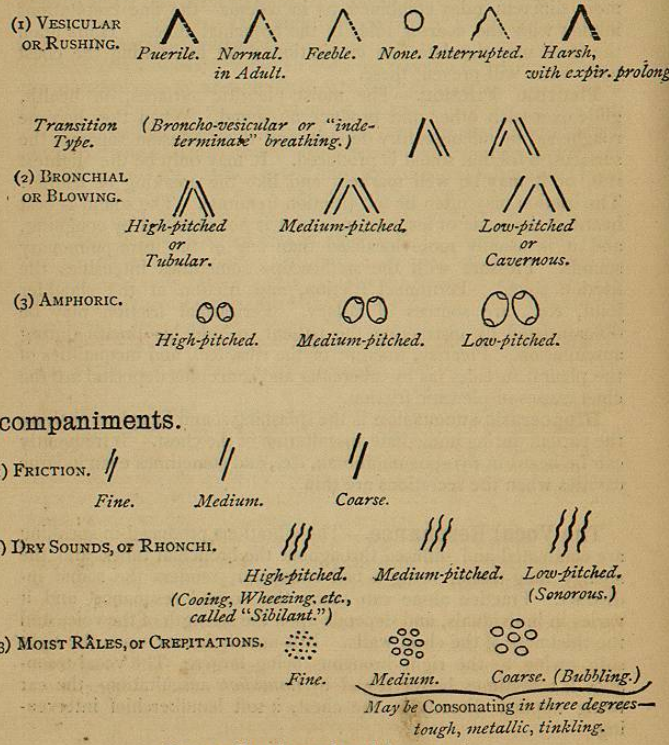
The *vocal resonance* may be—

Impaired:	Normal.	Increased:
(*muffled*)	...	(*Bronchophonic*)
(*absent*)	...	(*Pectoriloquous*)
...	...	(*Ægophonic*)
...	...	(*Amphoric*)

Assuming that the bronchial tubes are not blocked and that there is no interference with the conduction of the vibrations, the vocal

Types of Breathing.



Accompaniments.

Fig. 15.—Breath-sounds and Accompaniments.

resonance is impaired (muffled) or absent, when the lung from any cause is separated from the chest-wall (pleuritic effusions—liquid or gaseous—tumours, &c.). This is not *always* the case in pleurisy with effusion, as occasionally even the whispered voice may be heard over the dull part. The vocal resonance is intensified by whatever makes the lung approach nearer to simple solid, or simple air, as in all *consolidations* (pneumonia, phthisis, &c.), and especially is it intensified over *cavities*. *Bronchophony* is that clear resonance which is generally compared with that normally heard in the inter-scapular region. It has sometimes a *sniffing* character. *Pectoriloquy* is very clear bronchophony, heard usually over a limited area, and it is reverberating in its quality; but it does not always indicate a cavity. *Ægophony* is the bleating (Punch) voice sometimes heard in pleuritic effusions. *Amphoric* resonance is metallic in character, and it resembles the blowing or speaking into a large empty bottle, the sounds having a distinct echo. It occurs chiefly as a symptom of pneumothorax, but it may occur in large cavities.

The character of the vocal resonance (clearness) is more important than its actual *loudness*, and it sometimes is necessary to test the whisper over cavities, &c., as "the ground-tone of the voice drowns the articulated over-tones." The thoracic resonance may also be tested by the cough and cry—the latter especially in children. Dr. Wyllie graphically represents the breath-sounds and accompaniments, as on the preceding page.

CHAPTER IV.

DISEASES OF THE RESPIRATORY SYSTEM—

Section II.

Contents.—Pertussis—Asthma—Hay asthma—Acute bronchitis—Fibrinous bronchitis—Pulmonary collapse (Atelectasis)—Catarrhal pneumonia—Congestion and œdema of the lungs—Croupous pneumonia—Hæmorrhagic infarction—Chronic bronchitis—Cirrhosis of the lungs—Emphysema—Fœtid bronchitis—Bronchiectasis—Gangrene of the lung—Phthisis, and miliary tuberculosis—Cancer of the lung—Hydatids—Pleurisy—Hydrothorax—Pneumothorax, hydro-pneumothorax, and pyo-pneumothorax—Pleurodynia—**The classification and diagnosis of the diseases of the pulmonary organs**—Causes of hæmoptysis.

