We owe to Badham the introduction of the name bronchitis. Before him and Laennec the disease was confounded with the catarrhs, and more or less with phthisis and pneumonia; from the latter, however, in its broncho-pneumonic form it was distinguished by the name "peripneumonia notha." The name "peribronchitis" is reserved for an affection which chiefly implicates the outer coat; but the distinction is rather one of degree than of kind, the three coats being more or less involved in all cases. The clinical type of the disease is apt to vary with its distribution in the chest, with the degree of its severity, with its course and duration, and with its kind; and additional sub-varieties arise from its manifold associations with other diseases and from the multiplicity of its causes. The size and calibre of the bronchi concerned are also important factors. The patency of the smaller, and especially of the non-cartilaginous tubes largely depends on a free transmission of the mechanical forces of respiration; that is, on the even and symmetrical play of the surrounding pulmonary tissue. Tubes of minute diameter, whilst easily blocked by tenacious secretion, have little expulsive force for its removal; their inflammation is thus fraught with special consequences. The pathological results of bronchitis are not, however, limited to an interference with the air-conducting function, nor to changes in the mucous membrane; collateral changes may be set up. Bronchitis and bronchiectasis cannot, therefore, be satisfactorily studied in their various aspects without a brief preliminary reference to the anatomy and relations of the bronchial system.

The normal structure and relations of the bronchial tubes.— The distribution of the air-tubes in relation to the pulmonary substance is such that the lobules, which may be regarded as the pulmonary periphery, occupy not only the surface but also the centre of the organ. The perfect and even respiratory movements of the lung, associated with a minimum of pleural friction, are essentially dependent upon a uniform patency of the air-tubes. If the central lobules should fail to expand, compensating stress will fall upon the outer periphery—a result clearly seen in emphysema. The bronchi distributed to the more central parts of the lung being shorter and narrower than those proceeding to the
surface may perhaps be more easily obstructed; and in any portion of the lung, structural conditions may place some of the tubes at a relative disadvantage. The part which these easily obstructed bronchioles may play in the genesis of bronchiectasis will be explained under that heading. Their temporary obstruction in bronchitis would tend to increase any pre-existing hyperinflation of collateral lobules.

The relation of the bronchi to the pulmonary parenchyma is not merely one of direct continuity; close vascular connections establish a functional relationship between the respiratory surface of the air-cells and that of the intralobular bronchioles. With the pulmonary stroma the connection is also intimate. In each lobule the peribronchial tissue (as well as the periarterial) is continuous with the perilobular tissue, and therefore also with the interlobular connective tissue which binds together all the lobules. Lastly, with the visceral pleura the bronchi present a definite, though more distant relation. The deep layer of the visceral pleura is nothing more than the perilobular investment of the superficial lobules; and the interlobular septa throughout the lung may be regarded as a continuous prolongation of this subpleural layer. The structure of the bronchi is as follows:—The epithelial lining, consisting of three layers of cells, (a) columnar ciliated, (b) pyriform, and (c) flattened (Deboe’s membrane), rests, according to Professor Hamilton, on a tough, homogeneous, elastic membrane, the basement membrane, which is pierced only by the wide orifices of the mucous glands. An inner fibrous coat underlies this membrane, and is separated by the muscular coat from the outer fibrous coat in which are embedded the cartilages and the mucous glands.

The adventitia or outer fibrous coat is in intimate relation with the perilobular, and therefore with the intralobular tissue of each lobule. In the case of the larger bronchi the connection is also direct with the interlobular stroma. The adventitia is thus the medium of extensive communications, chiefly lymphatic, between the air-tubes and the rest of the lung; and in disease it shares in all those processes to which the term “interstitial” is applied.

The muscular coat, in addition to those functions which are obvious, may also discharge other physiological duties, a knowledge of which might throw light on pulmonary pathology. Hitherto we have heard more of the perversion of the function of the bronchial muscles than of their natural uses. It is generally admitted that they are liable to tonic spasm, and that this spasm and the resulting partial closure of the smaller air-tubes enter largely into the causation of asthma, and in varying degrees complicate the respiratory difficulties special to bronchitis.

The vascular system of the bronchi consists of the posterior or main bronchial arteries originating from the descending aorta, the anterior bronchial arteries supplied by the internal mammaries, and the small branches contributed by the cesophageal, mediastinal, and pericardial arteries; these vessels accompany the bronchi, supplying not them alone, but the entire pulmonary stroma with nutrient blood, the pulmonary
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artery being exclusively subservient to respiration. The capillaries of
both sets of arteries anastomose freely in the alveolar district, and
probably also in the mucous membrane of the air-tubes. According to
Zuckerkandl, "only the larger bronchi are irrigated by the bronchial
arteries, the terminal tubes being vascularised by the pulmonary artery,
and the intermediate bronchi by both."

A similar intercommunication exists between the bronchial veins of
the smaller air-tubes (and even, according to Zuckerkandl, of the larger
ones) and the pulmonary veins. The bronchial veins also anastomose
in the posterior mediastinum with the venous plexus formed by branches
from the esophageal and from the diaphragmatic veins (Hamilton).

The bronchial lymphatics take their origin in the inner fibrous layer,
which is in lymphatic communication with the tunica muscularis and,
through the thickness of the latter, with the abundant plexus of the outer
fibrous layer, where probably they are chiefly discharged into the peri-
arterial channels. Both fibrous layers contain lymphatics in abundance;
but since, according to Hamilton, these do not traverse the basement
membrane, no absorption would take place from the epithelial lining,
and the ununction of the latter would be effected directly into the
bronchial lumen.

Before and at its entrance into the lobule the lobular bronchiole is
in lymphatic connection with the peribroncholar and with the interlobular
network.

Within the lobule the lymphoid tissue described by Arnold (which
also occurs under the pleura) is distributed around the alveolar passage
and in the bronchial wall, as well as along the blood-vessels. The peri-
bronchial masses are said to occur on the side of the bronchus opposite
to that occupied by the accompanying pulmonary artery.

The activity of the intra-alveolar lymphatics is shown by the rapid
absorption of the products of pneumonia. The interepithelial spaces and
their connective tissue corpuscles communicate with interalveolar plas-
matic spaces or lymph capillaries, which converge either into the super-
ficial or into the deep lymphatic network of the lobule. The larger
vessels which arise from both these networks accompany the pulmonary
arteries and veins to the hilus; whilst another set reaches the latter from
the superficial subpleural lymphatic network. According to Hamilton,
the subpleural lymphatics have but little intercommunication with the
lobular.

In the carbon-injected miner's lung (which usually is not fibrously)
the entire lymphatic scheme is displayed; and this may be studied in
Hamilton's beautiful illustrations. According to Hamilton, the root
particles lie in the peribronchular and interlobular tissue, around the
pulmonary artery and bronchi, in the lymphadenoid bodies of the lung
and of the bronchial glands, in the alveolar walls (sparsely), in their
epithelial interspaces, and in their desquamated epithelial cells.

The absence of pigment from the visceral pleura might have been
expected; its absence from the bronchial mucous membrane (which
retains in the miner’s lung the pink hue of bronchitis), is explained by Hamilton and others in connection with the impermeability of its epithelium and basement membrane, the injection of the lymphatics taking place through the alveoli only, which but few of the inhaled particles would reach. The isolating property of the basement membrane thus demonstrated has much significance from a pathological as well as from a physiological standpoint.

**Classifications of bronchitis.**—The anatomical nomenclature.—Although a separation of the air-tubes, according to their size, into (a) the large bronchi, (b) the middle-sized bronchi, (c) the small bronchi and sublobular bronchioles, and (d) the intralobular or capillary bronchioles, is in great part conventional, still this supplies a convenient anatomical classification for the varieties of simple bronchitis, among which we may describe the following:

i. Trachea-bronchitis, or bronchitis of the large tubes. = An inflammation of the trachea, and larger cartilaginous tubes.

ii. Simple or mild acute bronchitis, or bronchitis of the middle-sized tubes. = An inflammation of the tubes of medium size, and of the smaller cartilaginous tubes.

iii. Severe acute bronchitis of the adult, or acute suffocative bronchitis, or bronchitis of the smaller tubes. = An inflammation of the smallest cartilaginous, and of the non-cartilaginous tubes down to the lobular bronchioles.

iv. Capillary bronchitis of infancy and of old age, or “peripneumonia notha.” = An inflammation of the sublobular and lobular bronchioles, extending into the intralobular bronchioles and air-passages.

These groups are not rigidly isolated but frequently combine. Whilst trachea-bronchitis and bronchitis of the middle-sized tubes most often occur independently of the later members of the series, and often indeed independently of each other, the most important of the mixed forms are those involving some of the small tubes in addition to the larger ones.

There is also a clinical nomenclature based upon the severity and duration of the attack. Although usually acute in their onset, trachea-bronchitis and bronchitis of the middle-sized tubes are, in themselves, seldom severe or dangerous. Bronchitis of the fine tubes is always a severe as well as an acute affection, and its termination in death or in recovery is usually not long delayed. This special feature of acuteness and of gravity explains, and to a certain extent justifies the use of the term “acute bronchitis,” which has been applied to it by rule of custom, but which is neither exclusive nor explicit. “Chronic bronchitis” is also a general name which has become specialised, without, however, involving much ambiguity: for, neither capillary bronchitis nor bronchitis of the small tubes being susceptible of a chronic development, this name can only apply to group ii., which is exceedingly prone to chronicity, in
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contrast with trachea-bronchitis which is apt to be recurrent rather than lasting.

The occasional severity of the acute stage of a primary bronchitis of the middle-sized tubes shows the disadvantage of monopolising the name "Acute Bronchitis" as a name for the affection of the smaller tubes; and secondary varieties will be described, which may also run through acute and subacute stages.

Lastly, a pathological nomenclature recognises a bronchitis pure and simple—not hitherto traceable, as in the diphtheric, the tuberculous, and some other forms, to parasitic influences—which presents the following well-marked varieties:

i. Catarrhal bronchitis: (a) simple mucous catarrh; (b) chronic or mucopurulent catarrh.
ii. Plastic bronchitis.
iii. Putrid bronchitis.

The immense majority of cases belong to the catarrhal group. The putrid purulent form is seldom met with. The plastic variety is so rare as to be little more than a clinical curiosity.

I. SIMPLÉ BRONCHITIS

Causation.—A. Remote causes.—(i.) No age is exempt; but during early adult life the disease is much less prevalent, in spite of greater exposure. Infants and the aged are particularly liable to it, and the periods of dentition favour its onset. (ii.) Except during the working periods of life, when men are more exposed, sex makes little difference. (iii.) Many occupations involve direct exposure to the extremes of temperature; others are indirect causes, through relaxing influences or confined air. Some trades lead to inhalation of fumes or particles which mechanically set up bronchitis, such as particles of steel, granite, chalk, charcoal or cotton. (iv.) Luxurious habits both in diet and in clothing, and the overheating of rooms, induce a liability which is especially regrettable in childhood, when the individual tendencies are capable of some measure of control. (v.) Heredity and temperament constitute distinct factors; a delicate bronchial membrane may be inherited as a delicate skin or any other outward peculiarity may be. Again, acquired constitutional weakness from any cause (poverty, overwork, prolonged illness, or intemperance) has an unfavourable effect. (vi.) Certain blood diseases favour the production of bronchitis in a special degree; such are Bright's disease, gout, diabetes, enteric fever, and, particularly, measles and rickets. (vii.) Heart disease is a potent factor, more especially those forms of it which lead to pulmonary and bronchial congestion. (viii.) Pre-existing chest affections — thoracic, pleural, and pulmonary —also dispose to bronchitis; but none in so well marked a degree as emphysema. (ix.) Relative impurity of air renders the inhabitants of large towns more liable to bronchitis than country folk; but the deprivation of an open-air life, and long sedentary hours in crowded dwellings,
are probably more potent influences; and those whose lives are chiefly spent out of doors, even if they perpetually breathe town air, probably do not suffer in the same degree. Dr. Frederick Roberts, in Reynolds' *System of Medicine*, states that in Cheshire and Lancashire, during the year 1868, the ratio of mortality from bronchitis to the number of inhabitants was 1 in 379.5; whilst in London it was only 1 in 442.3. It is suggested that this striking mortality is due to the sedentary lives led by so many mill-hands, to the high temperature of the factories, and perhaps to the effluvia which pervade the manufacturing districts.

(x.) The climate of this country, by its humidity and variability, favours the prevalence of bronchitis. Variations occur in the mortality year by year as the weather oscillates more or less. In 1867 it reached 1902 to every million living; but the mean rate for fifteen years, from 1850 to 1864, was 1344.4.

Although sudden changes to cold winds, and particularly to the north-easterly winds, are marked by a large increase of bronchitis, it does not appear that mere bleakness or habitual exposure to strong winds, particularly to the north and to the east winds, so largely tend to set up bronchitis as might be supposed. This is shown, in the figures for 1868, in the relatively favourable return from the eastern counties, which head the list with a mortality of 1 in 987.5 inhabitants, and of the north midland counties (1 in 876.2), as against the south-western counties (1 in 844.8), and all the other districts which have yet higher rates of mortality. We notice, however, that Monmouthshire and Wales (1 in 955.4) closely approach the position taken by the eastern counties.

The difference between the seasons is that which might be expected. Bronchitis is greatly more prevalent during the winter months than in summer, and the liability to it extends into early spring. Thus, whereas the greatest prevalence of pneumonia occurs during March and April, that of bronchitis belongs to the colder months.

(xi.) Aerial impurities may be solid, fluid, or gaseous. Strongly irritating particles or vapours may act as direct exciters of bronchitis, as for instance the vapour of ammonia, of iodine, of bromine; finely-powdered ipecacuanha, pepper, or tobacco; and, in the case of those specially liable, the pollen of certain varieties of flowering grass.

B. *Immediate causes.*—The most usual proximate cause is a chill. The patient is said to have "caught cold." The precise meaning of this phrase is obscure. So long as the adaptive mechanisms are in full efficiency, mere extremes of temperature do not constitute a danger to the mucous membrane, and a strong man may pass unscathed from one extreme to the other. Even infants and old people may breathe cold air with impunity, especially if it be dry, so long as they are adequately clad and in perfect health. The liability to "catch cold" is sometimes an individual peculiarity; more often it is acquired, but it is usually intensified by sundry debilitating causes and by faulty hygiene.

