz., considerable dilatation of the right heart with evidences of cardiac failure, such as great breathlessness, cyanosis, enlargement of the liver and veins of the neck, and ascites.

Treatment.—The extreme frequency of the disorder renders the treatment a matter of considerable importance. In acute exacerbations the treatment is that of acute bronchitis (§ 115). In slight cases, however, the patient can go about, but chill and exposure should be avoided. The important question of when a patient may go out must depend largely on the weather—cold and moisture, especially when in combination, are especially injurious. The choice of a suitable climate is of importance.

The chief points in treatment are: (i.) To repress an excessive cough, since this throws increased work on the right side of the heart.

For this purpose tr. opii camph. in large doses, or heroin lozenges may be used. (ii.) When the cough is dry, remedies directed to promote the secretion are given, such as ipecac., potass. iod., and alkalies. (iii.) When the sputum is too abundant, we may have to diminish secretion by such remedies as belladonna. Counter-irritants to the chest—*e.g.*, turpentine, camphor, or eucalyptus, are very popular with some. (iv.) When there is much spasm of the tubes, lobelia, iodide, ephedrine, and other remedies for asthma are to be tried. (v.) Measures to prevent dilatation and failure of the right ventricle are called for sooner or later where dyspnœa and other cardiac symptoms are present. (vi.) In suitable cases, vaccines have been found useful.

§ 130. **Plastic Bronchitis** is inflammation of the bronchi, with the formation of fibro-plastic casts, which are expectorated.

Symptoms.—The symptoms consist of (i.) violent attacks of coughing, with pain in the chest and expiratory dyspnœa, followed by (ii.) the expectoration of a fibrinous cast of a bronchus. (iii.) The patient generally suffers from chronic bronchitis, and a little hæmoptysis may follow the expulsion of a cast. (iv.) Sometimes there are no constitutional symptoms, but slight pyrexia, and in some cases even rigors may be present. Such symptoms supervening in a case of chronic bronchitis lead us to suspect the condition.

Physical Signs may be absent. If present, they are those of an obstructed bronchus —an absent or diminished respiratory murmur, accompanied possibly by impaired percussion note. Whistling rhonchi or "flapping" sounds may be heard.

Etiology.—The disease is twice as common in men as in women. It may occur at any age in subjects of chronic bronchitis.

Prognosis.—The condition is more serious than simple bronchitis. Two varieties have been described: (1) An acute form, lasting for some weeks; and (2) a chronic form, recurring at intervals for years, in the course of chronic bronchitis. Each attack may last for some weeks, and the casts may be coughed up daily. The condition occasionally leads to a fatal issue.

The *Treatment* differs but little from that of bronchitis. The removal of the membrane may be promoted by the administration of potassium iodide in order to liquefy the sputum: the inhalation of a weak solution of sodium bicarbonate atomised by means of a spray, in order to dissolve the mucin in the cast, has been advised by some. Various oils (*e.g.*, creosote oil, 1 in 40) have been injected as solvents, but the results have not been very promising.

GROUP B.—We now turn to those chronic diseases of the lungs which are accompanied by **dulness on percussion.** (*a*) The *common* diseases in which the dulness occurs, usually in regular and fairly DEFINED AREAS at base or apex, are: I. CHRONIC PULMONARY TUBERCULOSIS; II. HYDROTHORAX; III. PULMONARY CONGESTION OR ŒDEMA.

I. *The patient complains of gradual emaciation and perhaps cough; on examination of the chest* SIGNS OF CONSOLIDATION *may be found, most marked at the* APEX *of the lung; there is* INTERMITTENT PYREXIA, *and the sputum may contain tubercle bacilli. The disease is* CHRONIC PULMONARY TUBERCULOSIS (*Phthisis*).

§ 131. **Chronic Pulmonary Tuberculosis** (Phthisis). The word phthisis is objectionable because it only indicates one of the symptoms—viz., the wasting (*φθίνω*, I waste). For a full exposition of the pathogeny of

this disease the reader is referred to works on pathology. A brief account of the modern view may not be out of place. In civilised communities the universality of the infection by the age of puberty is generally admitted. The newly-born infant, even of a tuberculous mother, is not infected, and there is abundant evidence that children born of tuberculous parents are no more liable to the disease than other children, provided they are separated from possibility of massive infection.

Pulmonary tuberculosis is caused mainly by the human form of Koch's bacillus and is spread by direct contact; it has been shown that droplets of sputum sprayed from the mouth of a tuberculous subject may contain enormous numbers of bacilli. The first occasion of infection by tubercle bacilli, provided the dose is not large enough to cause immediate serious disease, results in a state of allergy or increased sensitiveness, after which a further dose of bacilli will call forth a type of local reaction not seen with the first inoculation (Koch's phenomenon). This reaction localises the bacilli, tending to destroy them and prevent their entrance into the body. If the dose (exogenous superinfection) is too great this reaction produces a more or less severe local lesion in which the bacilli spread. The virulence of the human form of tubercle bacilli varies little; the type of pulmonary disease produced depends on the size of the dose and the resistance of the patient. Certain diseases are known to reduce the resistance to tuberculosis; thus, after measles and whooping-cough, the disease is common in one form or another. After such maladies active disease may begin (without further infection from the exterior) from a previously healed endogenous focus (endogenous re-infection). Because of the variations in resistance, the course of the disease is very variable. It may cause death in a few weeks, or it may spread in successive areas so slowly as to cause little or no disability over a long lifetime. Such cases are dangerous sources of infection, especially to young children. They are usually without fever. The chief safeguard is to examine all specimens of sputum for tubercle bacilli.

Symptoms.—The disease is mainly a chronic one, and its onset is sometimes insidious. It is more amenable to treatment in the early stage, and since the introduction of modern methods of treatment early recognition of the disease has become of paramount importance.

(*a*) *Early Stages.*[1]—Clinically, phthisis has various modes of onset, e.g.: (i.) Progressive weakness, attended perhaps by cough; (ii.) hæmoptysis[2]; (iii.) dyspepsia; (iv.) tachycardia; (v.) pleurisy; (vi.) acute pneumonia (§ 121), bronchitis, or broncho-pneumonia. The pneumonic form may resolve almost completely, leaving a chronic lesion, or sometimes cavitation may occur with great rapidity and not long after the onset. Among the earlier *general symptoms* which should make us suspect the

[1] It must be remembered that in an appreciable number of cases active phthisis in the early stage may be an entirely symptomless disease, only recognisable by radiography (*vide infra*).

[2] Early hæmoptysis may occur before any physical signs are discoverable, except by the use of X-rays.

invasion of tuberculosis are unexplained debility, attended by languor and pallor on the one hand; or on the other hand loss of weight, with unexpected dyspepsia, or slight elevations of temperature in the evening. The temperature is an indication of the greatest importance, for *an* ACTIVE *tuberculous process is usually associated with pyrexia*, however slight. The type of this pyrexia is distinctive; it is *intermittent*, being normal or subnormal in the morning, and raised in the afternoon or at night; in rare instances this is reversed. If we have any suspicion of tuberculosis, the temperature should be taken every two hours, so that we may not miss any slight rise during the day. To avoid missing slight fever, keep the minute thermometer in place for six minutes. In the early stage the patient may not be aware of the feverishness, though occasionally he feels a chilliness in the evening, and as the disease progresses, night sweats are a characteristic feature.

The *Physical Signs* accompanying the earlier stages are necessarily somewhat vague and difficult to detect. The patient's chest should be completely stripped, and he should be taken to a room where perfect quiet prevails; and if with the above symptoms we find weak or unduly harsh breathing and prolonged expiration at one apex—especially if this is accompanied by fine crepitations—we must examine the sputum and have a radiograph taken. It is important to auscultate while the patient coughs, for râles not previously audible may thus become evident. The signs just named can often be heard best at the apex, behind, by placing the patient's hand on his opposite shoulder and listening to that part of the lung, which will thus be *uncovered by the scapula*. Fine crepitations may be heard in that situation weeks before any signs may be discovered at the apex in front. In front the earliest signs may be heard just below the clavicle. Sometimes, later on, we are led to detect phthisis by an undue loudness of the *heart* sounds at the apex of one lung. Absence of dulness, like the absence of bacilli, is not evidence of the absence of tuberculosis. The *sputum* should be repeatedly examined for tubercle bacilli, the presence of which is diagnostic. The early morning sputum is the most likely to contain the bacilli. In repeatedly negative cases bacilli may often be shown in the gastric contents or stools or on culture of the sputum. However, the absence of bacilli, even after several examinations, does not always indicate the absence of phthisis. X-ray examination by an expert is ABSOLUTELY ESSENTIAL in all cases of hæmoptysis or of cough lasting more than three weeks. It cannot be too strongly emphasised that in many cases an active spreading tuberculous granuloma in the lung is unaccompanied by any symptoms and is only recognisable by radiography.