Pertussis — Whooping-cough.* — The pathology is still doubtful. It is admitted that the disease is the result of a specific organism, but its exact nature has not been made out. Burger and Afanassieff have found organisms, cultures of which, when injected

* Before reading the following diseases of the pulmonary organs, the student should revise the classification and the notes upon the same on p. 115.

into the windpipe of dogs and rabbits, have produced symptoms resembling whooping-cough—but other agents are known to do the same. Some attribute the disease to the action of the poison upon the vagus nerve. In simple uncomplicated cases there is found only congestion of the trachea and bronchi, with some emphysema and collapse of the lung. The age at which whooping-cough is common is from two to ten years; but it frequently occurs in adults. The period of incubation has been proved to extend to fourteen days.

The **symptoms** are at first those of ordinary catarrh, with a dry cough, and fever, and these symptoms may extend from a few days to three weeks or more before the characteristic spasmodic cough is developed. During this stage it may be impossible, in single cases, to make any positive diagnosis, but a dry incessant cough, which is worse at night, should always suggest the possibility of whooping-cough when it occurs in a child.

The *spasmodic stage* is reached when the cough becomes paroxysmal. It consists of a number of short expiratory puffs, succeeded by a long in-drawing crow or "whoop," the attack lasting about one minute, but sometimes even a quarter of an hour. It may be started by imitation of another child suffering from pertussis, by indigestible food, or inhalation of cold air, &c. There is often much distress. The expression is anxious and frightened, the face is swollen and often cyanotic, the eyeballs protrude, and the child usually clings to a chair or table during the violence of the shocks. The conjunctivæ are frequently ecchymosed, and hæmorrhage from the nose is common, while the attacks may terminate in expectoration of viscid mucus, in vomiting, or in general convulsions in very severe cases. Hernial protrusions are common. Small ulcerations at the frænum of the tongue sometimes appear at the third to the fifth week. Albumen and sugar are found in the urine frequently. The physical signs in the chest, in uncomplicated cases, are those of bronchial catarrh, viz.:—Sonorous and sibilant rhonchi, with coarse crepitations; prolonged harsh expiration; and no dulness upon percussion. The heart's action is very rapid during the paroxysm. There is little or no fever, and the child, during the spasmodic stage, appears fairly well between the attacks. This stage may continue from about four or six weeks to even two or three months. The whoop gradually disappears, but it may recur during the convalescence, with any fresh catarrh; and even, in some cases, it may occasionally be heard as long as a year after the attack. During the convalescence—sometimes described as a third stage—the symptoms and signs are those of ordinary catarrh, if no complications have supervened. The secretions are muco-purulent, the cough is frequent, but it is not spasmodic unless the patient relapse, which is very common.

The most frequent complications are bronchitis, catarrhal pneumonia, and collapse of the lung. Sometimes bronchiectasis is produced; while in exceptionally severe cases emphysema, and even extravasation of air into the pleura (pneumothorax), &c., may result

from whooping-cough. The cerebral complications (convulsions and coma) are the result of mechanical obstruction to the circulation. Cerebral hæmorrhage may result from rupture of a blood-vessel. In healthy children the *prognosis* is usually favourable if the disease be not severe; but it is serious in the very young, and death is very frequent—either the result of complications or of simple asthenia. Death is not common during the paroxysm, except in the very weakly and strumous; but in these it is always possible from syncope, or from intracranial hæmorrhage, &c. Whooping-cough seldom if ever recurs, and it is believed in some very mild cases it may run its course without any characteristic "whoop" or spasmodic stage.

The **treatment** of the first stage is that of ordinary catarrh. The child should be kept in a warm room and ℞ 14 or 15 should be prescribed, and the chest should be rubbed with Bowe's liniment, or a linseed poultice may be applied, if there be much bronchial catarrh. The diet should be simple and nourishing, and consist chiefly of milk. The bowels should be kept regular in action. During the spasmodic stage, bromides may be given, in a little syrup of Tolu, in doses according to the age. ℞ 16 and 17 are also useful remedies. Syrup of codeia is also a powerful and efficient drug in severe cases. Quinine is said to be serviceable. Emetics are useful when there is much mucus obstructing the bronchial tubes. Very dilute carbolic (1 per cent.), or a 2 per cent. solution of salicylic acid, may be sprayed into the throat. The air of the room should be medicated with eucalyptus oil, volatilised over a spirit lamp. In lingering cases tonic treatment with a change to the seaside may complete the cure. The treatment of the complications is given under their respective diseases.