Very little is known concerning any functions of the aerial mucous membrane analogous to the regulating mechanisms of the skin for tem-
temperature. Their existence is rendered probable not only by the noticeable difference in individual susceptibility, but by the interdependence of the cutaneous and of the bronchial system in the process of "chill." There are two kinds of chill—that directly applied to the air passages by cold and damp air, the body being at the time warm and well covered; and that which is due mainly to exposure of the cutaneous surface. In both cases the sensation experienced at the time is apt to be referred partly to the skin, the patient "feeling chilly all over," and partly to the air passages; often to the pharynx or down the trachea. A nervous link is indicated by these paired sensations. Rossbach's experiments show that application of cold to the skin is followed in one or two minutes by a reflex contraction of the tracheal vessels, and a little later by venous congestion and an increased flow of mucus. Any fault in the regulating mechanism, and particularly in its nervous factor, would leave the mucous membrane unprotected against the physical results of continued exposure to extremes; or incapable of that rapid adaptation which is our safeguard against sudden transition from one extreme to another.

Smoke is a powerful irritant, whether by its scorching effect when inhaled hot, by the mechanical action of the suspended carbon or ash, or by the irritating nature of the volatile products of combustion.

Steam, when inhaled from the spout of a kettle by the children of the poor, usually checks inspiration, and its irritating effects are limited to the upper air passages; but when there is no escape from the inhalation the damage to the air tubes may be extensive.

Suspended cold moisture, as in ordinary mist, seems capable of irritating very sensitive bronchi, but it is difficult to eliminate the chilling effect of the mist on the body surface; and it is noteworthy that when an equivalent amount of moisture is inhaled in crystalline form, as in a severe frost, its mere cooling effect is not as a rule resented. The nasal passages, of course, exercise some warming influence.

Town fogs are directly responsible for a great deal of bronchitis. Consisting as they do of a mixture of suspended moisture with varying proportions of the products of combustion, fogs differ greatly in their irritating qualities. The fog is acid, and each droplet of water is coated not only with a minute proportion of some tar-like body, but with an equally minute quantity of sulphuric acid; a combination most likely to excite inflammation of the respiratory passages in delicate persons.

Irritant gases have been classified as non-respirable and respirable. To the first group belong chlorine, ammonia, sulphurous anhydride, and the vapours of iodine and bromine. The danger of their continuous inhalation is obviated by the intensity of the irritation causing spasmodic arrest of respiration. A single whiff of ammonia is commonly followed by a transient watery flow from the mucous membrane.

Among the mildly irritating vapours ether, so largely used for surgical purposes, deserves special mention. In the case of small children, in the aged, and in those with limited respiratory surface, its use is to be avoided; even though a proportion of the instances of so-called "ether
bronchitis” may be regarded as due to exposure of the surface during the operation, or to the cold produced by the evaporation of the ether, rather than to any direct irritation of the membrane.

As regards temperature, we know that standing in a cold draught, staying out at sunset with insufficient wraps, keeping on wet clothing after severe fatigue, or sitting long with wet or cold feet are so many modes of causation of bronchitis by cutaneous chill. When the impression of chill is confined to the mucous membrane itself, the mischief is usually due less to the intensity of the cold than to previous exposure of the membrane to hot and impure air.

Intolerance of any but the milder kinds of atmosphere is most commonly the artificial result of injudicious physical education. It also belongs to states of debility and to the extremes of age.

The popular belief in the contagious character of common catarrh has received from time to time considerable support from the prevalence of epidemic catarrh and influenza. Although the latter disease does not exclusively attack the respiratory passages, still the almost universal coincidence with it of more or less inflammation of the air-passages must give it a place among the causes of bronchitis. In many instances the irritation, whatever be its mechanism, is severe, the cough being of a harassing type which resembles that due to mechanical irritants, and not infrequently in-veterate. Ordinary bronchitis has never been attributed to a specific contagium, so far as I am aware.

Lastly, the possibility of a penetration, through any mucous abrasions, of the micro-organisms of disease, and particularly of the staphylococcus pyogenes, of the streptococcus pyogenes, or of the pneumococcus—not to mention numerous less harmful microbes found in normal air-passages (8 different streptococci, 21 bacilli, 10 micrococci, and several sarcinae, according to Panzieri)—should not be forgotten. Bronchitis might, it has been suggested, be brought about by a combination of influences, the micro-organisms finding access through an epithelial layer previously loosened or damaged by undue exposure.

Pathological anatomy.—Professor Hamilton’s researches,¹ from which the present account is largely derived, furnish us with the most recent data.

(i.) Acute catarrhal bronchitis begins with a relaxation and distension of the blood-vessels of the inner fibrous coat; a few hours after this the basement membrane becomes oedematous, much swollen, and folded: twenty to thirty hours afterwards it loses its ciliated cells in patches, and some of these may be inhaled into the smaller bronchial tubes. Immature cells are supplied in great number by the proliferation of Debove’s cells, and they constitute the cellular element of the bronchitic secretion. Absolute denudation of the basement membrane may occur but only temporarily, and over limited areas. Desquamation and active secretion of mucus take place at the same time in the mucous glands. The entire thickness of the bronchial wall is swollen, congested, and infiltrated with

¹ Cf. loc. cit.
leucocytes. Reparative changes are initiated by a diminution in the congestion, and in the dilatation of the vessels; and the cells gradually resume their normal development and functions. Throughout the attack the normal gray colour of the mucosa is replaced by a dull red.

(ii.) Chronic bronchitis.—(a) The common form, the result of a series of acute attacks, is usually associated with much permanent emphysema with intervening congested areas. The lower part of the trachea and the bronchial surface in general are congested and purple, and yellowish muco-pus can be squeezed out of the middle-sized and small air-tubes.

The characteristic smooth and shiny aspect of the mucosa is due to the basement membrane being laid bare, only a few ill-shapen cells adhering to it; it is not always much swollen. Some dilatations may occur in the smaller tubes; the larger ones on the contrary may be slightly narrowed by the great thickening of their coats. The coats are densely infiltrated with cells, among which are seen many dilated capillaries—some of which may project into the thickness of the basement membrane—many thickened arterioles, and over-distended lymphatics; these are especially abundant close to the cartilages which are vacuolated, and in various stages of absorption. The muscular coat may be hypertrophied, or on the contrary greatly atrophied; or even absent. The mucous glands also may be destroyed by cell infiltration, or on the other hand much enlarged, with active mucous transformation of the glandular and duct cells. Atheroma is frequently observed in the middle-sized pulmonary arteries in the subjects of chronic bronchitis.

(b) A separate form of chronic bronchitis is characterised by a peribronchitis fibrosa chronica (Virchow), and in some cases the fibrosis extends along the pulmonary lymphatics to the entire interlobular stroma. Instead of the common atrophic, rarefying emphysema, the lung tissue then presents diffuse condensing fibrotic changes.

Physical signs.—The physical signs common to all forms of bronchitis are so familiar that little more than a cursory review of them is necessary.

In shape and in size the chest tends to assume the inspiratory type, without deformity, but with marked elevation of the clavicles and shoulders, deepening the suprasternal and supraclavicular fossa. In consequence of this and of the hyperinflation of the lungs, the diaphragm, liver, and heart are more or less depressed.

The thorax moves at an increased rate, but to a diminished extent. In severe bronchitis the inspiratory efforts fail to expand the chest, except in its upper part; and there may be inspiratory recession of the lower interspaces, and in children of the lower ribs and sternum. The abdominal muscles are thrown into strong and prolonged contraction during expiration.

Bronchial fremitus is felt on palpation during the entire respiratory act, or may be confined to inspiration or to expiration. Vocal and tussive fremitus are not materially altered.

Percussion in most cases elicits an increased resonance, which may, however, be masked by the strong contraction of the inspiratory muscles,
to which is also due the peculiar tenderness of the chest. In small children the occurrence of broncho-pneumonia or of collapse may detract from the resonance, or may even cause an imperfect dulness.

Auscultation.—Except at the upper part of the thorax, where they are often exaggerated, the respiratory sounds are much diminished or may be inaudible. Their coarse and harsh character is indirectly due to the feebleness of the alveolar murmur, which no longer veils the sounds produced in the bronchioles; a condition also observed in emphysema.

The adventitious sounds arising in the chest in the course of a simple bronchitis include the two great classes of the dry and of the moist sounds. To the first belong the large or sonorous, the small or sibilant, and the intermediate or subsibilant rhonchi. The Æolian harmony often audible seems to be specially frequent where some emphysema is kept up under the joint influence of bronchitis and of muscular spasm. Another musical sound is the rhythmic sibilus which may be set up in the neighbourhood of the heart by each cardiac systole. Considerable extension and loudness of the bronchitic sounds, although indicating the implication of rather small tubes, and compatible with severe symptoms, are not usually of anxious import in themselves, since they indicate that air passes, though not freely, through a large number of tubes. Clicks are sounds of sudden and snapping character, lacking musical quality and difficult to interpret; being occasionally suggestive of a parched, at other times of a moist condition of the tubes. Hence they are described in different instances as moist clicks or as dry clicks, thus occupying an intermediate position between the rhonchi and the mucous râles. The moist sounds of bronchitis have a gurgling or bubbling quality. Nomenclature and description are much simplified by calling them mucous râles (large, medium-sized, or small), in contrast with the hard or metallic rattles, crackles, or crepitations which may occur in the same chest if broncho-pneumonia, or lobar pneumonia, or phthisis should complicate the bronchial catarrh. The fine crepitations which may become audible over limited patches in capillary bronchitis, in association with abundant mucous râles elsewhere, illustrate this distinction.

Cardiac signs.—In fully established bronchitis a more or less distinct epigastric heat is felt, the heart being not only depressed but enlarged also. The enlargement is mainly due to an over-filling of the right auricle and ventricle, evidenced by the distended jugulars; whilst the left ventricle presents little change. At the same time the absolute dulness of the heart is lessened in its size, and the heart sounds in their loudness by the inflation and encroachment of the sternal-fringes of the lung. A relative increase in the loudness of the second pulmonary sound also belongs to uncomplicated bronchitis.
A SIMPLE BRONCHITIS LIMITED TO THE LARGE AND MIDDLE-SIZED TUBES

Symptoms and course.—At the onset the attack may make itself felt as a severe cold in the chest, with deep-seated rawness, soreness, and parching; or it may begin in the larynx, or in the pharyngeal or nasal region, implicating also the ocular conjunctiva, the frontal sinuses, and the upper nasal cavities. Again, there may be more or less gastric and hepatic disturbance. Individual peculiarity and local susceptibility may help in each case to determine the site of invasion. That these are not, however, the only factors is shown by the regularity with which special forms, such as the bronchitis of measles, begin in special situations.

In acute cases much continued or intermittent chilliness, and in children slight delirium, or even convulsions (especially during the first detention), may open the scene.

With every variety of onset there is a uniformity in the general symptoms. The pulse and respiration are moderately quickened, and the temperature is raised two or three degrees. The patient complains of respiratory discomfort, malaise, aching pains, headache, mental and physical languor, drowsiness during waking hours, and restless sleep—the results of the sudden check to the secreting and exhaling functions of a large section of the respiratory membrane. Almost invariably the alimentary mucous membrane is involved: the appetite fails, the tongue is heavily coated, the liver inactive, and the bowels torpid.

The symptoms of the disease when in progress may be classed as general, local, and respiratory. The local pain is seldom acute. The sensation is almost always retrosternal; it is variously described as "sore," "raw," or "burning," and the cough as "tearing." Tenderness on pressure is also felt at the sternum, but greater tenderness arises later from the constant strain of cough, and is then felt over the entire chest, but particularly over the pectoral muscles and at the base of the thorax.

The general symptoms are those of slight feverishness. The dry heat of the skin which follows the stage of invasion in most cases soon gives way to moisture. The temperature oscillates in the usual manner between a morning minimum and a maximum at night, but does not often rise very high. The pulse is moderately quickened and full; at first it is excited in action and almost bounding, but subsequently, with the advent of diaphoresis, large, soft, and undulating. The tongue is furred but moist, and the appetite bad; vomiting is unusual, constipation almost the rule. The urine is of the febrile type, with rather high specific gravity; in healthy subjects it is free from albumin, but loaded with lithates, pigment, and urea; sometimes it contains less than the usual amount of sodium chloride.

Respiratory symptoms.—The patient's complaint is of tightness and oppression at the chest, rather than of dyspnea, though this would be brought out on any exertion. Even in the position of rest the respira-
ions are markedly quickened and proportionately more so than the pulse; they are shallow, and ultimately become laboured. Cough sets in early, especially in the laryngeal, tracheal, and bronchial forms of onset; rather later when the pharynx is affected first, and sometimes not for a day or two when the attack begins with coryza, in which cases sneezing is more common. At first, the cough is dry and irritating, and usually associated with a tickling sensation in the larynx or trachea; when these structures are involved it is much altered in tone. It is easily set up by slight irritation, and is difficult to check. At a later date the paroxysmal character is no longer due to simple irritation of the nerve-endings over a dry and parched surface, or to a congested uvula and epiglottis, but to the difficulty in expelling the viscid and frothy secretion.

The expectoration, in all cases of bronchitis, furnishes us with indications as to the stage and progress of the affection. From the healthy state of simple moisture, free from any perceptible excess of fluid or of mucus, the inflamed membrane, after a preliminary phase of checked secretion and of dryness, quickly passes through a stage of excessive hydration, during which the mucin of the cells is matured; though but little of it finds its way into the saline watery flux. After a very few hours mucus is poured out more freely, and renders the fluid ropy; but it is still as transparent as glass, and free from bubbles other than those produced in the mouth or in the larger tubes. The next stage is that of purely mucous catarrh. The secretion stiffens, and, in the smaller tubes, soon offers considerable resistance to the respiratory current. This is clearly seen in the amount of air-bubbles held in the mucus, which, although in itself hyaline and colourless, forms with them a white opaque froth. This is the “crude stage” described in ancient books. In cases of rapid resolution the mucus may soon undergo a secondary hydration, losing its bubbles, and coming up with less effort and in rapidly lessening quantities.

More commonly in the ordinary case of bronchitis the sputum passes through some degree of “coction” (to use again an obsolete term), losing together with its extreme viscidity and frothiness the hyaline colourless quality, and becoming either streaked or uniformly tinged with light yellow pus, whilst continuing to form as before a confluent mass in the receiver. In more protracted cases the admixture of pus gradually increases, and imparts a greater opacity and a greenish tinge to the sputum, which becomes less hydrated, quite free from bubbles, and ultimately nummular. This is a sign that the catarrhal process is lingering in the larger tubes. There is much analogy and yet a distinction between this expectoration and the more purulent and fluid discharge which from its quantity and inveterate character has received the name of purulent bronchorrhoea, and in which the individual spuTA fuse into a mawkish yellowish semi-fluid mass. In the later stage of bronchitis the spuTA remain distinct.