(*b*) *Later Stages.*—The physical signs usually begin at the apex, and are generally best heard at the back, sometimes at the apex of the lower lobe. Extensive tuberculous disease may sometimes exist with but little constitutional disturbance; on the other hand, considerable disturbance of health may be present without any abnormal physical signs—depending,

partly, on the distance of the lesion from the surface of the body. It has many times been proved that a cavity can exist and bacilli be found in the sputum of a patient presenting none of the usual physical signs. X-ray and sputum examination are therefore essential in diagnosing any obscure case, especially if cough is present.

The presence or absence of A CAVITY is in the majority of cases impossible to diagnose with certainty by physical examination alone. The percussion note is usually dull, but varies with circumstances. Thus the note may be resonant when the cavity is very large, or lies very superficially. When the cavity is large and superficial, and the communicating bronchus remains patent, a characteristic note, almost tympanitic, is obtained on percussion whilst the patient keeps his mouth open. This is known as the "cracked-pot" sound (*bruit de pot felé*). The breathing is amphoric.

The *Diagnosis* of the disease is not difficult except in the really early stages. (i.) Other causes of hæmoptysis may have to be differentiated (see § 104); (ii.) other causes of debility may have to be eliminated (Chapter XVI); (iii.) when the condition begins with dyspepsia, it is very liable to be overlooked unless the physician is aware of this mode of commencement; (iv.) other causes of cough (§ 101); and (v.) various pharyngeal and laryngeal affections may have to be excluded (§ 165). In the later stages the differentiation from the other causes of percussion dulness is not difficult (see list in § 128).

Classification.—Reference has been made to (*a*) the earlier stages and (*b*) the later stages of this disease, and a brief account of the symptomatology and physical signs has been given under each heading. However, correlation of the pathological with the clinical aspects of phthisis is not so obvious as most of the older text-books have led one to suppose. The Turban-Gerhardt classification, accepted for so long as the orthodox description, was based upon the anatomical character and extent of the pulmonary lesions, with which the clinical manifestations were assumed to correspond more or less accurately. Such a division of cases is so fallacious as to be extremely misleading; therefore the Turban-Gerhardt classification should be regarded as obsolete. Inman's division of cases of adult phthisis into three main groups: (1) *ambulant afebrile*, (2) *resting afebrile—ambulant febrile*, (3) *resting febrile*, conforms more closely to the actual clinical facts. The extent of the lung lesion is in some degree important, although extensive disease may exist with but little constitutional disturbance. X-ray examination at intervals can and does reveal the development of fresh lesions in patients who are practically without symptoms, and who not only are afebrile but may even be increasing in weight. The age of the patient, the length of time during which manifest clinical disease has developed, the character of the adventitious shadows in the radiogram, etc., are also points to be taken into account in the assessment of any individual case; for their proper appreciation reference must be made to special works.

Etiology.—To find tuberculosis in a newly-born child is extremely rare, even when the mother is in the last stages of the disease. The

separation of children at birth from infected mothers has met with undoubted success in France. The mortality rate of children so removed and brought up by foster parents justifies the modern belief that there is no such thing as inheritance of tuberculosis. Both sexes are almost equally affected, and the age at which the disease usually appears clinically is between sixteen and thirty. The patient may be attacked at any time of life, although clinical signs of lung disease are rare under two years. Any condition of malnutrition may produce a predisposition to the bacillus invasion, whether it arise from deficient food, from hyperlactation, from exhausting diseases such as diabetes, or the acute specific fevers, after which an attack of phthisis is by no means infrequent. It is a curious circumstance that pregnant women offer a high resistance; a phthisical subject becoming pregnant will frequently improve until after her confinement, when an exacerbation of the disease will occur often with a fatal result. Unhealthy surroundings play an important part in the spread of tuberculosis; indoor occupations in over-crowded and ill-ventilated rooms are especially dangerous. Excessive exposure to sunlight, artificial or natural, has sometimes precipitated an acute attack. A silica-laden atmosphere, such as that of stonemasons, knife-grinders, tin and copper miners, fustian-cutters, makes tuberculosis more serious. The exact effect of silicosis on tuberculosis has not yet been fully explained. The 1911 report of the Royal Commission on Tuberculosis confirmed the view that tuberculosis in mankind was due to two types of tubercle bacillus, one of human and one of bovine origin. Pulmonary tuberculosis is for the most part due to infection by the human bacillus, which is conveyed by droplet infection or by dust containing living bacilli—hence the importance of destruction of the sputum. In children, the bovine bacillus is found chiefly in the abdomen (peritoneum or glands), bones, joints, cervical glands, and in the lungs in miliary tuberculosis when the bacillus is carried from an infected focus by the blood-stream. Recent researches in Great Britain have shown that an appreciable number of cases of pulmonary tuberculosis are due to the bovine type of bacillus (*cf.* p. 203). The bovine bacillus enters the body *via* the alimentary canal and tonsils, and causes disease by the ingestion of infected milk of tuberculous animals. It is, however, a fact that mankind is naturally resistant to the tubercle bacillus. Birch-Hirschfield found in 4,000 post-mortems of persons dying from various diseases, that in 40 per cent. the lungs showed evidences of tuberculosis which had undergone spontaneous recovery. Other investigations show even a higher figure.

Prognosis.—1. Usual *course* and *duration*. Phthisis is essentially a chronic but progressive disorder, and formerly nearly all cases applying for treatment terminated fatally. The standardised annual death-rate from tuberculosis of the respiratory system, per million persons living in England and Wales, was 1,517 in 1892 and 532 in 1938. During the war of 1939–45 it rose sharply but fell to 473 (for civilians) in 1947. Rapid cases may terminate in death in the course of three to six months. When

the disease is indolent, and the patient resistant to the bacillus, it may drag on for years. There are four chief modes of death, which in order of frequency are—(1) asthenia, (2) hæmoptysis, (3) bronchitis and heart failure, (4) the occurrence of other complications.

2. The prognosis in individual cases has been greatly improved by four main advances in treatment: (*a*) early diagnosis; (*b*) rest and open air; (*c*) artificial pneumothorax; (*d*) surgical methods of collapse-therapy. The type of disease, its rate of progress, and its distribution are all important in prognosis. The age of the patient influences the course considerably, for it is much more rapid in the young adult than in people over thirty. The hygienic surroundings of a patient, as we shall see under *Treatment*, make a considerable difference to the course of the disease. Where the patient can be properly treated with rest, open air, and good food, he has nowadays a good chance of recovery. A correct prognosis cannot always be made until the patient's reaction to treatment is observed.

3. *Untoward Symptoms.*—(i.) Undoubtedly the most important feature is the temperature. Not only is active tuberculosis evidenced by pyrexia, but the degree of fever, and still more the extent of the diurnal variations, are a fairly precise measure of the activity of the tuberculous process. (ii.) The pulse rate is also important, and, quite apart from the temperature chart, is valuable as a measure of toxæmia. (iii.) The condition of the lung itself is, of course, not without significance, but physical signs must be interpreted in the light of additional evidence furnished by the temperature and pulse charts, and especially by serial X-rays. Variations in the adventitious sounds within a comparatively short period are of greater significance than the persistence of râles which remain unchanged in character. An uncollapsed cavity, with a positive sputum, is always a potential source of danger, and augurs a poor outlook for the ultimate future, though individual patients in such circumstances can live for many years in apparently good health and without much disability. (iv.) Absence of symptoms and increase in weight are of good import, subject to the proviso already mentioned in regard to the warning frequently given by the X-rays. (v.) Hæmoptysis does not appear to bear any constant relation to prognosis, though profuse hæmorrhage weakens the patient considerably, and may be fatal; it is occasionally followed by extension of the disease.

4. *Complications.*—The presence of complications is undoubtedly bad. The commonest complications are: (1) Pleurisy is very frequent, but the adhesions may be beneficial in preventing spontaneous pneumothorax[1]; (2) tubercle may occur in other parts—the peritoneum, meninges, and especially in the intestine, giving rise to ulceration and an exhausting diarrhœa[2]; (3) the larynx may be affected, and undoubtedly this adversely

[1] On the other hand, adhesions may prevent an efficient lung collapse when this treatment is indicated.