Asthma.—The term *asthma* is frequently applied to diseases associated with breathlessness. Hence we have *cardiac asthma* when the dyspnœa of heart disease is marked, and *gastric asthma* when flatulence in dyspeptic conditions produces embarrassed breathing. The ordinary dyspnœa which accompanies emphysema and bronchitis is sometimes loosely spoken of as an asthma. "Asthmatic bronchitis" is a convenient term, clinically, as true spasmodic asthma is invariably followed by irritation of the bronchial tubes; or on the other hand, and far more commonly, bronchitis is the primary disease and is the cause of the reflex spasm. *True spasmodic asthma*, however, might be classified with the nervous diseases, as pathologically it is "a *neurosis* of the breathing apparatus"; but practically it is best considered with the diseases of the respiratory system.

The symptoms are produced by sudden spasm of the bronchial muscles and diaphragm, and the laryngoscope has revealed that deep congestion of the upper part of the windpipe takes place during an attack. A sudden tumefaction of the mucous membrane may also be a cause of the attack. Chronic inflammation of the bronchial tubes results from the repeated attacks extending over years. The chest becomes barrel-shaped. In subjects predisposed by inheritance, gout, &c., to attacks of asthma, certain odours,

dust, pollen, and indigestion and flatulence, are all exciting causes; and nasal polypi are also known to be a cause of asthmatical seizures. Long continued whooping-cough, chronic bronchitis, and severe cases of measles, by the depression produced in the respiratory organs, are believed to produce conditions favourable to the development of asthma.

Asthma frequently alternates in the same person, or in other members of the same family, with other nervous diseases and with skin eruptions, the most common being urticaria.

The **symptoms** come on very suddenly, and generally during the early morning hours; but in those subject to attacks there may be warning, as by ordinary coryza, bronchial irritation, general *malaise*, or indigestion and acidity, for a few hours previous to the seizure. There is a sudden feeling of constriction about the chest, and the patient struggles for air with the appearance of great anguish. Beads of sweat stand upon the forehead, the face is cyanosed, and the eyeballs are widely staring. This is accompanied by loud *expiratory* wheezing. The spasmodic contraction of the diaphragm keeps the chest in the position of forced inspiration, and percussion of the lungs yields a clear note (tympanitic or *band-box* sound) which often extends an inch or more *lower* than the normal area occupied by the lungs. The expiration is greatly prolonged, and the vesicular murmur is obscured by sibilant and sonorous râles, and, later, by moist crepitations. The spasmodic condition may last from a few minutes to several hours, when relief generally follows the free eructation of gas, or the expectoration of thin watery mucus; or the attack may terminate in the discharge of a large quantity of pale-coloured urine of a low specific gravity. The expectoration sometimes contains elongated plugs—"Curschmann's spirals." Very often the bronchitis lasts for some days with frequent exacerbations of the spasmodic asthma.

Frequent attacks of asthma and bronchitis ultimately may lead to emphysema of the lungs, with dilatation of the right cavities of the heart, and dropsy; but the *immediate* prognosis is usually hopeful, death being rare, unless the attacks be associated with grave organic disease of the heart. The attacks are sometimes mild, and at other times serious; and they may leave the patient altogether, especially when occurring in the young. With older patients the prognosis should be more guarded, and it must be considered in relation to the history of the case, the number of the previous attacks and their effects.

The **diagnosis** is not usually difficult. Œdema glottidis, paralysis of the vocal cords, aneurisms pressing upon nerves, or stenosis of the trachea, all produce dyspnœa; but it should be noted that in asthma the dyspnœa is *expiratory*, and laryngoscopic examination may serve to differentiate the former diseases. Cardiac dyspnœa, emphysema, and bronchitis have other physical signs; but in the latter disease asthma may occur as a complication, or, again, the bronchitis may be the result of a previous asthmatical seizure. The history of the *onset* is most important, and especially is this the case in children, for, as Trousseau has pointed out, many cases of supposed broncho-pneumonia are really cases of true spasmodic asthma, as shown by their rapid recovery and their frequent recurrence. The family history is also important as regards the diagnosis of asthma in children. Kidney disease should be remembered, and the possibility of asthma being due to this cause.

[**Hay asthma** or **hay fever** is sometimes described as a separate affection, the symptoms being caused by the irritation of the mucous membranes, similar to that which occurs in asthma. There is severe coryza with frontal headache and general *malaise*, and sometimes the irritation extends to the bronchial tubes, producing slighter forms of asthma. It is peculiar to certain individuals, and apt to resist treatment; but a short sea trip always results in recovery.]