Hæmoptysis, in simple uncomplicated bronchitis, is of exceptional
occurrence; but a few streaks of blood may be seen in the earlier and
drier stage. They are probably due to the sudden detachment of super-
ficial layers of the membrane under the effort of cough.

Prognosis.—As to the duration of the attack prognosis is of necessity
somewhat uncertain, and is partly governed by atmospheric conditions.
In healthy children, youths, and adults, especially if not previously
affected, complete recovery under appropriate treatment may be looked
for within one or two weeks, according to the severity and extent of the
inflammation. Any antecedent bronchial trouble would modify and
unsettle the estimate. In infants and the aged it is wise not to fix any
date.

As to danger to life, it is only at the two extremes of age, and in
albuminuria, or diabetes, or heart disease, or cachexia, that doubt is
likely to arise. However much they may ultimately tend to shorten
life, even repeated attacks of this mild form of bronchitis are never
directly fatal in subjects otherwise sound. If the respiratory muscles be
feeble, as in infancy, old age, or obesity, there is risk of broncho-pneumonia
a retentis, the termination of which cannot be foretold; the other risk,
peculiar to the same group of patients, arises from weakness of the heart,
and especially of the right heart, which may undergo dilatation and
eventually paralysis; or the bronchitis, especially in the aged, may
become chronic, and prove at length a fatal drain on an exhausted
vitality.

B. ACUTE SUFFOCATIVE BRONCHITIS OF ADULTS, OR BRONCHITIS
OF THE SMALL TUBES

Symptoms.—The following sketch of the clinical history of suffocative
bronchitis may justify our attempt to deal with it as a separate study.
Walshe, who obviously appreciated the essential differences between it
and capillary bronchitis, nevertheless included their description under
one heading,—“General and capillary bronchitis—olim peripneumonia
notha”; and subsequent authors have followed him.

A first distinctive feature of simple asphyxial bronchitis is the
exceedingly rapid and general implication of the small tubes throughout
the lung. Walshe says: “I have known life destroyed in forty-six
hours, reckoning from the first moment of seizure, in a middle-aged adult,
who, in previous years, had had more than one seizure.” In the adult
(and it is noteworthy that young adults are rarely attacked) orthopnea is
the rule, and, as observed by Walshe, “Maintenance of the head on a low
level from the first, in a case otherwise grave, is of evil augury.” It is
hard to say to what extent superadded muscular spasm of the bronchioles
may increase the constriction due to inflammatory swelling.

More air is at first drawn into the lung by the strenuous breathing
than can be expelled by expiration. Subsequently, in spite of the
powerful contractions of the muscles of extraordinary respiration, the chest
moves comparatively little, and ultimately the character of the respiration
tends more and more to become expiratory and abdominal. The lower intercostal spaces are drawn in with each inspiration, but the ribs do not usually recede. The whole chest is enlarged, and the lungs over-distended by the powerful muscular forces applied to sufficiently rigid bones and cartilages. An excess of air is, as it were, locked in by the obstruction of the bronchioles: henceforth little passes through them into the lobules, whether in the shape of air or of secretion; and the direction taken by the latter is outwards, not inwards as in capillary bronchitis. The oxygen of the imprisoned air becomes exhausted, and the turgid veins and the asphyxial complexion of the patient warn us of the degree of the obstruction to the pulmonary circulation, and of the congestion of the overloaded right heart.

Expectoration is not suppressed as often occurs in capillary bronchitis. A fine white foam resembling "whipped egg" gives in the minute size of its bubbles the gauge of the tubes affected. An analogous "whipped egg" sputum (not, however, quite so fine) is sometimes observed in the sudden pulmonary congestion apt to complicate an anginal attack. In the absence of angina this sputum is diagnostic of suffocative bronchitis. A change to a coarser froth with the admixture of watery, hyaline, and subsequently of purulent mucus, gradually occurs in the later stages of the more favourable cases.

Asphyxiating bronchitis of the adult is not complicated with any parenchymatous inflammation of the lung. Pneumonia is perhaps mechanically obviated by the intra-alveolar pressure of gas, and by the stretching of the alveolar vessels. At any rate this immunity is attested by the pulmonary appearances after death and by the observations of every clinical observer. Walsh says: "True pneumonia, lobular or diffused, is of purely exceptional occurrence; the parenchyma is often even unusually pale"; and again, "but, without meaning to deny the possibility of the fact, I must observe I have never yet seen local collapse of lobules on an extensive enough scale in simple adult bronchitis (agonised as it is by the distending influence of the disease on the alveoli) to justify me in looking upon it as a sufficing cause of deficiency of tone"; once more he says, "Bleeding is useless for the prevention of pneumonia, seeing that, in the adult, idiopathic inflammation of the tubes does not pass on to the parenchyma."

The later course of the disease need not be detailed at full length. The symptoms are those of a progressive asphyxia—a prolonged struggle for breath, the duration of which is measured by the patient's cardiac energy. In Walsh's unsurpassed description:

"As long as his strength permits, the patient sits erect or bends forward; but the body gradually yields; and it is not uncommon to find patients, while still perfectly conscious, lying sideways or forwards with the head lower than the shoulders. In rare cases, a posture of this kind is adopted from the very onset.

"The sputa gradually diminish in quantity from failure of power to expectorate; the skin, generally livid or cyanotic in tint, falls in tempera-
tured, becomes covered with cold, clammy perspiration—sometimes copious, rarely attended with formation of sudamina; the expired air grows cool, the feet and hands swell, in protracted cases the anasarca rising to the trunk, unaided by coexistent disease of the heart, or of any other organ; expectoration of dropsy; fitful dozes lapse into a state of somnolence, constant, except from momentary interruptions by the cough; muttering delirium, associated in some instances with slight convulsions, precedes a comatose state which is the immediate forerunner of death.

The pulse gains in frequency as it loses in power, ranging from 120 to 150 or more. The respirations, varying from 36 to 50, may ultimately recede from the maximum rate they had attained.

The temperature is moderately elevated. Dyspnoea, oppression, retrosternal pain, restlessness, and cough are the chief symptoms complained of.

The urine is scanty and concentrated. There is occasionally a transient albuminuria, but, in spite of the great diminution in the oxygen supply, there is no sugar, and usually no excess of urates.

The physical signs are those of emphysema, as regards increased bulk of the chest and of the lung, depression of the diaphragm and of the heart, and pulmonary hyperresonance, coupled with bronchitic riles in the larger tubes owing to the ascent within them of the frothy secretion.

The prognosis is anxious even in the best subjects. The worst cases are those of pre-existing emphysema with incipient or advanced dilatation of the right heart; these subjects seldom long survive the onset of a genuine bronchitis of the small tubes. Cardiac defects, or inherent debility, whether from exhaustion or atheroma, chronic albuminuria and the various cachexies greatly reduce the chances of recovery.

The duration of a fatal attack may be reckoned in hours, or may "drag on to the tenth or twelfth day" (Walsh). The same authority has recorded unexpected recovery after long periods of an apparently hopeless condition, with cold clammy sweat and almost complete loss of conjunctival reflex. Such cases are rare; they seem to suggest that spasm of the bronchioles had contributed to the bronchial stoppage.

Morbid anatomy.—The post-mortem appearances are almost invariably those of an over-distended, non-collapsing lung, the pale pink colour of which contrasts strangely with the deep cyanosis of the body surface, and is readily explained by the influence of the residual oxygen of the distended air-cells on the reduced quantity of blood which their overstretched capillaries accommodate. The small bronchi present, on the contrary, a swollen and deep red surface of section. Their contents vary with the duration of the cases: in early deaths they consist chiefly of mucus; they are semi-purulent in those who have survived for several days. Exceptionally here and there a pulmonary lobule may be found collapsed, but pneumonic consolidation is absent. In all cases the heart presents the asphyxial condition, and the viscera are engorged.
G. CAPILLARY BRONCHITIS OF INFANCY AND OLD AGE
(Peripneumonia Notha)

'This name specially belongs to the inflammation which extends from the small bronchial tubes into the lobules and into the alveoli. It is in great part a pneumonia, and was originally observed and referred to as such long before the existence of bronchitis as a disease was thought of. Indeed the pneumatic changes, where they coexist with bronchitis, are obvious enough; whilst of all diseases, equally intense and clinically definite, none leaves after death traces slighter in themselves, or more easily overlooked by the inexperienced observer, than those of simple acute bronchitis.

It is singular that this liability to the pneumatic complications should be shared exclusively by the earliest and by the latest stages of life, in spite of most opposite anatomical conditions; the lung being atrophied and rarefied in old age, with relatively large air-spaces and tubes, whilst in the infant it is only partly developed, and is fully packed with relatively narrow air-tubes and air-cells yet imperfectly expanded: the thorax in the one case is roomy and almost rigid, in the other relatively smaller than at a later date, and exceedingly yielding; the morbid processes reflecting in the first the sluggishness of age, in the second the activity of budding life. It can hardly be doubted that the intimate changes in the two conditions must present essential differences, and that the similarity between them must reside mainly in the general lines of march of the disease, and in the direction taken by its extension. At any rate in the capillary bronchitis of infants we perceive a factor entirely foreign to that of old age, the tendency to rapid proliferation of the tissue elements under irritation, and to the choking of space by direct cell overgrowth. One peculiarity is common to both extremes of life—feebleness of the mechanisms of respiration, which allows the obstruction to tell in a degree not witnessed in the adult. In other respects the processes differ.

Infantile bronchitis attacking the small tubes almost inevitably disables some of them at an early date, owing to the very unequal local resistances of the chest walls, and to the influence of decubitus. At given spots the thorax fails to draw out the subjacent lung, and is dragged in instead. The subjacent lobules quickly become airless and collapsed, and are henceforth sealed against the entrance of gases, fluids, and solids alike: they are incapable of becoming pneumatic. Collateral emphysema results from the increased respiratory stress thrown on other parts, and, thanks to their early over-distension, these lobules also may remain free from pneumonia. It is in the remaining portions of the lung, imperfectly expanded and traversed by enfeebled respiratory currents, that the changes occur. The secretion, failing to be expelled by its stagnation, sets up intralobular irritation, and a tissue-reaction which is mainly proliferative.
Two forms are noticed—the strictly broncho-pneumonic with prevailing proliferation of epithelia, infiltration of the bronchial and alveolar walls, and consolidation of the alveoli by epithelial cells: and the purulent form in which loose, semi-fluid bronchial secretion accumulates in the smaller divisions, dilating many of them and setting up a form of acute generalised bronchiectasis; whilst a varying amount of pneumonic change is also present.

In old age rigidity of the thorax, degenerative changes in the lung such as widening of the alveoli and of the air-tubes, atheroma of the pulmonary artery and relaxation of the pulmonary veins, loss of inspiratory energy and considerable loss of general expiratory power, and especially of the expulsive power of individual districts the expansibility of which may have been reduced by pleural adhesions or by the reticular fibrosis left behind by former attacks, are some of the factors determining the variety of capillary bronchitis. The minute diameters of the tubes and the yielding of the thoracic parietes, to which are due the pulmonary collapse and collateral emphysema distinctive of the infantile form, are conditions conspicuously absent. The character is that of passive retention rather than of primary bronchial obstruction, though this element is not entirely excluded. Gravitation has a larger share in determining the locality of the changes; and the basic and posterior regions are affected with much greater regularity than in the infant. For these and other reasons the ingravescent, slowly developing form, beginning in the medium-sized tubes, is of special frequency in senile bronchitis. Again, the tissue reaction is of a different quality. Peribronchitis and alveolar wall infiltrations are ill-developed. The consolidations are more definitely broncho-pneumonic or terminal, and, owing to the even operation of gravitation, tend to be confluent. At the same time they are less dense and are usually combined with passive congestion and with oedema, which are not features of the infantile variety.

The preceding remarks must have made it clear that between capillary bronchitis and broncho-pneumonia it is difficult to draw a hard and fast line: the one and the other are made up of bronchitis and of pneumonia. Nevertheless, on clinical as well as on pathological grounds, it is desirable to uphold both forms in our nomenclature. Capillary bronchitis, as bronchitis, is always a general affection of the entire lung leading to severe dyspnoea. It is quite true that broncho-pneumonia in its worst forms often becomes generalised and leads to intense breathlessness, but these are results which need time for their manifestation; their evolution is comparatively gradual, in opposition to the early and often rapid onset of the dyspnoea of capillary bronchitis. Again, although capillary bronchitis, especially in infants, always tends to set up some

1 For two interesting cases of this kind, and for excellent drawings of sections of the lungs, see a paper by Dr. Sharkey in *St. Thomas's Hospital Reports*, 1894, and the writer's article on "Bronchiectasis" in the present volume. Bronchiectasis has also been suggested by Dr. Tooth and Dr. T. H. Fisher. *Vide Dr. Tooth, Path. Soc. Trans.* vol. xlviii. pp. 39-34.
parenchymatous inflammation, the occurrence of a broncho-pneumonia is not invariable, death may occur from the bronchitis before time is given for the consolidation; and in other cases the tendency is rather to peribronchitis, to purulent infiltration of the bronchi; and to dilatation, than to consolidation.

**Symptoms and Diagnosis.** — In the infant or young child the history of the attack, combined with the physical signs about to be described, generally suffices to establish the diagnosis.

The signs are those of pulmonary collapse at the anterior and lateral base of the thorax with inspiratory inward suction of the costal arch, and of emphysema of the upper part of the lung. The resonance due to the latter disguises the dulness which otherwise might have arisen from any pneumonic condensation. Nevertheless examination may reveal a lack of freedom and fullness of the auditory and tactile vibrations. Little air enters the chest in spite of the strenuous efforts of the upper inspiratory muscles and of the diaphragm, the contractions of which drag the sides of the chest inwards instead of expanding them. Sibilis may be heard at first, but they are soon replaced or silenced by râles the loudness of which, always great in the small chests of children, is intensified by any existing collapse or consolidation, and precludes the distinct perception of any bronchiolar or tubular breath sound. In reality these râles, the only sounds audible, do not arise in the capillary bronchi, but are produced by the to-and-fro movements of the secretion within the imperfectly swept medium-sized and larger tubes.