[2] Diarrhœa may occur as part of the toxæmia without ulceration of the bowels.

commonly becomes free from bacilli after a few months and therefore the patient is no longer infectious. The drawback is that this method requires special training and constant control by X-rays, and must therefore be left to the expert.

Contra-indications.—In the earlier days of collapse therapy, active disease in the contra-lateral lung was regarded as a contra-indication to the induction of an A.P. The scope of this treatment is now recognised to be much wider, and in many cases bilateral A.P. has restored to active work a patient who would otherwise have gone steadily downhill. Complications such as severe cardiac failure or renal disease are definite contra-indications. Advanced tuberculous enteritis, with an accompanying general breakdown of resistance, usually renders a patient unsuitable for pneumothorax therapy, which should in such circumstances be discouraged. Tuberculous laryngitis, formerly regarded by many as a contra-indication, is now known often to be materially improved by A.P.

Technique.—Air or any other gas is slowly absorbed from the pleural cavity; hence the injections must be repeated at intervals of a week or more. The volume injected is the amount required to maintain the correct degree of collapse without causing discomfort to the patient. Only the regular use of the radiograph enables one to judge this. For further details of technique and control of this important method of treatment, special works must be consulted.

(*c*) *Thoracic Surgery.*—With the development and improvement of surgical technique in the last few years the scope of surgery in the treatment of pulmonary tuberculosis has greatly increased. In cases in which collapse of the diseased lung by artificial pneumothorax is impossible or inadequate, extrapleural thoracoplasty may effect the desired result, and in properly selected cases may save patients who would otherwise go downhill. Temporary diaphragmatic hemi-paralysis (phrenic crush) may relax the elastic tension in the lung sufficiently to heal a minimal lesion, and even occasionally obliterates a small thin-walled cavity. Further elevation of the diaphragm can be produced by introducing air into the peritoneal cavity (pneumoperitoneum). In a small proportion of cases this procedure may obviate the need for major surgery.

(*d*) *Symptomatic Treatment.*—It will be seen that in the advanced stages there is not much hope of recovery, but even in the worst cases we can ameliorate the symptoms, and so ease the passage to the grave. (1) For the cough, tinct. opii camphorata and expectorants are not of much use. The best cough mixture is one containing liquor morphinæ, or better still, codeine in small doses with dilute sulphuric acid. Warm alkaline drinks promote expectoration. (2) Night sweats, which are often very profuse and exhausting, may be combated by belladonna and zinc oxide, especially the first named. Night sweats are seldom troublesome if there be free exposure to fresh air. (3) The diarrhœa is also very exhausting, and must be combated with catechu, opium, intestinal disinfectants, and mineral acids. (4) Pleural pains may be eased by stupes, or painting with tincture of iodine. (5) The concurrent dyspepsia must be combated in the usual way, but the vomiting is often a very troublesome symptom, and there are three kinds of vomiting which admit of three different methods of treatment. (*a*) If preceded by nausea,

it points to disorder of the stomach, and should be treated by bismuth, etc., on the usual lines. (*b*) If the vomiting be preceded and caused by coughing, it is a good plan to give hot drinks just before a meal, in order to encourage expectoration and get the paroxysms of coughing over before the meal is begun. (*c*) If neither of these is successful, vomiting may sometimes be relieved by opium; sometimes it is controlled by the will. (6) The treatment of hæmoptysis, pneumothorax, and laryngeal ulceration is dealt with elsewhere.

(*e*) *Other measures not widely used.* *Tuberculin* is still tried by some for pulmonary disease. The evidence of its value is unconvincing. *Gold salts* were used experimentally by Koch; the modern forms are sanocrysin, myocrisin, crisalbine, etc. Some claim good results from their use; others, after extensive trial, believe that the improvement observed can be otherwise explained. All, however, agree that chemotherapy has yet a long way to go. Apart from these serious attempts to find a remedy, there are numbers of secret sera and proprietary nostra, widely advertised, which deceive the uncritical layman and waste money which would be better spent on good food and holidays. Streptomycin is still in the experimental stage and *sub judice*.

(*f*) *Preventive Treatment.*—The prevention of pulmonary tuberculosis is a wide and complex problem depending for its solution upon a proper appreciation of the principles which underlie our present conception of the pathogenesis of the disease. Preventive measures come under four main categories: (1) the public health organisation, (2) the part played by the general practitioner, (3) the education of the layman, and (4) the education of the medical student.

The public authorities have made good provision for the adult and in some cases for the child in the active stages of the disease, though more might be done in the way of observation of contacts and of suspects who are not actually notified. Much has been achieved in the attempt to educate the public as to prevention of the communication of the disease from man to man and of its extension from animals to man. The proper disposal of sputum from infected patients is of great importance, the patient being instructed to spit only into some portable receptacle containing a disinfectant such as lysol, or into paper sputum cups or handkerchiefs which can be burned. Phthisical patients should not share the sleeping rooms of healthy individuals.

Bovine tuberculosis is conveyed by ingestion of the milk or products of diseased cattle. In one research it has been shown that out of 2,825 cases in which the organism had been definitely typed, 1,040 (*i.e.*, 36·8 per cent.) were cases of pulmonary tuberculosis. Of these 2·3 per cent. were proved to be bovine in origin. Lange, in a series of 40 cases of pulmonary tuberculosis, found the bovine bacillus in the sputum in 20 per cent. In the light of modern work, it is important that raw cow's milk should be pasteurised before being used as a food for infants and young children. Adequate inspection of cattle is necessary, with authority to deal with

infected meat and milk. Good educative work has been done by the tuberculosis dispensaries, but more co-operation is desirable between the dispensaries, the sanatoria, the hospitals and the medical schools.

Mass radiography of the chest, carried out by experts, has been responsible for the detection of early pulmonary lesions in apparently healthy subjects; this method of examination should play a greater part in the machinery of preventive medicine in our national life. Thus the work of examination of contacts can be improved and effective steps taken in the early diagnosis of the disease and in the tracing and removal of the source of infection.

§ 132. **Chronic miliary tuberculosis** has recently been described. The clinical and radiological features of this are similar to those of the acute form described in § 117, but the course of the disease is much more protracted, extending in some cases even to two years. In some instances recovery has been recorded, radiographs showing complete resolution of the pulmonary lesions.

§ 133. **Fibroid Phthisis** is one of the chronic forms of pulmonary tuberculosis. It may be defined as a tuberculo-fibroid disease of the lungs, occurring for the most part in elderly subjects, running a protracted course, and terminating in contraction of the lung. This disease is very apt to be confused with chronic fibrosis of the lung (§ 136).

Symptoms.—The disease is essentially one of insidious onset and long duration. The patient complains of a chronic cough for many years. Later on this may become paroxysmal, and especially troublesome in the morning. Progressive shortness of breath, clubbed fingers, slowly increasing weakness and emaciation, with little or no fever, constitute the other symptoms.

The *Physical Signs* begin and are almost always most marked at the apex. *Both lungs* are usually affected (which contrasts with interstitial pneumonia), but the signs of disease are afterwards more advanced on one side. There is impairment of the chest movement and contraction of one side of the chest with signs of consolidation of the underlying lung. The heart, trachea, and other viscera are displaced to the more affected side. Hæmoptysis sometimes occurs, and the tubercle bacillus may be discovered on careful and repeated examination of the sputum or by guinea-pig inoculation.

The *Diagnosis* from other forms of *phthisis* is made by the extremely protracted course of this disease and the age of the patient. Non-tuberculous *pulmonary fibrosis* resembles it very closely, both in its physical signs and symptoms, and the diagnosis can only be inferred (i.) from the absence of the tubercle bacillus after oft-repeated examinations, (ii.) from the more usual localisation in one lung, (iii.) and from the history.

Etiology.—Fibroid phthisis is more frequently met with at and after middle life. It may follow chronic bronchitis, broncho-pneumonia, or repeated attacks of pleurisy. In true Fibroid Phthisis the tubercle bacillus is primarily deposited in a healthy lung under the same circumstances as in chronic pulmonary tuberculosis, and then causes an indolent fibroid reaction.

Prognosis.—Its course is very indefinite and protracted. Sometimes acute tuberculosis supervenes. The chief complications are bronchiectasis, compensatory emphysema of the lungs, lardaceous disease of other organs, and cardiac failure. In general terms the prognosis depends upon the same conditions as those of pulmonary tuberculosis and the *Treatment* is conducted on the same general principles.