In the **treatment** of true spasmodic asthma, *four* to *eight* drops of a 1 per cent. solution of nitroglycerine, in water, is the most effectual remedy for relief of the spasm in adults, care being taken to watch the heart's action. The dose may be repeated in three or four hours if there be no depression from its use, and it may be continued cautiously, at regular intervals, should the paroxysms render it necessary. Three to five drops of nitrite of amyl dropped upon blotting-paper and inhaled, may be tried in place of the above. Fifteen to thirty grains of chloral hydrate, with forty grains of bromide of potassium, may be given at the same time, or in place of the nitroglycerine, when it is deemed unadvisable to use it; or one-twelfth to one-fourth of a grain of morphia may be used hypodermically. Inhalations of ether or chloroform may be tried, and large doses (fifteen grains) of iodide of potassium are recommended. Stimulants are often necessary, and digitalis may be used along with these remedies when the heart threatens to fail.

Strong coffee to drink, or the smoking of cigarettes of stramonium, belladonna, &c., or the burning of nitre-paper, sometimes affords relief; and ten drops of either ipecacuanha wine, or of tincture of lobelia, in water, every five minutes, until some nausea is induced, may serve to lessen the feeling of oppression. The treatment after relief of the spasm is much the same as in bronchitis. The diet is important—light and easily digested animal food being ordered, while starchy foods, sweet stuffs, and even milk, should be avoided. Arsenic, quinine, and iodides are indicated in the treatment of chronic cases, between the attacks. The asthma of children ("chill and bronchitis") is best treated by change, and by sound hygienic considerations.

For *hay asthma*, antiseptic douches or sprays may be tried, if the sea-side or a short sea voyage is unattainable. Painting the mucous membrane of the nose with a solution of cocaine may be of service, and in obstinate cases, destruction of the Schneiderian membrane, by the electric cautery, may effectually cure the disease.

Acute Bronchitis. — The pathological changes in acute bronchitis begin with redness and swelling of the mucous membrane of the air-passages. The bronchial tubes soon become filled

with mucus and muco-purulent secretions. In the *early* stages the expectoration is made up of ciliated columnar epithelium and of mucous corpuscles; but, later, it consists of mucus holding in suspension large numbers of small, round, or oval-shaped cells, shed from the lower epithelial layers, and undergoing fatty degeneration. The basement membrane becomes swollen and œdematous, and the mucous glands are stimulated to increased secretion. There is accumulation of cellular structures and leucocytes within and around the inner fibrous coats of the bronchi, and the bronchial glands, in the immediate neighbourhood, become enlarged. Should recovery take place, the swelling of the basement membrane disappears, and the epithelial layers are replaced.

The causes of acute bronchitis are numerous. It is most common in damp and changeable climates, and in such, the feeble, aged, or very young are readily attacked. While exposure is the commonest cause, acute bronchitis arises as a secondary change in a

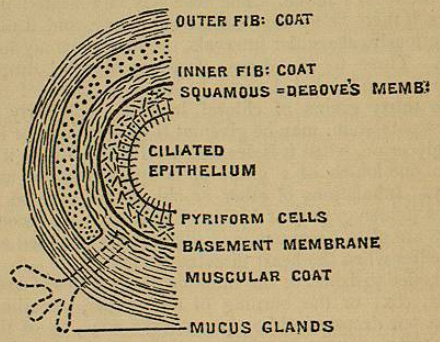


Fig. 16.—Diagram of bronchus.

great variety of diseases—*e.g.*, the exanthemata, rickets, gout, heart disease, diabetes, Bright's disease—all of which require to be remembered in the diagnosis. Irritating vapours in certain trades, dust, and micro-organisms may also set up the disease.

The symptoms vary according to the seat. If the trachea and larger tubes only be involved the symptoms are not so urgent as when the smaller tubes are affected, nor is the prognosis then so grave. In the first case—sometimes called *tracheo-bronchitis*—there is not much fever, and the chief symptom is a tickling cough with pain over the sternum. In the very mild cases, only the other symptoms associated with an ordinary coryza may be present; but if the bronchial tubes be still further affected there may be a considerable rise of temperature, greater frequency of the pulse, and some dyspnœa. The latter is chiefly *expiratory*. The accumulation of mucus in the tubes gives rise to râles which often may be heard

at a distance from the patient. The face is flushed, and in extreme cases there is cyanosis; but the latter symptom is well marked in the form involving the smaller tubes—*capillary bronchitis*. In bronchitis of the larger tubes the cough is very frequent and loud and the expectoration varies from simple glairy grey-white mucus to muco-purulent secretion, and sometimes almost pure pus. It may be scanty at first, but it soon increases, while it may be so very tenacious as to allow of the spittoon being reversed without its escaping. It becomes more watery under treatment, and in the later stages, should recovery take place. The tenacious character of the sputum, when in quantity, gives rise to a feeling of suffocation, hence the old name of "suffocative catarrh." The cough and suffocative sensations are worst in the morning when the accumulation of mucus interferes with the respiration. The patient often requires to be propped up in bed to allow of easier breathing.