Exhaustion is an early feature; the patients, if not too young to be able to sit up in orthopnoea, rapidly lose that power; and lie pale, livid, and helpless, with hurried respiration, distended nostrils, and extremely rapid pulse. Expectoration does not occur in the younger children, or but rarely, and from an early period in the disease cough may be absent; but both the cough and the dyspnœa are prone to paroxysmal aggravations after remissions.

The temperature varies with the amount of pneumonic action, but probably also with the susceptibility of the individual nervous system. It may rapidly lessen with the advent of cardiac exhaustion and coma.

The disease is usually fatal, and the *prognosis*, except in relatively robust constitutions, is practically hopeless. The duration of the urgent symptoms varies, but, for obvious reasons, is on the average much shorter than in ordinary broncho-pneumonia. The acute stage of the disease does not often exceed five or six days; it commonly destroys life at an earlier date.

In the aged the affection is usually ushered in by a pharyngeal, tracheal, or bronchial cold, which more or less gradually assumes the character of general bronchitis; or it may be grafted upon a chronic catarrh. The extension of the inflammation to the bronchioles is marked by moderate pyrexia, paroxysmal cough and dyspnœa, laboured expectoration, a dusky flush changing to pallor, a rise in the rate of pulse and respiration, and great prostration. All appetite is lost, the tongue be-
comes dry and brown, and muttering delirium sets in, to be followed by deepening coma. In the less rapid cases, evidence of a low form of broncho-pneumonia, associated with oedema and with the signs of bronchitis, may be ultimately obtained at the bases; but, as a rule, the exhausted state of the patient forbids any searching examination of the posterior pulmonary regions. In extreme old age treatment is unavailing, and the disease is almost invariably fatal.

D. CHRONIC BRONCHITIS, CHRONIC BRONCHIAL CATARRH, AND BRONCHORRHEA

In this brief review of a wide and important subject, Walshe's division into four clinical groups will be adopted.

(a) The simple winter cough, moderate, not disabling, accompanied with the easy expectoration of a yellowish white muco-pus, is merely an expression of the bronchial irritation set up by atmospheric conditions; it is frequently observed in children and young adults, as well as in older people.

(b) An aggravated form of the same winter cough is peculiar to chronic bronchial catarrh. The health and strength suffer; and the patients are invalids, though often struggling to pursue their avocations. Decided functional and some organic change may be traced in the organs of respiration, of circulation, and of alimentation; such as short breath, venous fulness both general and portal, and delicate digestion. The winter is spent in a succession of slight pyrexial relapses, during which the expectoration, habitually loose and muco-purulent, may, after being frothy for a while and difficult to raise, become unduly abundant and puriform. The feverish bouts may last a week or a fortnight, during which the appetite is in abeyance, the tongue, stomach, and liver are out of order, and considerable weight is lost. Between these attacks the patient regains some of the previous health and strength, but never shakes off the cough, which may even last, in a modified degree, through the summer.

These patients are protected from graver risks by their general delicacy and invalidism; but the process is progressive and devitalising through the inevitable changes it causes in the lung and in the heart. It induces premature senility and shortens life.

(c) Bronchorrhoea indicates a special group in which the constitutional factor has probably no less a share than the pulmonary changes. Two varieties need description:—

(i.) The thin mucous or thin watery bronchorrhoea is thus described by Walshe:—"In this variety paroxysms of cough and dyspnoea, which may be of almost daily occurrence, or even more frequent, are relieved by copious expectoration of a thin, watery fluid, or of aropy, gluey, transparent substance, like raw white of egg mixed with water; a quarter of a pint of this may be secreted in the course of half an hour
on the decline of a paroxysm." Though sometimes fatal in the aged, the flux is regarded by Walshe as occasionally useful in relieving pulmonary congestion due to mitral disease. This singular affection is well identified by the name of "bronchorrhoea serosa," given to it by Biermer, by that of "mucoid asthma," or by its original name "chronic pituitous catarrh," used by Laennec. The paroxysms of dyspnea and mucorrhoea may be of isolated occurrence in the morning after waking; and the chest, after two hours, may be comparatively clear for the day; or the discharge may be repeated once or twice, producing in extreme instances a daily output of three or four pints; and this may last for years (Laennec). Lebert mentions a case of survival to the age of eighty-two, after thirty years of bronchorrhoea; but Wilson Fox regards gradual failure as being the common tendency, together with increasing dyspnea and delicacy of digestion. Pulmonary and cardiac degeneracy progressively lead to emaciation, anæmia, cyanosis, edema and exhaustion.

Much obscurity still attaches to the pathology of the affection; and it is still doubtful whether the disease is primarily associated with emphysema and bronchiectasis, or whether these be merely secondary changes.

(ii.) Purulent bronchorrhoea or bronchial catarrh is a severe, in-veterate, and progressive affection refractory to all treatment except the climatic. The bronchial discharge resembles in general character that observed in the diffusent stage of chronic catarrhal bronchitis during the exacerbations noted under (b); but generally exceeds it in quantity, and in the semi-fluid, mucoid nature and mawkish odour of the pus. Pathologically the affection differs from simple chronic catarrh, chiefly in the extent of the bronchial and pulmonary change. The mucous membrane is thickened, the bronchial walls infiltrated, and the calibre of the smaller tubes increased, though there need be no sacculations or extensive cylindrical dilatations such as belong to bronchiectasis. Between these two conditions there is, however, no strict demarcation, and mixed forms are met with. Emphysema is a direct and never-failing result of the loaded state of the bronchial system, and of the constant strain of cough. The atrophy of the pulmonary parenchyma contributes the progressive element in the disease, and renders it intractable after it has lasted for considerable periods.

The amount of the expectoration, which may reach one, two, or even three pints daily, is in itself a serious drain; and the cough is a severe tax on the strength. Night sweats, an habitually subpyrexial temperature, and the recurring anorexia or dyspepsia are additional depressants. In connection with the latter, or with disturbance in the function of the liver, or with temporary retentions within, or inflammatory conditions of some of the bronchi, the mawkish secretion may become fetid in odour, sometimes almost gangrenous; and this reacts most unfavourably on the general state.

In spite of these distressing and wearing symptoms, the resistance of some of the patients to the fatal tendency of the disease is remarkable,
and should encourage every effort to procure for them the healing effect of appropriate climate. Failing this, recurring winters bring with them steady aggravation, and life may be cut short by intercurrent broncho pneumonia, or may lapse from gradual exhaustion and cardiac dilatation.

(d) In the fourth classical variety, that of dry chronic bronchitis, the sputum presents characters exactly opposite to those which have just been described. It is extremely scanty, and consists of semi-transparent, tough, pearl-like, roundish, small masses, apparently a highly concentrated and partly dehydrated form of hyaline mucus, in which Charcot-Leyden crystals or Curschmann's spirals are usually contained. The peculiarity of the sputum led Laennec to apply to the affection the name of "dry catarrh," although at times a little watery fluid may be expectorated. The distinctive clinical features are the distressing paroxysmal cough, causing much soreness at the chest; and the dyspnea and oppression of breathing, intensified by the cough, but in most cases kept up by the emphysema, which almost invariably complicates these cases. Laennec described this form as exceedingly prevalent; but, as pointed out by Wilson Fox, he included under "dry catarrh" not only the asthmatic cases, but all forms of nervous and sympathetic cough (gastric, hepatic, hysterical). Walsh regarded the symptoms as mainly due to active congestion of the tubes. Bronchial spasm is doubtless largely associated with the congestion. Indeed, bronchial susceptibility and bronchial irritation are its unmistakable etiological factors. One of the forms of chronic gouty bronchitis belongs to this type. Dry catarrh is also said to be prevalent at seaside places, and to occur after the cure of chronic cutaneous eruptions, and in those weakened by excesses (F. Roberts). The physical signs are those of the dry stage of acute bronchitis.

II. THE SECONDARY AND THE SPECIAL VARIETIES OF BRONCHITIS

A. INTERCURRENT BRONCHITIS

This malady is a complication common to many acute disorders; it will suffice briefly to indicate the relation which the bronchial affection bears to the several diseases.

(i.) The most important group is formed by prevalent affections such as whooping-cough, influenza, summer catarrh, phthisis, and measles; of the last bronchitis is an essential and prominent feature.

(ii.) In some of the continued fevers, but especially in typhoid fever, a varying degree of bronchitis is almost the rule; but its importance is rarely of the first order, and the same remark applies to cases of typhus fever. In enteric fever the severity of the early bronchial catarrh may occasionally mislead the physician for a day or two; and in protracted and exceptional cases the unabated persistence of bronchial râles may arouse uneasy suspicions of general tuberculosis. A malarial bronchitis has also been described.
In scarlet fever and in small-pox bronchitis is not a regular symptom. The occurrence of bronchitis in rheumatic fever, fortunately infrequent, was, before the introduction of the salicylic treatment, a most painful and dreaded complication; it still remains a serious trouble, in spite of the earlier relief afforded to the articular pain.

(iii.) In other affections bronchitis is only an occasional complication. Among them chronic disease of the kidney probably takes the chief place, both as regards the occurrence and the gravity of secondary bronchitis. Gout is also prominent for the frequency of bronchial symptoms. Reference has already been made to the "dry bronchial irritation" so often observed in the gouty, independently of any articular seizure. In cases of retrocedent gout bronchitis may assume alarming severity, and is then probably characterised by extreme congestion. Severe bronchitis of a congestive and catarrhal type may, however, also occur as a precursor of the arthritic attack, usually subsiding with the onset of the latter. A syphilitic bronchitis was described by Graves, by Stokes, and by Munck; and Walshe bestows two pages upon its discussion. It was supposed to occur prior to the cutaneous eruption, and to alternate in gravity with the latter. Bronchitis was also described as complicating cases of syphilis between the secondary and the tertiary stages; and again during the tertiary stage, when it might be unilateral, whereas in the secondary stage it was said to be invariably bilateral. Nothing has been added to Walshe’s description, which is reproduced by Wilson Fox. Indeed, syphilitic bronchitis does not now hold any independent place, and of late years has obtained no recognition; although considerable attention has been given meanwhile to the study of pulmonary syphilis (vide p. 311). The scrofulous bronchitis of Graves is another constitutional variety which has failed to obtain a permanent footing in the practical nomenclature of the disease. Among blood diseases, anaemia, chlorosis, and pernicious anaemia do not specially favour the occurrence of bronchitis. In scurvy, however, bronchitis is not an infrequent complication, and is often associated with haemoptysis, which does not belong in a comparable degree to any of the other forms, except the phthisical and the cardiac.

Walshe draws attention to the occasional admixture with the sputum of substances derived from the blood; such as bile in icterus, sugar in glycosuria and diabetes, urea or its products in uraemia.

(iv.) A special group may be made of those forms of bronchitis which are dependent upon pre-existing pulmonary or intrathoracic disease. Aneurysm, mediastinal growths, or cicatricial stricture of a bronchus (usually syphilitic, vide pp. 71 and 326) may give rise below the seat of stenosis to a localised bronchitis or bronchiectasis, and this may ultimately lead to disorganisation of the pulmonary substance. This result, which is very apt to follow in the rare instances of primary malignant disease of the bronchial mucous membrane, is not often observed in that of the lung, nor in secondary peribronchial malignant disease, whether generalised or occurring in single or multiple deposits. I have observed that the presence within the lung of separate malignant masses of moderate size, even in large
number, may, owing to the distension of the intervening pulmonary tissue, give no signs of consolidation either auscultatory or percussive, and yield only the common physical signs of bronchitis. Gangrenous ulceration into the root of the lung or into a large bronchus is a frequent mode of death in esophageal cancer, and is preceded by the signs of severe bronchial irritation. Emphysema stands in the most intimate relation to bronchitis, both as cause and effect. This association is fully dealt with in another article (vide p. 273). The close connection existing between pleurisy, bronchitis, and catarrh is a matter of every-day clinical observation, and it will be briefly studied under a special heading. Pulmonary phthisis is invariably in part, and often to a great extent, a bronchitic process: it is enough to indicate that, in addition to the general bronchitis which is an intermittent complication of most cases, the local deposits and the local pleurisies of early phthisis determine strictly localised bronchial catarrhs which often raise the first alarm and suggest an examination of the sputum. Lastly, acute pneumonia is sometimes associated with well-marked bronchitis, and forms a most serious, though by no means necessarily fatal complication. I have observed bronchial hemorrhage persisting for several days as a result of this combination. In the pneumonia of influenza the association with bronchitis is the rule; but here the relation between the two diseases is reversed. Bronchitis begins and pneumonia may follow. (Vide art. "Influenza," vol. i. p. 679.)

(v.) Another special place must be reserved for the truly secondary bronchitis of mitral disease, in which clinically, as well as anatomically, three stages may be indicated: (a) A passive congestion of the mucous membrane, the mechanism of which has been described by every writer on valvular disease of the heart as the chief cause of the well-known "heart-cough"—short, slight, dry and habitual, and especially common in mitral stenosis. (b) A mild chronic catarrhal bronchitis, easily set up and difficult to throw off, may occur in both kinds of mitral disease; but is most frequent in mitral regurgitation. It is not, or is but occasionally, associated with streaking of the sputum. (c) A disabling acute bronchitis is the almost invariable agent in overthrowing the fine adjustment previously maintained between the task and the strength of the ventricles. The rest and the treatment necessitated by the cardiac breakdown may subdue for a time the bronchial trouble; but in both forms of valvular disease the bronchial complication inevitably reappears with the relapsing failure of energy of the right heart. At this final stage the process is almost entirely passive and dependent upon the engorgement of the bronchial circulation. In cases of pure mitral stenosis previous pulmonary apoplexies may have cleared up; but their aggravated recurrence often has a direct share in hastening the fatal event. More commonly, in mitral stenosis combined with regurgitation as well as in pure mitral reflux, the expectoration becomes watery with the onset of hypostatic pulmonary congestion and edema; and the final obstruction of the air-tubes with frothy mucus is the immediate result of cardiac and of general failure.
B. MECHANICAL BRONCHITIS

Acute mechanical bronchitis.—Hay asthma is the most striking instance of the production of acute symptoms from the mechanical irritation of suspended particles. The stronger irritants, such as the sternutatories, cannot be long tolerated, and their effect is momentary and slight. No such safeguard limits the inhalation of the pollen of Anthoxanthum odoratum, so noxious to a small class of sufferers. The irritation may involve the entire respiratory tract, including its diverticula, from the frontal sinuses to the small bronchi. Violent and continued sneezing, dyspnoea occurring in paroxysms, oppression and retrosternal soreness, and wearisome cough, which is at first dry but ultimately may produce a varying amount of watery, mucoid, or faintly opaque expectoration, are the main symptoms in cases involving the bronchi. For a further account of this disease the reader is referred to the article on Asthma in the present volume (p. 286).