II. *The patient complains of breathlessness; on examining the chest,* **dulness** *is found at one or both* **bases**, *and* SIGNS OF FLUID *are detected there. The disease is* HYDROTHORAX.

§ **134. Hydrothorax** is a chronic collection of serous fluid in the pleural cavity, differing from the effusion of pleurisy in being non-inflammatory.

Symptoms.—The general symptoms may be but little marked if the fluid is small in quantity. The onset is usually gradual. Dyspnœa is generally present, especially on exercise, but its degree depends upon the amount of fluid. As hydrothorax is always a secondary condition, the symptoms may be masked by the presence of dropsy elsewhere, and it is remarkable how often hydrothorax is overlooked on this account. In rare cases the fluid collects with great rapidity. The sudden onset of signs of fluid in the chest, accompanied by shock or collapse, in a case which has previously presented the symptoms of aneurysm, points to the occurrence of hæmorrhage into the pleural cavity (hæmothorax). The *Physical Signs* are those of fluid in the chest (*vide* §§ 109 and 119).

Diagnosis.—The disease has to be diagnosed from other disorders giving rise to dulness on percussion (§ 113). As regards *pleurisy*, hydrothorax is distinguished by the absence of pyrexia at the onset, by the absence of pain, and by the fact that the fluid occurs usually on both sides.

Etiology.—(i.) Hydrothorax may form part of the *general* dropsy of subacute nephritis, in which circumstance both pleuræ are usually involved. Here the hydrothorax is of no very great importance *per se*, but the onset of dyspnœa in nephritis should always direct our attention to the pleuræ. (ii.) Similarly, it may form part of *cardiac* dropsy, in which circumstances one pleura (the right) is often solely or chiefly affected. (iii.) Malignant disease in the chest is frequently attended by hydrothorax. In this case the fluid is often blood-stained, and sometimes, as in pleural carcinoma, may be found to contain cancer cells. (iv.) Aneurysm or other intrathoracic tumours pressing on the veins of the thorax may give rise to hydrothorax on one or both sides. In this condition also the effusion may be blood-stained.

Prognosis.—The disease is essentially chronic, the duration depending very much upon the cause. In general terms the prognosis of the condition is unfavourable. The patient should be carefully watched for the occurrence of shivering, sweating, or intermitting pyrexia, as indicative of empyema.

Treatment.—Paracentesis (§ 119) should be performed when the amount of fluid is such as to cause symptoms : in congestive heart failure it is wise to undertake this early. Tapping may be repeated indefinitely. Diuretics or circulatory stimulants are useful. For the rest, the treatment must be directed to the primary condition (see also § 119).

III. *The patient complains of breathlessness ; on examining the chest,* **dulness,** *usually slight, is found at one or both* **bases,** *and on auscultation,* FINE CREPITATIONS *are heard. The disease is* PULMONARY CONGESTION, HYPOSTASIS OR ŒDEMA.

§ **135. Hypostasis of the Lung** (Pulmonary Congestion or Œdema) is a serous exudation into and around the air vesicles. It is synonymous

with the term "hypostatic congestion," or, as it is sometimes called, "hypostatic pneumonia." It determines the end of many serious disorders.

Symptoms.—(i.) It is never a primary condition, and therefore our attention is first directed to the symptoms of its cause. The advent of hypostatic congestion is always insidious, and it is only by careful watching that it can be detected. (ii.) A considerable amount of dyspnœa is present, which may amount to orthopnœa. (iii.) There is a frothy mucous expectoration, not infrequently tinged with blood.

The *Physical Signs* are somewhat indefinite but they are found, as is implied by the term "hypostatic," chiefly at the bases of both lungs. The percussion note is somewhat impaired, and the air entry at the bases is diminished, and is attended by abundant moist crepitations.

Diagnosis.—The condition is diagnosed from true pneumonia by the gradual onset, the indefinite signs, and the absence, for the most part, of pyrexia, and other constitutional symptoms. Any rise of temperature that may be present is due to the primary or causal condition or to the development of broncho-pneumonia in the hypostatic areas.

Etiology.—(i.) The disease is most frequently met with in elderly people, especially when bed-ridden. (ii.) Pulmonary œdema complicates various blood disorders and fevers, especially typhus and typhoid fevers. In subacute nephritis œdema of the lungs occurs as part of a generalised dropsy. (iii.) Cardiac and other diseases, leading to mechanical dropsy, produce œdema of the lungs, sometimes acutely. (iv.) Tumours pressing on the veins within the mediastinum may result in pulmonary œdema. (For acute pulmonary œdema, see § 118.)

Prognosis.—The prognosis is always grave, because pulmonary œdema indicates either considerable impediment to the circulation in the lungs, or a serious toxic condition of the blood. It frequently terminates life in circulatory disorders, and in specific fevers of the asthenic type. In pneumonia it heralds a fatal issue.

Treatment.—The indications are to relieve the cause, if possible, and to stimulate the circulation. Ammonium carbonate and other stimulating expectorants promote expectoration. The liberal administration of alcohol and other diffusible stimulants is called for. The administration of 7% CO_2 with oxygen for ten minutes, two or three times a day, is valuable. In the aged, among whom even slight disorders are apt to be attended by pulmonary œdema, it is well *to keep the patient propped up* in a semi-recumbent posture. For the same reason it is advisable, in cases of fracture and other surgical maladies in the aged, to get them up as soon as possible, even at the risk of doing harm to their surgical ailment, so as to obviate the occurrence of hypostatic congestion of the lungs. The mercurial diuretics are of particular value in those cases due to myocardial weakness: when auricular fibrillation is present, digitalis is useful (§ 62). Should acute pulmonary œdema supervene, see § 118.

Group B.—We now turn to the chronic diseases attended by

dulness on percussion, which (*b*) does NOT always occur in regular and DEFINED AREAS AT BASE OR APEX. The *common* diseases in this group are: IV. PULMONARY FIBROSIS; V. THICKENED PLEURA; VI. and VII. NEOPLASMS.

§ 136. IV. **Pulmonary Fibrosis** may be localised or diffuse, according to the variety, running a protracted course, and resulting in contraction of the pulmonary tissue. It may be associated with bronchiectasis (§ 143).

An increase of the fibrous tissue of the lung may take place under the following conditions, all being chronic processes: (i.) An indolent tuberculous process. Fibrosis is one of the ordinary terminations of a tuberculous focus; but when the progress is very slow and protracted, with excessive formation of fibrous tissue, it constitutes true *fibroid phthisis* (§ 133). (ii.) The constant inhalation of dust in certain trades (*e.g.*, fustian cutters, jute workers, wool sorters, stone, knife, and other grinders, and silica workers, notably gold miners, asbestos workers, etc.) gives rise to a gradually progressive fibrosis of the lungs (pneumokoniosis). In all the diseases of this group there is a slowly progressive change in the mucous membrane of the respiratory tract, leading eventually to a diffuse fibrosis throughout the lungs. The most characteristic feature of silicosis is the formation of fibrous nodules, recognisable at a certain stage of the disease in the X-ray picture (Fig. 48), which bears a certain resemblance to that of miliary tuberculosis. There is a tendency in these cases to widespread infection with tuberculosis, which causes corresponding modifications in the clinical and radiological phenomena. (iii.) Repeated attacks of *pleurisy* may be attended by a subpleural fibrosis (thickened pleura (§ 137)), and dense bands of fibrous tissue may extend into the lung. (iv.) *Acute broncho-pneumonia*, becoming chronic, may result in a pulmonary fibrosis. The commonest cause of fibrosis in young persons is broncho-pneumonia following measles or whooping-cough. (v.) *Syphilitic disease* of the lung is rare, except as a congenital manifestation in infancy, in which circumstances the change consists of a fibroid induration of the lung. Gummata also occur. Only (i.), which is a *tuberculo-fibroid* process, should be called "fibroid phthisis." The other varieties constitute cirrhosis of the lung, and if they are invaded by the tubercle bacillus, they form a *fibro-tuberculous* process, which in its later stages may be indistinguishable from fibroid phthisis. Silicosis in particular is often accompanied by tuberculosis. In such cases the prognosis is much worse than in fibroid phthisis., (vi.) Fibroid changes in the lung may occur as a late phenomenon in some cases of Boeck's sarcoidosis (§ 141*a*).