The *capillary* form of bronchitis is common in children, and it is generally ushered in by severe chills, convulsions, or repeated rigors, and the temperature rises as high as 104° Fahr. The head and upper part of the body may be covered with perspiration. The dyspnœa is the marked symptom, and the pulse is extremely rapid, but often full. The expectoration may be absent in the early stages of capillary bronchitis; but it should be noted that in children the secretions are swallowed, if any be coughed up.

Inspection of the chest, in the severe forms of bronchitis with dyspnœa, reveals considerable indrawing of the walls, particularly in the epigastric and hypochondriac regions; and in children all the lower ribs are seen to be drawn in with the difficult inspiration. There is no change in percussion over the lungs in uncomplicated cases, but if emphysema, collapse of the lungs, or broncho-pneumonia be present, the percussion tones may be impaired.

In auscultation, the breath sounds are altered. The vesicular murmur may be harsh, indistinct, or obscured by râles, or absent if the bronchial tubes be blocked with mucus. In the latter case coughing may develop the breath sound. It is prolonged in its *expiratory* part. The accompaniments are the coarser forms of râles, with sonorous and sibilant rhonchi, the vibrations of which may sometimes be felt through the chest-wall. Coughing may alter the character of the accompaniments. The râles are general, and are not localised, a point of some importance in relation to the diagnosis of phthisis. They do not consonate. The vocal resonance remains normal. The heart-sounds may be distant, and the cardiac dulness reduced by the encroaching lungs.

Simple acute bronchitis may run a course of fourteen days or more, and then terminate in recovery. It may, however, in extreme cases, be terminated by death in two or three days, especially in the aged and feeble, and the very young.

The prognosis will depend upon the severity of the attack and the presence or absence of complications. These are chiefly collapse of the lung, emphysema, broncho-pneumonia, and sometimes pleurisy. The disease frequently becomes chronic; but if it run to

a fatal issue, the breathing becomes more and more shallow and convulsive seizures may terminate the case.

The diagnosis of acute bronchitis is not difficult; but it is sometimes not so clear whether the bronchitis be primary or secondary—or whether it be simple or complicated. Careful examination of each case is necessary, keeping chiefly in view the exanthemata, rickets, chronic valvular disease of the heart, Bright's disease, tuberculosis, and scattered malignant nodules; while collapse of the lung, emphysema, catarrhal pneumonia, pleurisy, and croupous pneumonia are complications which should be noted and excluded.

Fibrinous Bronchitis or **Plastic Bronchitis** is a rare disease, consisting of the formation of a tough white-grey membrane within the bronchial tubes. It is not an extension of croupous membrane from the larynx, although similar in character. The symptoms are at first like acute bronchitis, but there is excessive dyspnœa, and it is not usually diagnosed until the expectoration of the casts reveals the nature of the disease. The character of the cough suggests stenosis of the trachea, or a foreign body. The prognosis is very grave.

Pulmonary Collapse (Atelectasis).—*Atelectasis* is the condition of uninflated lungs (or any part of them) in the new-born infant.

Pulmonary Collapse is a *secondary* condition, and it results from the primary disease so affecting the lobules that they are deprived of air. Gairdner's theory is, that pellets of mucus within the bronchial tubes may act as ball and socket valves, admitting no air during inspiration, but allowing of its escape during expiration. As this theory does not explain the entire absence of air within the collapsed lobules, and as Lichtheim has shown that complete absorption takes place after plugging the bronchial tube, Fagge thinks "that whenever even a small part of the organ fails to be acted upon by the forces which are concerned in inspiration, its elasticity brings about a total collapse of its substance, notwithstanding that the tubes which serve it may be patent." This theory serves to explain how acute bronchitis in a child produces collapse—the drawing in of the chest-wall and sternum causing collapse of the anterior borders of the lungs, and yet no plugging of the tubes is present.

Collapsed lung is very like red hepatisation. The lung is brown-red in colour. A section, however, is dry, smooth, and homogeneous, and the portions affected are depressed below the surface of the healthy lung. The collapsed portions are more dense and do not crepitate, and they may be inflated from the bronchial tubes. Sometimes the collapsed portions are œdematous and serum may be expressed. *Carnification* of portions of the lung is the result of continued pressure upon the collapsed lung, as in pleuritic effusions, tumours, &c.