Chronic mechanical bronchitis is the main clinical feature and the pathological starting-point of all pulmonary diseases due to the inhalation of dust; whether this be vegetable, as in the case of unloaders of grain-ships, grinders of cereals, hemp-spinners, cotton-batters, and coal-miners; or animal, as in that of wool-carders, bedding-makers, brush-makers, and bristle-drawers; or mineral, as in that of stone-cutters, quarrymen, glass-cutters, and calico-weavers (from the china clay used in calico-making); or lastly metallic, as in that of knife-grinders, metal-turners, and needle-pointers (Walshe). In the early stages of all these varieties the symptoms and the physical signs are exclusively those of bronchitis—the sputum alone yielding on examination the special clue to the nature of the irritant. Sooner or later in all of them the mischief strikes deeper; and to the bronchial catarrh, which becomes permanent, are superadded indurative or destructive parenchymatous changes, causing the affections to be classed under the heading of interstitial pneumonia or of phthisis, under which their description will be found. [Ide art. "Pneumoconiosis," p. 242 in the present volume.]

C. PARASITIC AFFECTIONS OF BRONCHI

More closely allied in some of their aspects to mechanical bronchitis than to any other affec tion are the parasitic pulmonary diseases affecting the bronchi,—detailed descriptions of which belong to other sections of this work; namely, hydatid disease and pulmonary distomiasis in the group of animal parasites (vol. ii. p. 1102), actinomycosis (vol. ii. p. 81) and aspergillosis (p. 257) in the vegetable group.

The Endemic Parasitic Hemoptysis of some parts of Japan, of Corea, and of Formosa was, in 1880, simultaneously and independently traced by Manson and by Balz to its cause, the settling of the Distoma Fingeri or
BRONCHITIS

Westmanii near the root of the lung, and the periodical discharge of its yellowish-brown sputa into the bronchi. The rusty expectoration resembles that of pneumonia rather than that of bronchitis, whilst the anemia and progressive wasting are analogous to those of phthisis but have a much more protracted course. Cases of pulmonary distomiasis have not hitherto been reported in this country.

Hydatid disease and the rupture of a hydatid into the bronchi may result in considerable bronchial irritation; but the clinical details of the affection cannot be described here. The occurrence of the cysticercus in the lung is exceedingly rare.

Lastly, we owe to Diesing the account of a unique case of the presence of Strongylus longevaquinatus in the bronchus of a child.

Among the vegetable parasites the most important is the Actinomyces. Pulmonary actinomycosis has long been mistaken for the catarrh of phthisis, of bronchiectomy, and of putrid bronchitis. It can now be readily identified by the discovery in the sputum of the clubbed radiating threads of the fungus, which were first described by Bollinger in 1870.

Pulmonary aspergillosis, relatively common in animals and rare in man, still occupies a somewhat doubtful position in pathology. Originally described by Virchow as a separate disease, the invasion of the fungus had since then been regarded as a mere complication of phthisis and of chronic bronchial affections. Latterly the tendency has been to ascribe to the aspergilli, and particularly to the Aspergillus fumigatus, primary pathogenetic effects. Renou, the latest writer on this subject, considers that in some cases the pulmonary and bronchial affections which had been attributed to tubercle or to actinomycosis were really due to the aspergillus (vide p. 257).

Glanders.—Although the bacillus mallei, like that of tubercle, is not limited to the lung, it deserves to be mentioned in connection with the bronchial catarrh to which it gives rise (vol. ii. p. 513).

D. BRONCHITIS AND BRONCHIAL CATARRH IN THEIR RELATION TO PLEURISY

(a) Acute pleurisy with bronchitis, or acute pleuro-bronchitis.—The not infrequent association of acute pleurisy with an acute bronchitis of the middle-sized tubes is the more worthy of attention, as there is not between these affections that necessary nexus which exists between pleurisy and acute pneumonia; and their occasional combination may be regarded as a definite clinical complex. This view finds support in the etiology and mode of onset, the two affections often arising from one and the same exciting cause and with a simultaneous invasion. I have long been in the habit of using the name "pleuro-bronchitis" to suggest something more than an accidental coincidence; some definite tendency in the subject, and some definite relationship between the pathological processes. Rheumatism seems to be the constitutional tendency, and a simultaneous implication of the pleural and bronchial lymphatics the most plausible
explanation of the process. The occasional occurrence of bronchitis in conjunction with rheumatic fever makes it the more probable that the rheumatic tendency, in itself so often answerable for attacks of pleurisy, may be at the root of this association, even in the absence of any arthritic manifestations; in the same way as non-articular gout is a common and fully recognised factor in the causation of bronchitis.

Cases of this kind are usually classed as "pleurisies with bronchitis as a complication"—a description justified by the relative prominence of the two sets of symptoms. When the pleurisy, as often happens, is of the dry variety, the physical signs of the bronchitis are those most easily obtained; whilst the most urgent symptoms belong rather to the pleural affection. In cases with considerable effusion this relation is reversed; extensive dulness is a prominent physical sign, but the urgency of the symptoms is largely due to the bronchitis, and is often in excess of the loudness of the auscultatory signs special to the latter. When the diaphragm is implicated in the pleurisy, the combined affection assumes unusually severe features, owing to the acutely painful dyspnea, and to the interference with the mechanical function of cough in clearing the air-tubes.

(β) Chronic bronchial catarrh associated with pleuritic adhesions.

—Strictly speaking, the affection which has been described has no chronic form, since, although bronchitis may be chronic, the results of the pleurisy, in opposition to the inflammatory process, are lasting. It is unusual for the acute attack to be continued into a chronic bronchitis; on the other hand, an eventual agglutination of the pleural surfaces, and especially a scaling up of the diaphragmatic groove, are fertile sources of recurring and ultimately of permanent bronchial trouble, in the shape of a localised basic catarrh. Of all local bronchial catarrhs the most common is the apex-catarrh of phthisis, or the recurrent simple apex-catarrh so often determined by the indurated and adherent scar of an old tuberculous lesion. In both cases the same mechanical influence is exerted by the adhesions in hampering the pulmonary movements and in interfering with the systematic play of the expiratory currents.

At the base, and particularly at the lateral base, distinguished in health by its active inspiratory movements, the local catarrh is apt to lead to extensive tissue changes. It is customary to speak of the affection as a "chronic pulmonary catarrh," and of the ultimate anatomical condition as a "chronic interstitial pleuro-pneumonia." We should not lose sight, however, of the essentially bronchitic origin of the mischief. The localisation and the permanence of the catarrh are primarily due to the paralysing influence of the adhesions. The combined irritations exerted within the air-passages by the retained secretion, and without by the recurring respiratory traction, may set up a purely secondary fibrosis; and in some cases the fibrosis is mainly perilobular. Sometimes, however, the affection remains to the end essentially bronchitic with a tendency to rarefaction rather than to condensation of the pulmonary substance. Further consideration will be given to this subject in the article on "Bronchiecstasy."
E. PLASTIC BRONCHITIS

This curious and rare disease, referred to by Galen and studied in 1697 by Clarke and Lister, has been repeatedly described since that time. Biermer deals with a series of fifty-eight reported cases; but Peacock had previously given the first collection of cases on record. Lebert treats exhaustively of the same subject. Dr. Samuel West has collected fifty-two cases recorded since Lebert's article, and compiled a full bibliography. Plastic bronchitis, according to Biermer, occurs twice as frequently in the male as in the female sex, but is not confined to any age from early infancy to advanced life; though most commonly observed in the intervening period. It is still a pathological enigma.

The membranous exudations sometimes occurring in the air-passage form a large and varied group. False membranes may originate from the action of strong fumes or irritating fluids. The inhalation of steam (Parker), or of the fumes of ammonia, or of alcohol in the shape of eau-de-Cologne, are well-known instances. Again, the introduction into the air-passage of strong solutions, such as lactic acid, has been followed by plastic exudation (cf. Hoffmann); and Fritzscbe describes a case in which he attributed the latter to the internal use of iodide of potassium.

As a result of disease, thin false membranes have been observed in the bronchi not only in instances of diphtheria, phthisis, erysipelas, variola, scarlet fever, measles, typhoid fever, and sewer infection (as in the cases of Picchini, quoted by Magniaux), but also in ordinary bronchitis, or pneumonia (R. Koch), in various pulmonary and cardiac diseases, in articular rheumatism (Degler), and in pemphigus (Mader).

From all these varieties of membrane, as well as from the rare forms which have been described as primary diphtheritic and primary pneumococcic (Magniaux), the membrane of plastic bronchitis differs in its greater firmness, which allows it to be expectorated in considerable arborescent masses. The casts occasionally brought up after haemoptysis could alone compare with the latter in size and in consistence, but their origin and their composition are both sufficiently manifest. Thus whilst presenting distant affinities with the minute bronchiolar and sometimes the coarser bronchial plums of pneumonia, with the tubular casts of diphtheria and of membranous tracheitis, and even with the occasional intratubal mucous iuspisssions of acute bronchitis seen chiefly in children, the formation of a continuous arborescent mould of a considerable portion of the bronchial tree stands, by itself as a well-defined, although hitherto unexplained pathological process.

Whether this feature may be trusted as a sufficient indication of the pathological individuality of the affections is doubtful. Plastic bronchitis may possibly be not always of the same kind; it may be due to a variety of causes, just as there are distinct varieties of pseudo-membranous affections. Again, the fact that most of the latter have been traced to a bacterial origin, suggests that a similar causation may at some future
time be made out in plastic bronchitis. In spite of this uncertainty as
to the unity and as to the mode of origin of the latter, we note in the
cases a general agreement which binds them together into a distinct
nosological group characterised anatomically by the recurring exudation,
both rapid and extensive, of coagulable material in the bronchial tubes,
coupled, it is said, with denudation of epithelium, and nosologically by
the mechanical results of the exudation, namely, paroxysmal dyspnoea

and the partial or total expulsion of the casts; or, in the more severe
cases, suffocation and death.

Clinically the disease is clearly distinct from any of the affections
enumerated, occurring rather in connection with some personal idiosyn-
crasy than under the influence of any recognised predisposing circum-
stance, diathesis, or disease, and affecting robust subjects as well as those
suspected of actual or of threatening tuberculosis. It was observed by
Oppolzer recurrently during menstrual periods, with intermittence during
pregnancy; whilst Biermer records several cases occurring during preg-
nancy (Wilson Fox). In its exciting causes—climatic, seasonal, and others—it is closely analogous to common bronchitis, and is in its beginnings almost indistinguishable from the latter, the exudation supervening upon an initial catarrh.

The symptoms are those which would ensue from any extensive obstruction of air-tubes. The cough, which may have a peculiar tone, varies in intensity with the extent and consistence of the recurring thrombi; when they attain considerable size and extension their expulsion is preceded by hacking dry cough and dyspnoea lasting for hours, and in the expiratory type of the spasms resembling that of asthma. The cyanosis is usually moderate. Permanent dyspnoea is present in the proportion of the existing obstruction; during the intervals of freedom from membrane it is not complained of. Slight haemoptysis, more often following than preceding the expulsion of the casts, is very frequent in nearly one-third of all cases (Biermer); or in one-third of the acute cases (Lebert). Sometimes it is considerable; and this has given rise to an opinion that the casts might consist merely of coagulated blood; but they contain no blood-discs, except in their outermost layers which are frequently streaked with blood (Wilson Fox, Biermer).

Ordinary mucous sputum is apt to alternate with the casts, or to accompany them throughout when they are expectorated piecemeal; and a mucous expectoration precedes the expulsion of the larger masses, which are commonly ejected balled up in a slimy investment. Five to ten days is the most common period of retention of the casts; but this may range from one or two days to upwards of three weeks. The daily expectoration of casts may be considerable for long periods, or limited to a few fragments for a few days (Wilson Fox).

The constitutional symptoms in average cases are slight; including little or no pyrexia, except in the early stage, but occasionally a recurrent pyrexia with rigors, little emaciation, and, in a few cases only, dropsy, epistaxis, diarrhoea, or albuminuria, which may not exceed the duration of the attack. The spleen is sometimes enlarged.

A convenient division has been made between a small group of cases running an acute course (from one to four weeks or more), and a much larger group of chronic course, extending over years with intermissions and relapses of varying durations. Biermer again subdivides the acute cases into a mild variety, of shorter duration, in which the ordinary symptoms of a slight bronchitis are simply varied by the expectoration of a few casts; and a severe variety, pyrexial and suffocative, in which death may occur (six cases fatal in a series of ten) before any of the casts have been expelled. The chronic form may long simulate ordinary bronchial catarrh; or it may declare itself early. It also resembles bronchitis in its relapsing character.

The physical signs, ill-defined where the plugs are small and few, are well marked in cases of extensive obstruction. Inspiratory retraction of the chest may occur. At any rate the respiratory movement is locally impaired; and pulmonary collapse may give rise to dulness, whilst full or
exaggerated resonance is elsewhere obtained. The respiratory murmur is diminished or absent; or it may be replaced by sibilant râles, by moist râles of various sizes, and, on the coagula becoming loosened, by loud whistling (Corrigan), by tubular breathing and coarse râles (Van Meerbeck), by a peculiar valve sound (Barth and Cazeaux), or by various flapping sounds described by German authors as schnatterend, schnarrend, and flatter-gerausch (Hoffmann). On palpation a tactile fremitus may also be felt, which has been attributed to the flapping of the bronchial casts.

Prognosis.—The association of the disease with tuberculous phthisis in a certain proportion of the cases somewhat artificially raises its mortality. Putting aside this latter group, and the unusual instances with severe onset and rapidly fatal tendency, in which grave dyspnoea coinciding with scanty expectoration and with extensive collapse of the lung are the most anxious features, the disease, as generally observed, “neither destroys life nor does grave damage, general or local” (Walshe). The liability to attacks may last for considerable periods. The case recorded by Kisch extended over twenty-five years.