The general *Symptoms* consist of progressive weakness and dyspnœa. There is no fever unless there is infection—a common occurrence in bronchiectasis. The *Physical Signs* may be found either at the base or the apex, though usually the former. Except in the variety due to the inhalation of silica, only *one lung* is involved as a rule, thus differing from fibroid phthisis, in which both lungs are usually affected.

There is deficient mobility of the diseased side, which later on undergoes contraction, so that there may be considerable difference in the measurement of the two sides of the chest. There is dulness on percussion. On auscultation, bubbling râles may be heard, but sometimes the only symptom is weak bronchial breathing or a weak respiratory murmur. Clubbed fingers are often present. The expectoration sometimes contains blood. The tubercle bacillus must always be looked for. Severe pulmonary fibrosis tends to displace the heart more than does fibroid phthisis. It is also commoner in the young.

The *Diagnosis* of pulmonary fibrosis from fibroid phthisis is sometimes very difficult, as may be seen by the description of the various processes just named. X-ray affords great assistance. The diagnosis and localisation of bronchiectasis has been particularly aided by the use of iodised oil B.P. (lipiodol), which is opaque to X-rays.

The *Etiology* of the condition is given above. It occurs chiefly in male subjects under fifty—especially between fifteen and thirty. A very common form of chronic fibrosis is that met with in persons engaged in trades attended by the inhalation of irritating particles. Sometimes it is a sequel to other pulmonary disorders.

Prognosis.—The general prognosis is unfavourable except when a foreign body is the cause, and can be removed. As regards the duration of life, the prognosis is good if the patient is not losing weight and the disease is not too extensive. The *complications* are dilated right heart, and emphysema occurring in other parts of the lung. Tuberculosis with silicosis is very serious.

Treatment.—The treatment of pulmonary fibrosis is that of the primary inflammatory disease of the lung which has caused it. A suitable climate is of importance. In the occupational fibroses, the work should be changed as soon as the condition has been diagnosed. In the tuberculous variety the chief concern is to prevent the heavy infection of contacts, especially small children. If fibrosis is associated with infected bronchiectasis the treatment is that of the latter condition, which is described in § 143.

§ 137. V. Thickened Pleura is a condition which sometimes succeeds dry pleurisy, especially recurrent dry pleurisy. It is more commonly seen in association with chronic empyema. It is important to recognise it, lest it should be mistaken for some more serious condition, though it is difficult to diagnose. It is more often localised to one part, and that most commonly at the apex, associated with chronic phthisis. *Physical Signs:* (i.) localised enfeeblement of the respiratory murmur; (ii.) dulness on percussion; and (iii.) diminished vocal resonance and fremitus.

The *diagnosis* is arrived at (i.) by the history of the case—*e.g.*, there has been an attack of pleurisy or pneumonia in the past—and (ii.) by the absence of signs of active disease when the patient is kept for some time under observation. The condition is often discovered only by chance, when the patient seeks advice for other ailments. It may be recognised by radiographic evidence of calcified plaques in the pleura. Treatment is

of no avail ; and, if only moderate in degree, the disease is not of much consequence. (See § 136, iii.)

§ **138.** VI. **Malignant Disease of the Bronchus.**—Since the war of 1914–18, post-mortem statistics both in Europe and America seem to prove that primary cancer of the bronchus has become more common. Carcinoma is more frequent than sarcoma. Beginning in the bronchial mucous membrane, cancer spreads to the parenchyma of the lung, forming a large intrathoracic tumour which gradually leads to death by pressure on surrounding structures. It is most frequent in middle age or in elderly persons, and is three to four times more common in men than women.

Symptoms.—(1) The onset is very insidious ; the case may long be mistaken for one of bronchitis. (2) Hæmoptysis, apparently causeless, in a middle-aged subject, suggests malignant disease. (3) Persistently recurring pleural effusion in a patient of middle age, with no history of tuberculosis, is a suspicious sign, especially if on exploration the fluid is found to be uniformly blood-stained. (4) Dyspnœa is frequent, often appears early, and is out of proportion to the physical signs found on examination. (5) Intrathoracic suppuration is sometimes an early sign. (6) Cough may occur at any time ; (7) pain, when it appears, is usually a late symptom. The *physical signs* vary with the stages of the disease and the size of the growth. Areas of dulness may occur early, especially over the right upper lobe, due to collapse of an area of lung when the main branch of a bronchus is obstructed. Gradually the lung is replaced by the malignant mass. There are often weak breath sounds, accompanied by many adventitious sounds, especially dry rhonchi and also dry friction as the pleura is involved. Moist sounds are audible when the growth breaks down. The heart and trachea are often displaced to the side of the affected lung, on account of the collapse due to obstruction of the bronchus. The lymph nodes are involved and sometimes the glands above the clavicles become hard, painless masses, easily seen and palpated.

The *diagnosis* rests upon careful consideration of the whole clinical picture—the history, symptoms, physical signs, systematic X-ray investigation (Fig. 47), and in doubtful cases a direct examination by means of a bronchoscope. The radiographs may be conclusive, but in some cases it is necessary to inject iodised oil into the trachea in order to obtain details required for diagnosis. Sometimes it is possible to remove a superficial gland, when histological evidence of carcinoma is found.

Prognosis and Treatment.—Up to a few years ago the prognosis was usually regarded as hopeless, despite the combined resources of surgery, deep X-ray therapy, radium, and remedies such as lead and selenium. Although the outlook is still grave, the prospect of achieving real success by surgery is now considerably improved ; more and more cases are recorded of the successful removal of a malignant growth by lobectomy or pneumonectomy, the patient being alive and well some years after the operation. Deep X-ray therapy has an undoubted place in treatment as a valuable palliative in inoperable cases.

VII. **Secondary Malignant Disease of the Lung** occurs as numerous, scattered, more or less circumscribed nodules, and is always secondary to cancer of the breast or abdominal organs. Sarcoma of any part of the body may give rise to secondary deposits in the lungs.

Symptoms.—The most common symptoms are increasing dyspnœa, cough, and cyanosis. Much respiratory distress occurs when a large area of lung tissue has become involved. Miliary carcinomatosis, which is usually secondary to carcinoma of other organs, especially the stomach, causes the greatest dyspnœa and cyanosis (Assmann), apart from that due to stenosis of a large bronchus. The *physical signs* are often very indefinite. When a large surface of the pleura is involved, the first sign may be an effusion into the pleura; on exploration the fluid may be found to be hæmorrhagic.

(*b*) We now turn to the less common and the rare chronic diseases attended by **dulness on percussion,** not always in regular or DEFINED AREAS AT BASE OR APEX. These are: VIII. COLLAPSE OF THE LUNG; IX. HYDATID CYST; X. SYPHILIS OF THE LUNG; XI. Some of the DISEASES DUE TO FUNGI AND PARASITES.

§ 139. VIII. **Collapse of the Lung,** or **Atelectasis,** is a condition in which the lung tissue is in an unexpanded state. The term "atelectasis" is usually applied to lung tissue which has never properly expanded, a congenital condition, due to imperfect development. The term "collapse of the lung" is applied to lung tissue which has previously expanded, but in which the air vesicles have subsequently collapsed.

Atelectasis is a *congenital* condition. The *symptoms* occurring in the new-born child consist of cyanosis, with shallow, rapid respiration. The lower part of the chest is drawn in by each respiration. On auscultation, the respiratory murmur is found to be very faint.

The *Symptoms* of **collapse of the lung** follow and complicate those of the disease which has led to the condition; for instance, the patient may not recover so rapidly as he ought, or the breathing is more embarrassed than can be accounted for by the concurrent disease in the chest. The physical signs vary considerably with the degree of collapse. Thus:

(*a*) In *complete* collapse of a part of the lung, as, for instance, in collapse due to compression or complete obstruction of a bronchus high up, there is impairment of the percussion note, with a diminution or absence of the breath sounds and of the vocal resonance and fremitus.

(*b*) Where the collapse is only *partial* in degree—*e.g.*, where the bronchi remain patent, as occurs sometimes when the lung is compressed by pleural or pericardial effusion—there are signs resembling those of consolidation (§ 109), except that the percussion dulness is not so marked, and the breath sounds, though bronchial in character, are somewhat feeble.

(*c*) Where the collapse is *slight* and limited, the chief sign is an enfeebled respiratory murmur. During deep inspiration fine rustling crepitations are heard, due to the expansion of the collapsed vesicles.

The *Diagnosis* is made usually by the existence of a causal condition.