Any obstruction of the bronchial tubes by tumour growths, enlarged glands, or aneurism, may produce collapse; while it is, of course, associated with all large pleural and pericardial effusions, and

tumours which displace the lung. In acute bronchitis and in whooping-cough collapse is only common in children. It may only be temporary; and the important symptom is the increase of the dyspnœa superadded to the previous symptoms. Percussion will only reveal dulness when the collapsed portion is sufficiently large, and then, also, the other physical signs of consolidation will be present. Collapse of the lung may terminate fatally, or in recovery, more or less complete. If recovery be imperfect, it may leave damaged lungs with emphysema, or it may lead to caseous pneumonia and phthisis in those of the strumous constitution. Inhalations of compressed air, and active expansion of the chest by gymnastic exercises, should be practised as early as possible.

Broncho-pneumonia: Catarrhal Pneumonia: Lobular Pneumonia.—When an acute bronchitis extends back to the air vesicles, a catarrhal pneumonia is produced, an event which is somewhat rare in adults, but is common in childhood, especially after measles, other fevers, and whooping-cough. The *sub-acute* form, however, or "cold settling down in the chest," is common enough in adults of delicate health, and in the aged or feeble. It may lead to phthisis, if the weakened tissues be invaded by the tubercle bacillus. The lung, on *post-mortem* examination—besides showing some lobules in a state of collapse, and others compensatorily dilated, with likewise the signs of bronchitis already described—is seen to have grey-white, semi-solid patches, slightly elevated and scattered throughout its substance. They are not nodular, and, when squeezed, they emit a yellow muco-purulent discharge. Microscopically, the alveoli are seen to be infiltrated with large round cells, which are very granular, and have one or more nuclei. They are derived from the epithelial lining of the air vesicles, and they are in various stages of proliferation. The process closely resembles that of acute bronchitis, Debove's membrane in the bronchial tube running continuously into the alveolus. These inflammatory products may become absorbed if recovery take place; but very frequently, in bad hygienic conditions, the disease becomes more or less chronic, and caseation of the nodules ultimately leads to the phthisical condition. There are often small patches of pleurisy present, especially if the patches of catarrhal pneumonia be superficial; but, according to Hamilton, there is no pleurisy in the simple acute catarrhal pneumonia, but almost invariably is it present in the second stage, viz., of *caseation*.

In children, rickets and bad ventilation, &c., predispose to the disease; while in adults probably secondary irritation is the cause, as from fœtid purulent matter from the bronchial or nasal mucous membranes being drawn along the bronchial tubes. In children the violent inspiratory efforts drawing into the alveoli the morbid inflammatory products of bronchitis, has been suggested by Hilton Fagge as a highly probable explanation of their greater liability to catarrhal pneumonia. The diagnosis is often uncertain; but if superadded to the symptoms and physical signs of acute bronchitis, there be a sudden rise of temperature—especially if a finer râle be now detected—it may be surmised that the alveoli have become affected

by the general bronchial catarrh. If several contiguous lobules be involved, then the physical signs of consolidation—dulness on percussion, bronchial breathing, and bronchophonic resonance—may be made out. The pulse is increased in frequency, and in children it may be very rapid (150 to 200). The cough may cease, the child may become very restless, the breathing shallow, and the case finally terminate fatally. Empyema and tubercular meningitis may require differentiation; but it should be noted that in children pneumonia is often ushered in by convulsions in the very earliest stages of the disease. The absence of the bacillus tuberculosis in the sputum differentiates catarrhal pneumonia from phthisis. The *prognosis* is less grave after measles, but even this depends upon the constitution and strength of the subject. Catarrhal pneumonia may terminate fatally within a few days, or it may run a course of from two to three weeks, and still ultimately recover. The sub-acute and chronic forms of catarrhal pneumonia are further considered with phthisis.

Congestion or Hyperæmia of the Lungs.—**Pathology.**—The lung is dark-red, heavier, and firmer, and it crepitates less than the normal lung. It looks like the spleen when severely congested. The bronchi are filled with bloody, frothy serum. The capillaries are swollen, and compress the alveoli. There are numerous extravasations throughout the lung-tissue.

Œdema of the lungs usually accompanies congestion. The exuded serum infiltrates the interstitial connective-tissue and the alveoli; and if this be in sufficient quantity, the lung may pit on pressure. The serum exuded is red in colour, but if œdema result from general dropsy, it has the usual pale-straw colour. Œdema occurs most readily at the bases of the lungs. In chronic cases of congestion with œdema, the lung may also show the appearances of *brown induration.*

The causes of congestion and œdema are the inhalation of cold air or irritating vapours; the ingestion of cold drinks when the body is warm and perspiring; diseases, as pneumonia, collapse, &c., of the lung, producing congestion of other parts; and the passive congestion due to valvular disease of the heart or to simple dilatation. Bright's disease, and increased force of the heart's action from any cause, may also produce a congestion of the lungs.