Morbid anatomy.—(i.) The casts may be expectorated in mere fragments or in their unbroken state. When freed from mucus, by suspension in water, undamaged specimens are found to reproduce the structure of the bronchi, from the tubes of the diameter of a goose-quill (rarely, of much larger size) down to the finest ramifications, with such perfect accuracy that the site of their formation can be readily identified by comparing them with a cast of the bronchial tree obtained by artificial injection and corrosion. They are, with the exception of the smaller branchings, of firm consistence, and often perceptibly tubular; their bore being commonly plugged with mucus. “Their colour is whitish or pearly gray. They are distinctly stratified, and consist of a structureless or fibrillated basis in which are embedded inflammatory cells, mucous corpuscles, pus cells, pigmented cells, and altered gland cells, and, in their outer layers only, blood cells. They are soluble in alkalies, and also in lime-water” (Wilson Fox).

The expression “plastic bronchitis” does not define the nature of the exudation; and in this there is an advantage, since the casts are invariably mixed products, and may consist largely of mucus, as shown by the action of the solvents just mentioned. Nevertheless they are mainly fibrinous, and owe to fibrin their characteristic consistence. Of this an indirect proof is found in the great rapidity with which fresh casts may be formed after the expectoration of previous ones. Waldenburg, and subsequently P. Lucas-Championnière, had described the occasional presence of fat in the casts. This observation has been confirmed by Model, who finds that the fat occurs as a fine granular deposit, or in droplets between layers of fibrin; it is sometimes to be seen floating in the sputum, which may contain so much of it as to suggest an escape of lymph or of chyle from the bronchial membrane.

Among the formed elements detected by the microscope in the casts
should be mentioned bacteria, and occasionally haematoïdin crystals, Curschmann's spirals, and particularly Charcot Leyden's crystals and eosinophilic cells.\(^1\)

(ii.) The bronchi after death may contain casts in place, or imperfectly solidified curdy collections; or they may be quite clear and present catarrhal mucus only. The membrane may be injected, or pale, as in Biermer's case, in which the epithelial lining persisted under the cast. In Kretschy's instance of an exceedingly rapid reproduction of the casts, the bronchi affected were deprived of their epithelium, and it was evident that the casts were not due to desquamation and transformation of cells, but to a genuine outpouring from the blood-vessels or lymphatics.

Emphysema is almost invariably present. Cases are sometimes cut short by intercurrent acute bronchitis or pneumonia. Traces of pleurisy, recent or antecedent, are sometimes found. Tubercle is present in a small proportion of the fatal cases. Model has recorded its occurrence in 20 cases in a series of 21 cases of the affection. Dilatation of the bronchi has been very rarely found. Mader attaches some etiological importance to the coincidence of pemphigus with plastic bronchitis.

The diagnosis can only be made after the expectoration of some of the coagula. The characters special to the latter, when recognised on examination, should enable us to distinguish the case from cases of intrapulmonary haemorrhage and clotting, of diphtheria, of acute bronchitis, and of asthma.

Treatment of an effectual kind has yet to be discovered. The solubility of the casts in lime-water, originally discovered by Dixon, which strongly suggests the presence within the casts of a large proportion of mucoïd, led Biermer to recommend the inhalation of atomised lime-water, and a case of its successful employment has been reported by Waldenburg.

The only other rational treatment which has been specially recommended is the use of emetics.

The natural process of catarrh by which the plugs are loosened tells in favour of the emollient action of an atmosphere of vapour. This measure, strongly advocated by Walshe, has the advantage of being harmless; and Dr. Ogle suggested that the steam might be medicated with tar or with other stimulating ingredients. Iodide of potassium, internally, was favourably spoken of in 1854 by Thierfelder and Wunderlich. Creosote, tar, turpentine have also been advocated; and Biermer recommends the free administration of mercury in acute cases.

The ordinary treatment of bronchitis is suitable for the generality of cases; and this applies also to the climatic indication, in spite of the disappointing results which have been reported.

An important precautionary measure in connection with the severe dyspnoea to which such patients are liable, is to provide, in all ascertained or suspected cases of the disease, a readily available, if small,
supply of oxygen for immediate use in the event of a sudden difficulty of breathing, whereby time may be afforded for procuring more abundant supplies, and for the adoption of other measures of relief.

Among the latter I would also suggest in future cases the trial of two other rational methods of treatment. The local treatment of the mucous membrane and the removal of the casts are clearly our first indications; and we are now in possession of a method by which they seem likely to be fulfilled, namely, the cautious intratracheal injection of oil or of some mild solvent. These forms of treatment have not yet, so far as I am aware, been resorted to in plastic bronchitis; but I believe that they may ultimately be found more successful, in promoting the expulsion of the casts and in obviating their recurrence, than any less direct method hitherto adopted.

Had we a free choice, our preference would be for some safe means of speedily and completely detaching the bronchial cast; and on applying the remedy we should be aiding nature by following her own method. We might even improve upon the latter if the agent employed could exert some healing action upon the damaged mucous surface. Oil may prove to fulfil both requirements. Its non-irritating character, its power of penetration, and its property of rapidly spreading over, and of protecting moist surfaces, even when used in small quantities only, are important recommendations. The intratracheal method suffers from the lack of any means of regulating the course taken by the injection and of ascertaining whether the latter comes more into contact with the healthy mucous membrane or with the casts. In the case of oil this is happily an unimportant objection: we can trust it to find its way wherever any space offers. At the same time there remains, even in connection with its sparing use, the important reservation that the airway is already greatly obstructed, and that any form of injection might aggravate the dyspnoea.

Some support is given to this suggestion by the happy result obtained in a case of diphtheria, where obstruction of the trachea with membrane was set up after a previous tracheotomy. Creasoted oil (1 in 20) dropped at intervals through the tracheotomy tube excited the desired amount of cough, and enabled the membrane to be expectorated with remarkable facility, so that the case ended in recovery.

The notion of breaking up the membrane agrees less closely with rational principles and with the lines of the spontaneous process of cure. Whichever be the solvents selected for injection, their concentration has to be slight; their action will therefore be slow, and their bulk must be relatively large. Moreover, their influence upon the diseased mucous membrane itself is an anxious question. Above all, we cannot forget that our object is the removal of the plug rather than its destruction. Integrity of the bronchial casts is an important help towards its

complete expectoration: its solution piecemeal might be a doubtful gain, if the smaller branches of the cast were to be left behind.

Among the solvents at our disposal, lime-water would probably be the one least open to objection, putting aside the serious risk connected with the bulkiness of the injection. Lactic acid and the digestive ferments, which Dr. Rolleston has suggested to me as alternatives, are perhaps not equally suitable. Lactic acid has been credited with setting up pseudomembranous bronchitis when accidentally dropped into the trachea, and its employment even in dilute solutions might be open to question. The digestive ferments have been tried in diphtheria of the fauces with very unequal results. Trypsin is free from the chemical objection which may be urged against pepsin, and to a slighter extent even against the vegetable ferments papayotin and papaïn, which act best, though not exclusively, as does pepsin, in acid solutions. The results obtained with papayotin in diphtheria were not encouraging. The favourable opinion entertained by Rossbach has not been shared by other observers, the solution of the ferment having been either too dilute to be effective, or, when of a strength sufficient to destroy the false membrane, not free from damaging effects upon the mucous surface. Papain itself, the more powerful product yielded by the fruit of Carica Papaya (papayotin being derived from the milky sap), has been recommended; but the success of its employment, even with the advantage of the relative accessibility of the surface to be treated, has not been such as to bring it into general use. The effects of the intratracheal injection would need to be studied experimentally before it could be confidently recommended, since any advantage might be outweighed by the slightest irritation set up in the mucous membrane.

In conclusion, the suggestion of a local treatment of the affection, whilst opening up a promising therapeutic prospect, may prove in the event impracticable. In any case the attempt to carry it out should be made with the utmost caution. The local treatment by bactericidal agents and the hypodermic treatment by antitoxins are possibilities contingent upon the results of future pathological discovery.

F. PUTRID BRONCHITIS

In the course of an invertebrate purulent bronchorrhœa the expectoration occasionally becomes putrid; and to this condition in its worst form the name putrid bronchitis has been applied. Putrid expectoration occurs in bronchiectasis, and is commonly associated with destructive pulmonary lesions. Instances of the uncomplicated kind are comparatively rare, the majority of the cases occurring as a late complication of long-established bronchial dilatation.

If foulness of the expectoration in itself constituted a putrid bronchitis, we might group under that name, together with many cases of bronchiectasis, all cases of pulmonary gangrene, of gangrenous tuberculous phthisis, and of putrid empyema discharging through the lung. All
these, however, are removed into other categories by reason of the prevailing importance of their extra-bronchial lesions. It is to the remaining cases, in which the bronchial trouble either stands alone or largely predominates, that the name strictly applies. Although even here the affection is seldom, if ever, absolutely primary, the pre-existing catarrh, emphysema, pleuro-pneumonia, interstitial pneumonia, or fibroid degeneration, whilst they account for a delayed expectoration of the bronchial contents, do not in themselves explain their putrid decomposition. The cause of the latter is intra-bronchial; and two views have been taken of its etiology. According to some bacteriologists putridity is mainly due to the influence of micro-organisms, and the bronchitis is secondary to the microbian invasion—a view to which we shall presently refer. Other pathologists have regarded the bronchitis as the primary event, and have sought to trace the septic process to definite structural changes in the bronchial mucous membrane.

The association of gangrene with bronchiectasis had been dwelt upon by Laennec. It was more definitely described in 1841 by Briguet as affecting the terminations of the dilated tubes. Marfan has recently endeavoured to apply the same explanation to putrid bronchitis. He assumes the existence of a primary gangrene of the bronchi which, he contends, attacks the middle-sized and smaller tubes independently of any bronchiectasis, or in association with but small terminal dilatations. Additional evidence will be needed before this view can be regarded as proved. Meanwhile it is significant that lesions of this kind have not been noticed by other observers; and that in one case in which they were specially looked for after death they were reported, by See, to have been absent.

The view more generally accepted is that an ordinary bronchitis may degenerate into the putrid form, which may or may not be a merely passing phase, but cannot persist for long periods without progressive damage to the bronchial structures and serious risk to life.

That putridity may be set up within the air-tubes by the inhalation of septic matter is a possibility suggested by cases such as that of Tiedemann, in which this was brought about by a leakage into a pulmonary cavity from a traction diverticulum. The attempt to attach the blame to any individual variety of micro-organism is rendered difficult not only by the number of microbes gaining access to the bronchi, but also by the necessity of explaining this occasional failure of the protective mechanisms which normally succeed in repressing them even in cases, for instance those of phthisis, apparently most liable to infection.

The bacteriology of the sputum has already grown to considerable proportions. Among the numerous micro-organisms discovered in putrid expectoration, several of which have been cultivated, J. Luminizer has succeeded in isolating a bacillus which perhaps may be the same as that isolated by Bernabei, giving, after a few days, the same odour as the sputum. Bernabei is inclined to regard putrid bronchitis as directly due to the growth of the specific bacillus which he has described. Hitzig has
likewise described two bacilli, not unlike the bacilli coli communis, also yielding a fetid odour.

The inhalation of oidian albicans was regarded as the cause of the affection in an isolated case reported by Rosenstein; and Canali has reported a case in which actinomycosis was either a cause or a complication.

The sputum sometimes presents a brownish discoloration; it is intensely fetid, either of gangrenous or of foul, sweetish odour. It separates into three layers—an upper mucous purulent and frothy layer, a middle translucent opalescent layer, and a lowermost dirty, yellowish, granular layer containing the solid constituents which have been deposited. As far back as 1850 Ditrich had described the plugging of some of the bronchial tubes by small friable masses, varying in size from that of a millet seed to that of a bean, made up of cellular debris, pus-cells, granules, oil globules, hematoctidin crystals, and various micro-organisms, including the monas and cercomonas described by Kannenberg and by Streng, and leptothrax pulmonalis. These “Ditrich’s plugs” make their appearance in the expectoration and, together with the intensely fetid odour, settle the diagnosis. Fatty crystals (palmitic and stearic), volatile fatty acids (valerianic and butyric), leucin and tyrosin, methyamin, ammonia and sulphuretted hydrogen are also found. Leptothrax pulmonalis occasions a purple, violet, or blue discoloration of the sputum when treated by iodine, a reaction observed by Virchow and by Ganggee. Jaffé’s observations of the presence of minute quantities of leucin and of tyrosin are of interest in connection with the ferment obtained from the sputum by Filehne and by Stolnikow, which they regard as analogous to pancreatic ferment. The same observers confirm the observation that Ditrich’s plugs contain a substance striking blue with iodine.

Morbid anatomy.—Pathological changes special to the affection are comparatively few. The post-mortem appearances are those of an intense bronchitis and peribronchitis with pneumatic infiltration of the surrounding tissue. Pneumonic consolidation may be found extending over more or less extensive patches; but the greater part of the lung is in a state of congestive and puriform edema, and the bronchial glands are swollen and soft. Some of the bronchi may show ulceration, or the mucous membrane is softened in places and deprived of its epithelium; or it may become involved with the adjoining pulmonary tissue into genuine gangrene. Cases of this kind have doubtless supplied Marfan with the basis for his separate description of a gangrene of the bronchi. The collateral changes are varied according to the morbid antecedents of each case.

The symptoms accurately given by Ditrich consist in a sudden onset of feverishness soon assuming a typhoid character, intense depression, collapse, coma and death. The attack is accompanied or preceded by an equally sudden change in the sputum, from the habitual mucous-purulent type of chronic bronchial catarrh to the putrid variety. At the approach of death expectoration diminishes and finally ceases.
The long paroxysmal coughs peculiar to advanced excavating disease, and the gushes of sputum of bronchiectasis are not witnessed; but the frequency of the cough and of the expectoration is often severe, particularly in the pleuro-pneumonic and fibroid cases, in which the thoracic excursions are much restricted. Fever of a remittent type is usually present throughout the putrid stage; and it may be regarded as a measure of the septic absorption.

The diagnosis is based upon the clinical history and upon the negative results of a physical examination for the lesions of bronchiectasis, of phthisis, and of pulmonary gangrene.

The prognosis varies according as the putrid condition of the bronchial contents is grafted upon a simple chronic catarrh, or is combined with deep-seated tissue irritation and overgrowth. In the first group of cases recovery may take place after a few weeks, but relapses will be apt to occur. In the second group the fatal tendency may be hastened by catarrhal pneumonia, acute bronchitis, pulmonary gangrene, pylephrya, metastatic abscesses (including cerebral abscesses), or endocarditis.

TREATMENT OF BRONCHITIS.—Some account has been given of the treatment of plastic bronchitis: that of putrid bronchitis will be dealt with under the heading of Fétid Bronchiectasis. The other varieties of bronchitis will now be considered in turn from the point of view of the abortive, the curative, and the palliative treatment; and of prophylaxis.