When this is detected, attention may then be directed to the physical signs in the lungs. It will be observed that the signs of partial collapse resemble the signs of consolidation, and those due to slight collapse resemble early pneumonia. If the collapse is extensive the heart and mediastinum are drawn over to the side of the lesion. In addition to the evidence supplied by physical examination is that given by radiography, the airless portion of the lung appearing as a more or less homogeneous opacity. In cases of localised collapse there may be little or no evidence beyond that afforded by X-ray examination. Now that routine X-ray examination has become a matter of practice, localised pulmonary collapse, without symptoms, appears to be far more frequent than was originally supposed. It is particularly common in children, despite the absence of obvious clinical manifestations of disease.

Etiology.—The causes are of four kinds: (*a*) Causes which produce *obstruction*, such as (i.) a tumour at the root of the lung (*e.g.*, aneurysm); (ii.) obstruction in the throat (*e.g.*, the larynx); (iii.) stricture of a bronchus (*e.g.*, new growth or gumma); (iv.) secretion obstructing the bronchi; (v.) foreign bodies obstructing the larynx or bronchus. (*b*) *Compression* of the lung may be produced by pleural or pericardial effusion, an enlarged heart, or tumours of the mediastinum or of the abdomen. The condition may be the result of gross spinal curvature. It may occur after abdominal operations and anæsthesia (*acute post-operative collapse*), and give rise to difficulty in diagnosis, unless the possibility of its occurrence is borne in mind (§ 125). (*c*) *Paralysis* of the intercostal muscles or diaphragm, as in diphtheria or other cause of neuritis. (*d*) *Injury* to the chest wall with or without involvement of the thoracic contents, especially by high velocity projectiles, is a fertile cause of collapse of lung either on the same or the opposite side.

In *adults* collapse is most often met with as the result of pleural effusion or tumours in the chest; in *children*, as the result of bronchitis or slight catarrhal affections of the respiratory tract.

Prognosis.—The course of the disease depends very much upon the cause. Recovery as a rule soon takes place after compression by effusion, obstruction or stricture of the bronchi, and throat affections.

The *Treatment* is unsatisfactory. It should be directed to the removal of the cause, and especially to the recovery of any concurrent pulmonary disorder. That form which yields best to treatment is met in children with bronchitis. In adults, respiratory exercises should be given.

§ 140. IX. **Hydatid** of the lung and pleura is much more common in the Argentine and Australia than in this country. Generally cysts are single and tend to involve the base of the lung, especially on the right side. Clinically they remain latent in 75 per cent. of cases till the supervention of some complication like hæmoptysis, rupture or suppuration. A history of the expectoration of "grape skins" is pathognomonic; cough is frequent. The physical signs resemble pleural effusion, but the area of dulness is localised and has a rounded outline. Apical hydatid simulates tuberculosis, but the pulse is slower and fever absent or less marked.

The *Diagnosis* of deep cysts is often impossible until X-ray reveals the characteristic spherical shadow surrounded by translucent lung tissue. Eosinophilia is

generally absent, but the intradermal skin test is almost invariably positive, the complement fixation and precipitin tests less frequently so. The sputum may contain hydatid elements such as hooklets, scolices or laminated membrane.

The *Prognosis* largely depends on the presence or absence of complications. Many cases undergo spontaneous cure by rupture into a bronchus; others may be drowned in the process. Rupture into the pulmonary artery or heart is fatal. Secondary infection may lead to pulmonary abscess or empyema. Cysts of the liver frequently coexist (§ 347).

Treatment is surgical: resection of the adjacent ribs, incision and evacuation of the cyst content with or without drainage. Aspiration is dangerous owing to the danger of flooding the bronchial tree.

§ 141. X. yphilis of the Lung.—In infants this disease may take one of two forms: (*a*) The pneumonic condition of lung, found in infants, usually still-born, is regarded as an interstitial pneumonia of syphilitic origin. (*b*) Gummata are occasionally met with in the lungs of infants who are the subjects of hereditary syphilis; still more rarely they are met with in adults. Dyspnœa is usually the only symptom. The signs are those of consolidation and collapse. In adults syphilis of the lungs may take other forms—*e.g.*, broncho-pneumonia, bronchiectasis, etc.—and may lead to extensive infiltration and breaking down, or to fibrosis. Pulmonary syphilis is rare enough to be a curiosity. For mediastinal gummata see § 81. V.

§ 141a. XI. **Sarcoidosis** (Besnier-Boeck-Schauman Disease).—Much attention has been directed in recent years to the pulmonary manifestations of this generalised disease which may involve skin (§ 647), mucous membranes of nose or throat, parotid and lachrymal glands (§ 9), tonsils, uveal tract, lymphatic glands, lungs, bones, and internal viscera such as the spleen and alimentary tract.

IN THE CHEST the most constant feature is enlargement of the hilar glands. In addition, there is a general reticulosis which may progress to an interstitial fibrosis of the lungs with a tendency to nodulation. Occasionally, diffuse and confluent parenchymatous infiltrations are seen throughout the lung-fields.

Symptoms.—Patients may be symptomless, the condition being discovered by routine X-ray examination. The most frequent symptom is dyspnœa: there may be slight constitutional disturbance, *e.g.*, pyrexia, lassitude, and anorexia, with occasional cough. If the condition progresses to an extensive fibrosis, dyspnœa is likely to become more severe, and the patient may even be cyanosed. The radiological picture is that of a diffuse reticulosis with or without interstitial fibroid changes, and with marked enlargement of the hilar glands.

Etiology.—Typical sarcoid lesions consist of collections of pale staining epitheloid cells among which giant-cells occur. The main distinction between these lesions and tubercles is the absence of caseation and of tubercle bacilli. The three chief hypotheses as to the pathogenesis of these lesions are that they are (*a*) due to tuberculosis of an atypical and anergic form, since the Mantoux reaction is negative; (*b*) a non-specific tissue response called forth by a variety of pathogenic organisms such as the tubercle and lepra bacilli; (*c*) a manifestation of a systematised disease of the reticulo-endothelial system, similar to lymphadenoma and of unknown etiology.

The diagnosis is from miliary tuberculosis, silicosis and other forms of industrial lung disease, and secondary carcinomatosis. In an X-ray the enlargement of the hilar glands is the most constant and reliable feature. Schaumann noted a relative monocytosis: eosinophilia has been reported by others: an increase in the serum globulin has also been noted. The diagnosis has been established in some cases by liver biopsy.

Prognosis.—Usually this is favourable, the disease pursuing a chronic course and tending to recovery. Death has been recorded from heart failure supervening on progressive pulmonary fibrosis: in some cases, there is a terminal development of active tuberculosis.

Treatment.—The disease is uninfluenced by any treatment.

XII. Diseases due to Fungi and Parasites are recognised on examination of the sputum, and are referred to in §§ 145 and 146.

Group C.—Chronic Diseases attended by **Hyper-resonance** on percussion: I. In quite nine out of ten cases of hyper-resonance it exists on both sides, and is due to Emphysema. Other conditions which give rise to it are: II. Pneumothorax (§ 126); III. Skodaic Resonance above the level of an effusion (§ 108 and Fig. 49). The diagnosis is given in the form of a table (Table VIII).

Table VIII.—Causes of Hyper-resonance.

Cause.	Hyper-resonance.	Auscultation.	Other Diagnostic Features.
I. **Emphysema.**	Bilateral and universal.	R.M. distinct but weak and expn. much prolonged; signs of bronchitis, if present.	Barrel-shaped chest, cardiac dulness obscured.
II. **Pneumothorax,** An acute condition.	Hyper-resonance always unilateral, though it may extend beyond middle line.	Absence of R.M. and V.F. over affected area; sometimes amphoric breathing. Bell sound.	Organs displaced.
III. **Skodaic Resonance** —*i.e.*, the high-pitched note above a large *pleuritic effusion*, when the lung is otherwise healthy.	Unilateral; level may shift with position of patient.	Loud R.M.; V.F. felt over affected area.	History of pleurisy; signs of fluid in lower part of chest.

I. *The patient has complained of breathlessness for some years. There is* **hyper-resonance** *on* **both** *sides of the chest. The disease is* Emphysema.

§ **142. Emphysema** is a chronic non-febrile disease of the lungs in which the air vesicles become hyper-distended, the walls separating each vesicle become atrophied, inelastic and ruptured. As a result the aërating surface is greatly diminished, and the lungs are deficient in their elastic recoil.