The **symptoms** are slight fever, with a feeling of heat and oppression in the chest, more or less dyspnœa and cyanosis, with a quick but full pulse. When œdema is superadded, these symptoms are much exaggerated—especially the dyspnœa. Cough, with frothy expectoration streaked with blood, will also be present. The physical signs consist of the presence of sub-crepitant and fine râles which obscure the vesicular breath-sound; and should there be œdema, there will be the signs of consolidation at the bases, as dulness upon percussion, bronchial breathing, and bronchophony. An acute congestion may terminate fatally in a few hours or days, death being due to carbonic acid poisoning. There is then gradually increasing somnolence, less and less effort made to expectorate, and lastly, coma. Recovery may take place by resolution. The passive

forms of œdema, occurring in chronic valvular diseases of the heart and Bright's disease, develop more slowly. They may, however, prove *rapidly* fatal.

Croupous Pneumonia: Lobar Pneumonia.—**Pathology.**—The first stage—*congestion*—lasts for twenty-four or thirty-six hours, and the appearances are the same as described in congestion of the lungs. The second stage—*red hepatisation*—consists of the consolidation of a lobe or two, and sometimes even the whole lung. It is usually the lowest lobe, and most frequently that of the right side, which becomes affected. The diseased part is brick-red in colour, and has the appearance of liver-tissue. It is tough, non-crepitant, and elastic, and it does not collapse. It sinks in water. The affected parts present some of the appearances of œdema of the lungs, but they exude no serum on pressure. The non-solidified parts of the lung, however, may exude serum, as they are deeply congested and frequently œdematous. The air vesicles are filled with fibrinous lymph, and there is almost always an exudation of lymph upon the pleural surface adjacent to the inflamed lobe. Microscopic examination reveals the air vesicles filled with fibrin, leucocytes, blood corpuscles, and desquamated epithelial cells. Blood-clots are frequently formed in the main stems of the pulmonary artery, and they seem, sometimes, to be the cause of death.

The third stage—*grey hepatisation*—succeeds the red in a few days, the colour of the affected lung being now *grey-granite*. The lobe is not so tough, and if squeezed a purulent secretion is forced out. The fibrin becomes granular, and the leucocytes undergo fatty degeneration. The colour is due to this, and also to anæmia. The exudation becomes more serous, and absorption takes place in those cases which terminate in recovery. Rarely, gangrene of the lung and abscess may follow acute croupous pneumonia. More common sequelæ are pleurisy, pericarditis, endocarditis (sometimes ulcerative) and peritonitis, and sometimes even meningitis. Bristowe has described an associated colitis. Pneumonia occurs as a secondary condition, frequently in typhus and other fevers, erysipelas, diabetes, diphtheria, puerperal septicæmia, Bright's disease, and rheumatism, &c.

The causes of croupous pneumonia are not very clear. It appears to be frequently the result of exposure to cold and wet. A low state of health, or some wasting disease, such as diabetes, favours the occurrence of pneumonia; but it often attacks the robust. It is common in mid-winter and spring. Gout, rheumatism, and diabetes, and especially chronic alcoholism, are well known to be constitutional states associated with, and sometimes actually concerned in, the production of croupous pneumonia. Fränkels pneumococcus, or diplococcus, is almost invariably present, but as other micro-organisms are found, it is not yet clear that pneumonia is due to this microbe. The diplococcus does not fulfil all the conditions of a pathogenetic organism. The fact that it is sometimes *epidemic*—and must be regarded as a fever—is a proof of its microbic origin.

The **symptoms** usually commence with well-marked rigor, the

temperature rising in a few hours to 104° or 105° Fahr. Sometimes there is no rigor, and only a feeling of chilliness; and frequently pneumonia begins very insidiously. If the onset be severe and sudden, there is sometimes vomiting—or perhaps a convulsion—with a marked flush upon the cheeks, headache, and general *malaise*. The pulse is quick, full, and bounding. The respiration is markedly hurried, and the *alæ nasi* dilate with inspiration. The skin has a "pungent heat" at this stage when tested by the hand, although later there may be profuse perspiration. Pain is very frequently complained of in the side, and there is usually a short husky cough; later, the cough is accompanied by the expectoration of tenaceous rusty-coloured sputum, which is characteristic. It is like Gregory's mixture in a very small quantity of water or albuminous fluid, and "badly mixed." Sometimes it is less viscid, and like "prune-juice." The spit-jar may be reversed without the matter escaping. Sometimes there is no expectoration throughout the whole course of the disease. Microscopic examination of the sputum almost always reveals (after staining) the presence of diplococci, as oval or rounded organisms with thick transparent capsules, in pairs, single, and in groups. The tongue becomes furred, dry, and brown, the bowels are generally constipated, and the urine is high coloured, scanty, and acid, and it deposits urates in abundance. The inorganic salts in the urine, especially chlorides, are diminished in quantity, and sometimes albumen is present. Jaundice is frequent. Herpes affect the lips. The temperature is subject to irregular variations, falling suddenly, and as suddenly rising. Delirium may be present early, and especially in the intemperate, and then, very frequently, the upper lobe of the lung is the part affected (see Influenza).