I. TRACHEA-BRONCHITIS.—(a) The abortive treatment of simple chest cold, at its preliminary stage of coryza, has probably been more often attempted than in any other ailment, and with a greater variety of methods, most of which are based on diaphoresis and diuresis. The suppression of the coryza has, however, been sometimes attempted by a direct local action on the mucous membrane of the upper air-passages, or by way of the nervous and vaso-motor system. The direct application of powders or snuff variably compounded of quinine, camphor, subnitrate of bismuth, morphine and astringents, and the inhalation of stimulant camphorated vapours, in which ammonia, carabolic acid, iodine, and essential oils are prominent ingredients, have often been prescribed; and remedies of this kind have at times been advertised as specifics. Of internal medication two methods have been used—the tonic and the sedative; on the one hand, liberal doses of quinine or, as strongly advocated by my friend Dr. Isambard Owen, of the tincture of perchloride of iron; on the other, large doses of potassium bromide or some of the antipyretic remedies recently brought into use, especially phenacetin.

The diaphoretic methods do not need any detailed description: they include the traditional help of a Dover's powder, of hot grog, of blankets, of the hot air or Turkish bath, or of the vapour bath, a medicated modification of which has enjoyed some reputation in country districts. In practising this homely and doubtless efficacious method the patient stoops over a vessel of boiling water in which are infused a quantity of selected herbs, while the body is entirely covered with a sheet or blankets; after
a few minutes’ inhalation of the aromatic vapour profuse perspiration is induced, and a cure may result. The late Sir Andrew Clark’s favourite diaphoretic treatment was the hourly administration of ammonium citrate assisted by warm drinks and warm wraps.

(b) The curative treatment.—If in spite of all efforts trachea-bronchitis should advance, its rapid relief can only be secured by rest in bed, fluid diet, warm drinks, and assiduous treatment beginning with a quickly acting purge and combining diuretic and sedative action with the all-important diaphoresis. A hot foot-bath with the addition of mustard, a mustard poultice to the front of the chest, and the inhalation of steam medicated with terebene, eucalyptol, or the compound tincture of benzoin, are valuable adjuncts to the treatment. As soon as decided improvement becomes manifest, iron and quinine should be substituted for the saline remedies, and the ordinary diet resumed.

II. Simple acute bronchitis of the middle-sized tubes.—This affection, though not usually dangerous, calls for judicious and active treatment. The preliminary measures are directed to the relief of congestion of the liver and of the alimentary canal by two to four grains of calomel followed in an hour or two by a black draught. Meanwhile arrangements are to be made for the regulation of the temperature of the room, at a mean of about 65° F.; for the occasional supply of steam, and for its medication with eucalyptus, thymol, or wool-sir oil. The delay before purgation may afford time for a foot-bath or for the application of mustard leaves to the calves, to the upper sternum, and to the shoulders,—or of dry cups over the back. Blisters are unnecessary, and may be inconvenient during the subsequent perspiration. The patient should be kept in bed and allowed to assume the position of greatest comfort, probably one of slight elevation of the head and shoulders.

The more quickly diaphoresis can be obtained the greater will be the hope of checking the spread of the bronchitis. The wet-pack is sometimes used; but more generally the vapour-bath will be preferred, some form of which may easily be improvised. Internally the administration of acetate of ammonium, spirits of nitrous ether and of chloroform, with syrup of squills or of red poppies, and infusion of senega, will be found useful together with other means of keeping up the perspiration. In more active inflammation nothing will relieve the tightness at the chest and the hardness of the cough, whilst reducing arterial tension and keeping the skin moist, better than antimony. Relatively small doses (not exceeding 8 Ml) of antimonial wine, combined with small doses of Dover’s powder or of bimeconate of morphine, the tendency of which is likewise to relax arterial and bronchial spasm and to reduce active congestion, afford much relief. A fluid diet of beef tea, milk, gruel, and tea of moderate strength, belongs to this stage; and, when administered warm, adds much to the action of the treatment.

Expectorants.—Antimony, used as indicated above, undoubtedly loosens the phlegm and promotes its expectoration; but a different combination is called for as soon as the initial discomfort has been allayed, and the
skin, kidneys, and liver have been thoroughly brought into action. It is now time for the direct expectorants—squills, ipecacuanha, carbonate of ammonium, and especially potassium iodide, which, in cases presenting much spasm of the air-tubes, may be successfully combined with the ethereal tincture of lobelia and spirits of chloroform.

Belladonna, one of the early remedies for bronchitis, has not permanently held the position repeatedly claimed for it. This suggests that it may not be equally suitable to all cases, and that in some instances the adjustment of the dose may be a matter of unusual importance, as might well happen with a drug possessing several powerful physiological actions. Each of the latter has in turn been credited with the remarkable results reported by observers. As the element of bronchial spasm in varying degrees enters into all cases of bronchitis, belladonna would be more useful where this factor more largely prevails; as in the bronchitis of asthma and sometimes of emphysema. In cases of this kind the remedy has, in my experience, occasionally afforded more relief when worn as a plaster over the chest than in the form of internal medication. Recently Dr. Sydney Ringer has recalled our attention to its efficacy in bronchitis in relieving the incessant cough and checking the flow of mucus, which, whether viscid and scanty or profuse and watery, is regarded by him rather as an increase of secretion than as an inflammatory product. He prescribes 10 min doses thrice daily or oftener. On the strength of the same property of checking the secretion he suggests its employment in other bronchitis, and in those cases in which aspiration of the chest is followed by a profuse and sometimes suffocating amount of expectoration. The value of belladonna is also advocated by Dr. Murrell, who points out that the same advantages may be obtained by a solution of homatropine. Lastly, Mr. Davies of Sherborne has dwelt upon its "magic" effects as an inhalation, not only in asthma but in acute bronchitis. He recommends the use of 1 grain of the extract in 1/2 oz. of water with Siegel's inhaler, which has the additional advantage of moistening the atmosphere.

Inhalations.—To allay the irritable cough conium or chloroform may be added, in the steam inhaler, to representatives of the turpentine group, such as thymol or eucalyptol; but, for the relief of spasm of the smaller tubes, the dry inhaler is usually found more effectual. It consists essentially of a Woolfe's bottle, provided with a long inhaling tube and mouthpiece, and packed with tow or loose cotton-wool steeped in volatile principles which impregnate the air inhaled through the bottle. The chief sedative agent to be used in all the mixtures for inhalation is undoubtedly spirits of chloroform; the other constituents may be freely varied according to indications and to suit the patient's taste.

Emetics and bleeding, formerly much in vogue and regarded as almost indispensable, are still not infrequently resorted to in some European countries; but they have long been neglected in England. Against this neglect Dr. C. J. Hare has raised an energetic protest. He has

1 For a fuller description of this apparatus by the writer, vide Clinical Journal, 21st Dec. 1892.
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particularly insisted on the great value of emesis in acute bronchitis. In addition to its general and hepatic action, it not only removes the existing accumulation, but, by its mechanical effect, squeezes out of the mucous membrane a large quantity of the effete cellular and mucous material, thus warding off the danger of an implication of the smaller tubes. Its early employment before the onset of this complication would be free from the risk of overtaxing the heart at a time when recovery in great measure depends upon the cardiac energy being fully sustained.

Bleeding was prescribed early in the attack in bygone days. At the present time it is not systematically used as a prophylactic, but is reserved for any more urgent symptoms which might supervene. The treatment of the catarrhal mucopurulent stage of this milder form of bronchitis is practically the same as that of chronic bronchial catarrh, and to that section the reader is referred.

III. Acute suffocative bronchitis of the adult.—In all cases of severe bronchitis, or in any case of mild bronchitis threatening to become severe, the first and all-important indication is to provide a ready supply of oxygen. In a dilute form oxygen cannot fail to be of use even before the onset of urgent dyspnea; and it cannot do harm. When dyspnea has set in, the amount of relief its undiluted administration will afford is limited only by the difficulties of respiration. The objection that the air-distended chest and the choked bronchioles often refuse to inspire has led some authors to regard the treatment by oxygen as useless; but this should not discourage our efforts, for we must bear in mind how relatively small is the bulk of oxygen which corresponds to the ordinary intake of air: during the stage of greatest severity the inhalation of oxygen in some form or other should be maintained continuously. This method tends to fulfill two needs, the pulmonary and cardiac. The excellent results of the local treatment of cutaneous ulcers by an atmosphere of oxygen as originally prescribed and practised by Dr. George Stoker would lead us to expect a like beneficial action upon the mucous membrane. But the second is perhaps the more important function: final success in a protracted and severe struggle for breath is directly dependent upon the vigour of the heart and upon the endurance of the respiratory muscles. Any improvement in their effectual working will tend to increase the subsequent intake of oxygen, and with it the cardiac and thoracic energy.

Another therapeutic agent of importance is moisture supplied as steam. Its application, however, needs care. An excess of steam, or still worse, of the heat directly due to it and to the lamp or fire used to raise it, is injurious. Steam and oxygen work well together; the dryness of the oxygen is tempered by the steam, and the depressing effect of the vapour is relieved by the stimulation of the gas.

In the medicinal treatment three objects must be kept steadily in view from the first, all being urgent:—(i.) to keep up the patient's strength; (ii.) to relieve the bronchial spasm as much as possible; (iii.) to mature, that is, to loosen the catarrh. A preliminary dose of calomel, followed
by a saline, will do good in every way; but this is the full extent to which any depressing treatment or methods involving exertion on the part of the patient can be countenanced. The question of an emetic should, however, be considered, and will need much judgment. This remedy is one exclusively for early employment. A large jacket poultice, made as light as it is possible in front, is of distinct advantage.

Alcoholic stimulation.—Whenever, as in this dangerous malady, the patient's safety lies in the correctness of the estimate we can form of his vital powers as a guide to treatment, the worst evil would be a delusive aspect of strength: the early and over-zealous administration of alcohol entails this risk. Although in certain cases a need for alcoholic stimulation may seem to exist from the first, let us bear in mind that alcohol is not curative in suffocative bronchitis; it should not be our first resort, but be brought in rather as a powerful reserve to carry a desperate position or to ensure its being firmly held. It is impossible, of course, to lay down any general rule as to the time for the employment of alcohol. The physician's estimate of the actual and prospective store of cardiac energy in the individual case is the best guide.

Cardiac stimulants.—Meanwhile, however, cardiac stimulants are to be administered without any delay. A mixture containing carbonate of ammonium in sufficient amount, liquid extract of cinchona, iodide of potassium (3 grains), and antimonial wine (3 m), with syrup of squills and senega, may be administered every two or three hours at first. A few doses of the following mixture may also be at hand for separate administration: 15 to 20 drops of tincture of digitalis, 5 m of liq. strychninae, 20 m of sulphuric ether or of aromatic spirits of ammonia, with compound tincture of lavender or some other excipient. A dose or two should be prescribed against the risks of the night, and may be given at suitable intervals or under special indications in the day.

The frequency of the administration of the expectorant is modified according to the progress of the case; and an occasional intermission of it, with some cooling acid draught as a substitute, may be welcome to the patient. Perceptible amendment should be noticeable within the first twenty-four hours. In the worst cases it will not be a discouraging result if the patient has done no more than maintain his strength.

Mechanically aided expiration.—As previously stated, the existence of emphysema is a specially dangerous factor, and may call for something more than medicinal treatment. I have found decidedly good results from mechanical assistance to expiration; this may be carried out by the attendant, who places his hands, well spread out, over the axillary bases of the patient's lungs, and exerts very carefully timed pressures, judiciously adapted to the phase of spontaneous expiration. The special appropriateness of this method rests on the fact that in emphysematous cases an important part of the dyspnœa and impeded expiration is dependent upon the inherent weakness of the elastic fibre of the air-sacs, over and above the mechanical obstacle produced within the small tubes by the viscid secretion. The larger the share of the first of these
two factors in the individual case, the greater will be the relief obtained. The method may be tried in all cases, but requires to be used with considerable discretion, and with due regard to the patient's feelings, and the effect produced upon the depth and frequency of breathing.

A case cannot remain stationary at this stage; if it do not improve it is rapidly deteriorating, and at any moment, owing to progressive congestion of the right heart, exhaustion may set in. Our duty is to apply the only adequate remedy, venesection, without waiting for the manifestations of extreme cyanosis, cold sweats, jaundice and fluttering pulse. Direct puncture of the right auricle is for obvious reasons impracticable; but the next best means to a sudden and ample depletion of the cavity is to open the external jugular vein, from which 8 to 10 oz. should be boldly abstracted. The benefit obtained is immediate and considerable; the duration of it will depend upon the degree of remaining cardiac energy.

At this moment alcoholic stimulation, if not previously pushed with imprudence, should prove a boon. This is also the time to bring every cardiac tonic to bear, and to inject, if necessary, under the skin 1/10th to 1/10th grain of strychnine. Oxygen, if it had been discontinued, should again be inhaled. Any resulting rally in the cardiac and general energy will afford a fresh opportunity for clearing the chest of loose mucus. After a series of mechanically aided expirations the patient should be encouraged to cough up the accumulations; and, by repeating this process, a good deal may be got rid of.

Very shortly after the bleeding—as soon as the respiration has been attended to—the treatment of the right heart should be resumed. It is all-important to save it from a return of its previous engorgement. A liberal supply of India-rubber cups (six to eight) should be applied to the chest simultaneously, utilising any position accessible without undue disturbance to the patient; and each of them should be reapplied in rotation, so that the depleting action may be kept up for a relatively considerable period. At the same time mustard leaves may be used to the calves.

Good results may be obtained from this alternation of the cardiac and of the respiratory treatment, and from the continued administration of digitalis, bark, and ammonia. If all these fail, no other measures, such as electricity in its various forms, will succeed.

The treatment to be followed in favourable cases, after recovery from the asphyxial stage, is analogous to that which has been described under a previous heading.

IV. Capillary bronchitis.—In infants and small children the same dangers have to be reckoned with, but they are complicated with that of pulmonary collapse, which is practically beyond our power of control, and with the yet more uncontrollable pneumonic changes. Fortunately the onset is often less rapid than in the acute suffocative bronchitis of the adult, and affords a somewhat wider opportunity for treatment.