Symptoms.—(1) The onset of the disease is imperceptible, and generally supervenes gradually after repeated attacks of bronchitis, the patient becoming more and more breathless after each attack. (2) This breathlessness is practically the only symptom, and it differs from all other kinds of breathlessness in this, that the chest remains *permanently in the inspiratory position*—in other words, owing to the inelastic state of the lungs and the shape of the chest, the patient finds it more difficult to expire than to inspire. A certain degree of cyanosis is usual. (3) Symptoms of bronchitis are *nearly always present.*

The *Physical Signs*, expressed *briefly*, are a barrel-shaped chest, hyper-resonance, and prolonged expiration. The shape of the chest is special to emphysema (*cf.* § 106).[1] The chest assumes permanently the shape of

[1] According to Cabot (*Physical Diagnosis*, 10th Ed., p. 309, London, 1930, Baillière, Tindall and Cox), the condition of "Barrel-chest," though characterised by a definite clinical syndrome, hereinafter described, is not necessarily associated with macroscopic emphysema, and may be due to abnormal conditions of the chest wall.

a healthy chest in a position of deep inspiration. The antero-posterior diameter is considerably increased. The hyper-resonance is always bilateral, and it obscures the dulness of the neighbouring organs—namely, the heart, the liver, and the spleen. These last two organs are also displaced downwards. The apex beat may not be palpable, but epigastric pulsation is usually felt. On auscultation, the respiratory murmur is modified; the inspiratory sound, which is full, is followed by a pause, and then by a prolonged expiratory sound. There are no adventitious sounds proper to emphysema, but, as just mentioned, bronchitis (*q.v.*) nearly always accompanies it. The heart sounds, especially at the base, may not be heard, or only with difficulty. Well-established emphysema interferes considerably with the pulmonary circulation, on account of the ruptured alveoli, and consequently the right side of the heart in course of time becomes dilated.

Variety.—In old people there is sometimes hyper-resonance with weak breath sounds, but no enlarged barrel chest; this is called *Atrophic* Emphysema, and is due to the giving way of degenerate air vesicles.

Etiology.—(i.) The condition occurs usually in elderly subjects. Both sexes are affected, but it is much commoner in males owing to the prevalence of bronchitis and asthma in them. (ii.) Heredity is said to play no part in the disease, but undoubtedly a hereditary tendency can frequently be traced. (iii.) The disease is frequently associated with senile degeneration, chronic nephritis, and cardio-vascular changes. (iv.) Bronchitis is the most frequent of the exciting causes, owing to the prolonged coughing and straining to get up phlegm, and owing also to the blocking of certain tubes with thickened secretion, which prevents the access of air to some alveoli, and unduly distends others. (v.) Asthma is also a potent exciting cause, owing to the constant strain on the elastic tissue of the lungs.

Prognosis.—Patients may live with emphysema to a good old age, and provided it is only moderate in degree it does not necessarily shorten life, though it predisposes to, and adds to, the seriousness of other pulmonary disorders. The gravity of any particular case is best measured by the extent of cardiac involvement (*q.v.*).

Treatment.—The indications are: (i.) To relieve the accompanying bronchitis (see § 129); (ii.) to improve the cardiac condition. The diet is of considerable importance in advanced cases, for any distension of the stomach greatly adds to the respiratory distress. It is a good rule never to let patients take a solid meal later than two o'clock in the day; otherwise their nights become considerably disturbed by the breathlessness. Ephedrine in repeated small doses (*e.g.*, gr. $\frac{1}{4}$) often relieves the respiratory distress.

Group D.—There are three chronic pulmonary conditions in which the percussion note varies considerably in different cases, but the **character of the sputum** suggests their presence—viz.: I. Bronchiectasis and Fœtid Bronchitis; II. Gangrene and Abscess of the Lung

(*vide* § 144). In Abscess the sputum is not invariably offensive. III. ACTINOMYCOSIS and other diseases due to fungi and parasites affecting the lung can usually be diagnosed only by examination of the sputum.

§ 143. I. **Bronchiectasis** is a cylindrical or saccular dilatation of the bronchial tubes. In order clearly to understand the significance of this it must be remembered that bronchiectatic dilatation is a pathological process, usually accompanying or resulting from some other morbid condition, and not strictly speaking a disease *sui generis* (see § 136).

Pathogenesis.—Traditional teaching has hitherto attributed the development of bronchiectasis to two main factors—(1) weakening of the bronchial wall by infective processes, with consequent loss of elasticity, and (2) increased intra-bronchial pressure due to excessive coughing. This view is still held by most teachers, but of late years increasing attention has been given to the association between bronchiectasis and pulmonary collapse, and many cases of so-called atelectatic bronchiectasis have been demonstrated in which bronchography has shown the presence of dilated bronchi within a collapsed lobe. So frequent is this association that atelectasis is now admitted to be one of the factors concerned in the pathogenesis of bronchiectasis. According to certain recent observations, partly clinical, partly experimental, collapse of the lung is regarded as an invariable antecedent of bronchial dilatation, the traditional theory of infection as a primary cause being considered no longer tenable.

According to this view, which is not yet generally accepted, bronchiectasis, cylindrical or saccular, is in itself a symptomless condition, only demonstrable by bronchography. If and when the factor of infection supervenes, symptoms arise, and eventually there appears the typical clinical picture, familiar in text-books. The following description applies to cases in which infection of the respiratory tract has occurred: we have as yet no accurate knowledge of the incidence of infection in pure atelectatic bronchiectasis.

Symptoms.—Although bronchiectasis may exist for long without sputum formation, sooner or later this occurs; in characteristic cases there are bouts of coughing at intervals varying from a few days to a few hours, accompanied by expectoration of a large quantity of sputum. This is purulent owing to secondary infection, and sooner or later may become extremely fœtid and offensive owing to stagnation and to the presence of saprophytic and/or anærobic organisms. The cough and expectoration are often started by some change of posture, and are therefore frequent when the patient wakens in the morning. In early cases, even with little sputum, the characteristic odour of the breath may be detected, but this may be absent. In advanced cases the total amount of sputum expectorated during 24 hours may be considerable (up to 20 ounces or more). In such circumstances the fœtor is usually pronounced, but this depends on the bacteriological factors present. Constitutional disturbance is frequent, and in bad cases there may be considerable pyrexia, sweating and loss of flesh. Often the sputum is found on standing to have become divided into three more or less distinct layers, the upper one frothy and muco-purulent, the middle more fluid, and the lower one chiefly pus, in which may be found "Dittrich's plugs," which consist of the debris of leucocytes, fat, and epithelial cells; crystals of fatty acids and numerous organisms may also be present. Hæmoptysis,

occasionally an early symptom, may occur at any stage, and in advanced cases may be considerable, often causing a diagnosis of phthisis.

Dry (hæmorrhagic) bronchiectasis (Syn.: Silent Bronchiectasis). In this form of the disease, which is now recognised as a clinical entity, infection has not occurred, and consequently the clinical picture above described does not appear. The condition is characterised by hæmoptysis of varying severity occurring at intervals; between these attacks the patient exhibits neither symptoms nor abnormal physical signs. This variety of bronchiectasis, which is one of the possible causes of sudden hæmoptysis, is only demonstrable by bronchography.

Polycystic disease of the lung (Syn.: Congenital Cystic Disease of the Lung), resembles bronchiectasis or, rather, bronchiolectasis, with which it has been identified by some authors, though others regard it as etiologically distinct. The chief *symptoms* are cough, shortness of breath and recurrent hæmoptysis. The X-ray appearances are characteristic and give rise to the so-called "soap-bubble" lung.

Solitary cysts of the lung are frequently recognised on X-ray examination. Usually clinical symptoms are absent, but hæmorrhage and infection may arise in the cysts.

Physical Signs.—These necessarily vary according to the degree of structural damage. When sufficient to cause definite physical signs, we meet chiefly those of fibrosis and retraction of the lung, with or without cavitation; co-existent are the signs of accompanying bronchitis. Cyanosis is common. Clubbing of the fingers is almost always present, and varies in extent according to the degree of infection and toxæmia.

Diagnosis.—Chronic cough, aggravated by movement or change of posture, and accompanied by the typical offensive sputum, is usually sufficient to distinguish the presence of bronchiectasis, especially when accompanied by the physical signs of fibrosis and excavation. In earlier cases, where there is little structural damage to the lung tissue, and little infection, the diagnosis may only be established by radiology; without the aid of iodised oil (*vide* Fig. 44), even an X-ray film may fail to show appearances which are pathognomonic. It is sometimes difficult to distinguish bronchiectasis from abscess of the lung, especially as the two conditions are frequently associated and are often part of one pathological process (see § 144).