The **physical** signs in a typical case are very striking. The most important is the detection of fine crepitations, which are very characteristic. They are heard upon deep inspiration, and at the end of the act. They may occur in œdema, collapse of the lung, and even in capillary bronchitis, however, and they are, therefore, not quite pathognomonic of croupous pneumonia. They are often heard also at the bases of the lungs in patients suffering from some debilitating disease, and who have been in the recumbent position for some time; but a few deep inspirations cause these crepitations to disappear. It may require careful examination for their discovery, and they are only heard at the *edge* of the consolidation; and sometimes, if the pneumonia be deep, they may not be heard at all, or only, after a few days, when the disease becomes more superficial. The vesicular murmur is obscured by these crepitations, or replaced by *bronchial* or characteristic *tubular* breathing—*i.e.*, if the bronchial tubes be not plugged by mucus. When so, the breath-sounds and crepitations are not heard unless coughing remove the obstruction. There is dulness upon percussion over the diseased lobe, and the vocal fremitus is increased. Bronchophony is present. When resolution takes place the fine crepitations appear again in a slightly coarser form—known as *redux crepitations*. The pleurisy which usually accompanies the pneumonia may give rise to a friction sound, and if there be effusion, the physical signs which characterise pneumonia may be very much modified.

Croupous pneumonia runs a definite course, and the fever usually terminates *by crisis* on the fifth to the tenth day. If prolonged beyond the fourteenth day some complication is present, usually. A rapid pulse, with weak heart-sounds are bad symptoms. The crisis is usually followed by profuse sweating, by sudden diarrhœa, or by the voiding of large quantities of urine.

The *prognosis* is always grave. Children recover more readily. The constitution and strength of the patient must always be considered in estimating the probable result. It is very grave when associated with alcoholism. All danger is not at an end although the *crisis* be past, as many cases die of sudden collapse, shortly, or two to three days thereafter. Sudden supervention of œdema, or failure of the heart, may cut off the patient at any time; or death may occur during the course of the disease by gradual exhaustion.

Secondary pneumonia is always more dangerous and is often fatal. A pneumonia of the upper lobe of the lung (apical pneumonia) is not more dangerous than the ordinary basic pneumonia, when the patient is not alcoholic.

Embolic Pneumonia. Hæmorrhagic Infarction.—Emboli may be simple or infective. The simple, when lodged in a terminal blood-vessel—so that no collateral circulation can nourish the parts supplied by the obstructed vessel—give rise to wedge-shaped infarctions, the base of the wedge being towards the periphery. The part affected is of a deep blood-red colour, sharply defined, and exuding blood upon pressure. When older they appear more granular, from fatty degeneration, and the tissue is more indurated. The parts adjacent are conjested and œdematous. Atrophy and cirrhotic contraction may ultimately obliterate the part, and a cure thus take place. A patch of the pleura is usually affected, as infarctions are superficial. Infective emboli are not wedge-shaped, and they give rise to suppurative inflammation and abscesses, and sometimes to gangrene. Infarctions are most common in the right lung. They are caused by clots breaking off in some part of the venous system, or right cavities of the heart, and these entering the circulation ultimately block the smaller arteries.

The **symptoms**, when the infarction is large enough, are sudden dyspnœa, followed by bloody expectoration, and the subsequent evidences of consolidation, as dulness upon percussion, bronchial breathing, and bronchophony. Infarctions most usually occur in heart disease; but they may arise from thrombosis of any part of the venous system. When small, they give rise to no symptoms, and even when large the co-existing disease may entirely mask the symptoms and signs. If the large branches of the pulmonary artery be blocked, the dyspnœa is very acute, and death takes place within a few minutes. In the infective form, there are repeated rigors, but not very high fever. The chills are marked in cases of pyæmia. There is sharp pain when the pleura is involved. The prognosis