Etiology.—Fibrosis and bronchiectasis are often said to occur after the broncho-pneumonia of measles and whooping-cough. There is good reason to believe, in the light of modern observations, that previous illnesses which have been hitherto regarded as attacks of pneumonia have in reality been attacks of massive collapse of the lung. Where there have been several alleged pneumonias, it is likely that a condition of atelectatic bronchiectasis has occurred upon which periodic infections of the respiratory tract have supervened. A foreign body or neoplasm, a tumour, aneurysm, or syphilitic stricture may cause blockage of a bronchus, with consequent pulmonary collapse, and bronchiectasis. In these cases infection frequently follows with the clinical picture already described.

Prognosis.—The condition is a very serious one, and is often incurable. The patient may live for many years. The prognosis is much worse

in bilateral cases, and in cases associated with extensive disease of the lungs or pleura.

The *Complications* which may occur are fatal hæmorrhage, gangrene of the lung, broncho-pneumonia, cerebral abscess, and pyæmia.

Treatment.—Until a few years ago the treatment of bronchiectasis was purely medical, the early attempts at radical surgery being attended by a very high mortality. The development of thoracic surgery in the last few years has resulted in such improvement in operative technique as to make lobectomy the treatment of choice in properly selected cases, *i.e.*, where the disease is unilateral and localised in one lobe. For extensive bronchiectasis involving more than one lobe of a lung, total pneumonectomy has given satisfactory results. Thoracoplasty is a possible alternative to pneumonectomy, and is a less severe procedure, but it does not offer so good a prospect of radical cure. Postural drainage and bronchoscopic aspiration are important pre-operative measures when toxæmia is a marked feature ; in any case a preliminary period of rest for a few weeks is desirable before a major operation is undertaken. When operative measures are contra-indicated, or when the patient will not submit to surgery, medical treatment can relieve the distress occasioned by large quantities of offensive sputum, and can build up the general health. Penicillin, administered by the inhalation method or by subcutaneous injection, may be of real help in combating toxæmia due to penicillin-sensitive organisms. The usual stimulant expectorants may be given at intervals to assist expectoration, and postural drainage should be carried out systematically. Liberal inhalations of turpentine, coal tar or creosote should be given on account of their antiseptic and deodorant effects. The most valuable form of treatment is the creosote vapour bath. Patients (whose eyes are protected by closely fitting goggles) are placed in a more or less air-tight room in which crude creosote is volatilised by placing it in a shallow iron dish which is supported on a stout ring and heated by a Bunsen burner. Terebene or creosote (refined) may be given by mouth in capsules (3 to 5 minims of the oil) three times a day. A sedative cough linctus may have to be given if sleep is much disturbed by cough. General tonics such as arsenic, nux vomica, cod-liver oil, etc., are indicated for cases with debility, anorexia, etc., and open-air treatment should be carried out when possible.

§ 144. II. **Abscess** and **Gangrene of the Lung.** *Symptoms.*—The clinical history is often indefinite, though (i.) cough is usually an early and prominent symptom. (ii.) The sudden expectoration of a considerable quantity of offensive sputum, followed by a sudden drop in the temperature of a patient previously febrile, suggests the presence of an abscess which has discharged into the bronchus. (iii.) Pain is a variable symptom ; it is seldom severe. (iv.) Clubbing of the fingers develops rapidly in many cases.

The *physical signs* are usually those of consolidation, but vary according to the stage of the disease and the extent to which the abscess has drained by rupturing into the bronchus. There is usually little or no displacement of the heart and mediastinum.

X-ray findings vary. In the earlier stages, before there is extensive breaking down of the lung, the skiagram may show only a dense homogeneous opacity. If a definite cavity has formed, it may be possible to see a fluid level, which remains horizontal in spite of changes in the position of the patient. This phenomenon is practically diagnostic of abscess.

Etiology.—Between these two forms of intrathoracic suppuration it is hardly possible to draw a sharp line of distinction; both result from invasion of the lung tissue by pathogenic organisms, and the consequent damage varies in character and extent according to (*a*) the nature of the invading organisms and (*b*) the virulence-resistance ratio of the individual. Another factor which determines the character of the inflammatory changes in the lung is the blood supply of the particular part affected. In the case of rapid and extensive necrosis of the lung parenchyma, for which the term gangrene should really be reserved, the effects of an unusually virulent toxin are probably aided by the vascular occlusion which accompanies the process. A gangrenous necrosis frequently precedes abscess formation; on the other hand, a lung abscess may arise insidiously, without evidence of previous acute inflammation. For practical purposes it is convenient to consider abscess and gangrene of the lung under one heading.

The distribution of lung abscess may be single or multiple. Single abscesses are more often found in the right lung than in the left, and in the lower more often than in the upper lobe. If abscess formation in the lung is a result of embolism following septic infection in other parts of the body, the lesions are likely to be multiple. Two main causes of intrapulmonary suppuration (abscess and gangrene) are: (i.) aspiration of infective material into the lower respiratory tract; (ii.) embolism. There are two schools of thought, each favouring one of the above causes; probably both play a part. The development of a lung abscess often follows operations upon the mouth, nose and upper respiratory tract, when it is probably due to inhalation of blood, mucus, or portions of soft tissue into the lower respiratory passages. (iii.) In other cases abscess formation follows the occurrence of a pulmonary infarct. Sometimes it complicates pneumonia. Often there is no history of previous acute illness; the patient comes with an obvious abscess of the lung, for which no definite cause can be found, the symptoms having gradually and insidiously increased for many weeks. (iv.) An underlying new growth is a possible cause. (v.) A liver abscess (especially the "tropical abscess"), or a suppurating hydatid may rupture into the lung. Any of the well-known pathogenic organisms may be present; streptococci, staphylococci, pneumococci, and various saprophytic organisms. Anærobic organisms also should always be sought for, especially in cases of acute and rapid gangrenous necrosis of the lung.

The *Prognosis* is serious, but with early diagnosis and skilled surgical intervention the outlook is by no means hopeless.

Treatment. As above mentioned, an abscess frequently ruptures into

a bronchus, leading to spontaneous cure; if there is free drainage of pus in this way, there is seldom immediate need for surgical intervention. If, however, drainage is inadequate, or if no pus has been coughed up, and the patient still shows evidence of toxæmia, surgical drainage by a two stage operation must be instituted. Artificial pneumothorax has been advocated, but carries with it a grave risk of rupture of the abscess into the pleural cavity; where drainage through a bronchus is in progress it might conceivably be useful. In practice the actual results of pneumothorax treatment of lung abscess have been almost always unsatisfactory, and frequently disastrous. As in other infections of the respiratory organs, the value of penicillin in addition to any necessary surgical measures is becoming increasingly apparent.

§ 145. III. **Actinomycosis** may affect the pleura or the lung, imitating the signs of empyema, pneumonia (§ 121), phthisis, or bronchiectasis. In the absence of cutaneous or other lesions it is rarely diagnosed except by an examination of the sputum, when the little yellow pellets containing the ray fungus are visible. The streptothrix may by cultured anærobically. The disease is usually fatal.

§ 146. IV. **Paragonimiasis,** caused by *Paragonimus westermani*, the common lung fluke of Japan and China, gives rise to pulmonary symptoms, including cough and hæmoptysis. The physical signs may suggest bronchiectasis, broncho-pneumonia or pleurisy, the diagnosis being made by finding operculated eggs in the rusty-brown sputum. Diarrhœa and Jacksonian epilepsy due to involvement of the intestine and brain respectively may occur. No specific drug treatment is known.

ASPERGILLOSIS. The fungus *Aspergillus fumigatus* may cause signs resembling tuberculosis. The disease affects pigeon-feeders, who chew the seeds containing the fungus. It may undergo spontaneous resolution.

BLASTOMYCOSIS and SPOROTRICHOSIS may affect the lungs. Cutaneous and other lesions are usually present in addition (§ 648). ASCARIS infections may be hard to diagnose in early stages.

PSITTACOSIS, ANTHRAX, PLAGUE, GLANDERS and DISTOMA may affect the lungs, and can be recognised only by the sputum and concurrent signs. Psittacosis (§ 506) often resembles pneumonia.

Various OTHER FUNGI have been identified in association with broncho-pulmonary inflammations, *e.g.*, *monilia*, *geotrichum*, *coccidioides*, etc. These conditions are mostly seen in Eastern countries. The presence of *mites* has also been identified with asthmatic and other respiratory symptoms.