We are acquainted with three measures of primary importance:
poulting, emetics, and the combined inhalation of steam and of oxygen. Poultices frequently renewed are specially useful in the small chests of children, but it is essential that they should be light. The old practice of the early induction of vomiting is probably the most effectual means of saving life, and is not in itself a source of danger, the act being relatively easy in small children. If the case be seen before the onset of marked respiratory distress the strength will be quite equal to this treatment; and any sign of respiratory retraction of the thoracic base should call for its immediate employment. For threatening pulmonary collapse vomiting is probably the best, if not the only cure. It tends to fulfil two essential needs, namely, the dislodgment of the mucus from the bronchioles, and the inflation of the lobules by the deep inspirations connected with vomiting. This is indeed the safest way of carrying out the method briefly described in the treatment of the adult; namely, that of affording some mechanical assistance to the thoracic and pulmonary movements. Tartar emetic is generally considered to be unnecessarily depressing. A dose of sulphate of zinc, followed by relays of ipecacuanha wine and of lukewarm drinks, is a prompt and effectual agent. Dr. Rolleston has found good results from the hypodermic injection of apomorphine $\frac{1}{16}$ gr. with liq. strychninæ $\frac{1}{2}$ to prevent collapse.

Steam is readily supplied in sufficient quantity with the help of the steam-tent. The latter should never form a complete investment, but be limited only to the head of the box, or to three of its sides. It is dangerous to render the atmosphere oppressive. The inhalation of oxygen needs special management in children. No attempt should ever be made to place the tube into the mouth; it is quite enough to direct the stream of gas towards the nostrils. The first tendency to resist the apparent interference is easily got over, and even infants take kindly to the gas when they have experienced the relief it gives. The administration need not exceed more than a few minutes at a time.

Medicinally the lines to be followed are, with some minor differences, almost identical with those indicated for the adult. Belladonna is a remedy not to be lost sight of in the capillary bronchitis of infants and of young children. Dr. March, who is loud in its praise, administers it in minim doses every four hours for infants of six months old, but reduces the dose on the slightest indication of improvement. He ascribes its value to its stimulant action on the respiratory centres; and this is to be set against the objection sometimes made that it checks the action of the skin and the bronchial secretion, both of which we have been taught to promote.

In the capillary bronchitis of old persons neither emetics nor bleeding are admissible under ordinary circumstances. In them treatment must consist in careful feeding and stimulation, the saving of energy, the promotion of expectoration, and constant and judicious nursing. Oxygen is indispensable; and the regulation of the temperature and of the moisture of the atmosphere is also a point of much nicety.

Theoretically, mechanically aided expiration would seem to be
specially indicated; but the rigidity of the senile cartilages, although not always so great as might be expected, is an apparent objection to the method. Moreover, the other conditions are not quite simple, and aged patients are often intolerant of any mechanical interference with the thorax.

Among internal medicines the stimulant and balsamic expectorants are specially appropriate, and, up to a certain point, successful. Quinine or caffeine may have to be associated with carbonate of ammonia, although they are not in themselves remedies for the cough. Digitalis and strychnine must also be thought of, and called to aid if necessary. Strong counter-irritation cannot be recommended without reservation, and blisters are not advisable. A milder form of stimulation of the skin may, however, be obtained from the application to the front of the chest of flannel sprinkled with a drachm or two of terebene, which also serves the purpose of an insensible inhalation.

As previously explained, capillary bronchitis at an advanced age is a most fatal affection, and the chief aim and result of treatment may be but a short prolongation of life.

V. Acute gouty bronchitis.—The special form of acute bronchitis occurring in gouty subjects, sometimes as a precursor, at other times as a phenomenon of recession of the arthritic trouble, is apt to be alarming in its onset, and sometimes fatal. The special features are the degree of the pulmonary congestion and the irregularity of the heart. The sudden subsidence of these grave symptoms on the reappearance of the arthritis has suggested the old treatment of applying mechanical irritation to the great toe or other joints with a view to calling back the local inflammation. If this attempt should succeed, pulmonary relief will frequently follow; but the remedy is an uncertain one. Moreover, the bronchitis does not always stand in this relation to the articular paroxysms; it may be independent of them; and it should be borne in mind that its gravity is sometimes the expression of a complicating renal difficulty. The indications in the more urgent stage are stimulation and derivation. Among derivatives the most convenient are mustard foot-baths and dry cups freely applied; whilst a rapidly acting purge, such as calomel and senna, should be followed up by mild doses of colchicum and of an alkali, if no special contra-indication should exist.

VI. Symptomatic bronchitis.—The treatment of the bronchitis associated with the infectious fevers, sometimes, as originally observed by Laennec, throughout their course, does not often call for separate attention. The management of the bronchitis of asthma and of hay-fever, of mechanically induced bronchitis, of the bronchitis of phthisis, and of that incidental to other parasitic diseases, will be considered in other sections of this work.

VII. Chronic bronchitis.—The varieties of chronic bronchitis call for some detail in their several treatment; but for all of them our therapeutic agents may be arranged under four main indications: (i.) the atmospheric treatment, including the climatic; (ii.) the topical, including counter-
irritation; (iii.) the medicinal, and (iv.) the constitutional, including the balnear treatment.

(i.) The value of climatic treatment is demonstrated by the rarity of chronic bronchitis among inhabitants of more temperate zones, and by the improvement of invalids from the north whilst under the warmer influences. For the larger number distant journeys are impracticable; artificial atmospheric conditions must therefore be devised. The essentials in an artificial atmosphere are purity of the air-supply, freedom from suspended particles, and due regulation of temperature and moisture. A constant renewal of air without oscillations in the temperature, and a proper supply of moisture—the dryness of artificially-heated air being specially noxious in chronic bronchitis—are problems claiming earnest attention in practical hygiene. Evenness of temperature and of moisture, if they can be secured, will enable the chronic bronchitic to spend indoors the periods of more wintry weather, whilst occasionally enjoying exercise in the open during warmer spells. But this after all is merely protective treatment, rather devised for safety than for cure.

(ii.) Topical treatment. — Atmospheric therapeutics aim at something more than mere prophylaxis, and are needed in the more active stages. Strictly, the term should be limited to the volatile agents, which can be used to impregnate the air at the normal temperature. Members of the turpentine group—terebene, pinol, cresol, eucalyptol, creasote, tar, carbolic acid, iodine, and the like—are all in some slight degree volatile; though not to the extent observed in the case of chloroform, alcohol, and ether. Chloride of ammonium vapour, supplied by means of a special inhaler, may be combined with some of the vapours enumerated. "All these substances may be inhaled in greater concentration when combined with steam, and this method has the most beneficial effect. The practical means of volatilising carbolic acid and other agents at varying temperatures have received much attention from Dr. Robert Lee.

Reference has already been made to the dry inhaler by means of which the more volatile, as well as a slight proportion of the less volatile, substances can be directly inhaled with the inspiratory current. Lastly, the fine atomising or nebulising sprays, for which some excellent apparatuses have recently been introduced, enable us to add to the list of the atmospheric agents almost any of the non-volatile substances, provided they be soluble. Common salt, bicarbonate of sodium, chloride of ammonium, alum, tannin, and various astringents may be thus used as required. A proportion of the spray probably passes the glottis, though doubtless the greater part is condensed on the pharyngeal walls. To this minimum introduced into the lung we cannot fail to attribute a share in the marked benefit obtained; and we recognise in it a first step towards the more vigorous topical treatment by intralaryngeal injections, from which excellent results may be expected in a large number of cases. The laryngeal insufflation of fine powders is less commonly used, and, owing to the ciliary function, it is doubtful whether their action would extend much below the trachea itself.
Counter-irritation is of undoubtedly value in most forms of chronic bronchial catarrh, for the treatment of the exacerbations. Its usual modes of application are the irritating liniments and applications, such as croton oil, blistering, and the actual cautery. The latter is extensively used in France under the name of "pointes-de-feu," for the relief of cough, of local pain, and of profuse expectoration. For the same objects blistering is invariably useful. In patrial bronchitis blisters may prove of decided service in checking both the factor and the amount of the expectoration; and in those cases where, owing to fibrosis of the lung, Chaplin's treatment by creasote inhalation is not successful, this mode of relief should be tried.

(iii.) Internal treatment has regard not only to the immediate relief of the bronchial trouble, but also to constitutional requirements. The list of those drugs which are beneficial to the membrane need not be given in full; their active constituents are usually such as can be exhaled into the lung, so as to take effect on the bronchial membrane. All the derivatives of tar, and tar itself, the turpentine, and the balsams are valuable in the treatment of chronic bronchitis. The more direct expectorants are also sometimes needed, especially when tonics, which are otherwise to be preferred, act as a source of irritation. The preparations of conium, squills, ipecacuanha, senega, in combination with mild salines, will prove of value in these irritable forms; and if there should be much spasm, morphine, belladonna, hydrocyanic acid, lobelia, and like agents may be required. Of the internal remedies taking special effect on the secreting function of the membrane four groups may be especially mentioned: (a) Certain balsams, such as balsam of Peru, of tolu, and the compound tincture of benzoin; among the oleo-resins copaiba, and among the tar derivatives creasote and guaiacol (to be taken in capsules). These remedies stimulate the membrane and tend to diminish the catarrh. (b) Iodine in all its combinations, and particularly as iodide of potassium, has the opposite tendency, and is especially useful when the mucous membrane is dry and the expectoration scanty and difficult, as in the so-called dry catarrhs. (c) Sulphur and the sulphides have long enjoyed a reputation for the relief of suppulsive conditions, and their checking influence on the profuse muco-purulent discharge of bronchorrhea and the worst forms of catarrhs is striking. When this can be combined with the tonic effect of a bracing air and with thermal treatment, results may be obtained such as have established the reputation of Harrogate in this country; and of Eaux Bonnes, Cauterets, Luchon, Aix-les-Bains, and other stations abroad.

At all thermal stations patients are subjected to a limited course of treatment by baths, mineral-water drinking, and exercise in the open air. When sulphur is administered to a patient treated at home the same attention should be given to a limitation of the period of administration, lest irritability of the mucous membrane or irritability of the skin should be induced. Lastly, (d) cod-liver oil, when tolerated, is an invaluable remedy.
(iv.) Constitutional treatment.—As a rule, a slightly purgative plan is of
great value; indeed this is one of the favourable aspects of the treat-
ment by sulphur. Various mineral waters may be used, and, with the
same object, patients are sent to various medicinal springs.
The cardiac indication is usually obvious. The right heart needs not
only to be cured of its dilatation, but if possible toned up. Strychnine,
digitalis, strophanthus are thus direct agents in relieving chronic
bronchitis by reducing the pulmonary congestion. We should not for-
get that an excellent way to strengthen the right heart is to strengthen
the left. In chronic bronchitis shortness of breath leads to muscular
inertia and atrophy; for this there is a remedy in oxygen inhalations, or
in their equivalent, systematic purposive hyperpnoea. Patients would
gain much by training themselves to breathe to the utmost mechanical
advantage, and by cultivating general muscular exercise, at first purely
passive, but ultimately active. A general recovery of neuro-muscular
energy, other circumstances being favourable, will act most beneficially
on the chest through the great improvement in cardiac strength. For
artificial methods of lung gymnastics the reader is referred to the chapter
on Aerotherapeutics in the first volume.

Lastly, hematinic remedies are wanted in a large number of cases;
this is a special indication in the groups of protracted mucus-purulent and
of all severe purulent catarrhs; and these are also the cases which most
benefit under cod-liver oil. The administration of iron is not to be
limited to those patients whose anaemia and wasting are obvious; iron
and quassia, or some other bitter, and particularly cinchona, are not only
well tolerated, but of direct value as stimulants to the relaxed and con-
gested bronchial membrane in cases where venous embarrassment gives
rise to a deceptive appearance of plethora.

In all cases of inveterate catarrh, but particularly in those which
from their severity deserve the name of bronchorrhon, a warm and
equable climate during the winter is indispensable. Various sheltered
stations have been recommended in this country, such as the Undercliff,
Torquay, Falmouth, Ilfracombe, Minehead, the Scilly Isles, and others.
Some patients will derive great benefit from a winter's residence in the
bracing atmosphere of Thalass. Nevertheless, whenever this is possible,
the Mediterranean seaside resorts are to be preferred; and among them
the more sheltered, such as Mentone, San Remo, Alassio, Rapallo,
the Riviera di Levante, Capri, Malaga, Corfu, Egypt, and suitable re-
sorts on the North African coast. This large subject is fully treated
in the article on "Climate in the Treatment of Disease" in the first
volume.

Unless the membrane be protected from irritation for prolonged
periods no lasting improvement in the condition can be looked for.
Permanent residence for some years in a favourable district is the only
really curative treatment; but this may with benefit be combined with a
summer visit to one of the hot sulphur springs; or to Ems, Soden, or any
of the saline mineralated and carbonated springs, suitable for the individual
case. The opportunities for permanent residence in eligible climates are widening year by year.

Prophylaxis. (i.) Prophylactic measures between the attacks. — No risks should be incurred by the chronic bronchitic patient. Sudden changes of temperature, as at sunset, or from walking out of heated rooms into the cool of the night, or into cold and damp buildings after exposure to the sun, cold winds, dampness of air and of soil, dusty localities and occupations, great variations in the amount and thickness of clothing, chill from damp underclothing after perspiration, and, almost above all, inactivity of the liver should be sedulously guarded against. The merely passive avoidance of obvious dangers is, however, a lame policy; we should be prepared for those which are apt to fall upon us unawares. Bracing resorts help us in this by toning up the nerves and tightening the membrane. A great deal can be done by the patients themselves in utilising the opportunities afforded by protective climates for the combined development of muscular energy and of respiratory activity. It is to be observed that vesicular emphysema is almost entirely a passive change, not brought about directly by voluntary expansion of the chest. In my opinion, systematic and graduated respiratory exercises, though they may stretch, tend to strengthen rather than to weaken the elastic fibre; and since they are based upon the performance of effective expirations, they would appreciably relieve the passive emphysematous distension. Much of the hepatic and of the local bronchial congestion will also be corrected by the greater activity of circulation thus initiated; and increased oxygenation will promote the growth of a less irritable and delicate epithelium.

The same tonic system can profitably be applied to the skin by means of a well-planned course of rubbing, bathing, and douching. All these measures need long perseverance before their beneficial effects can be fully secured; but their sedulous employment will bring with it an almost assured reward.

(ii.) Prophylaxis in early bronchial delicacy. — Yet more important is the subject of prophylaxis in infancy and childhood. The bronchial tubes are apt to suffer early in life; worst of all is the mischief arising from a severe attack of whooping-cough. Inherited family tendencies may in some children point also to a future liability to bronchitis. Moreover, in the case of all children, and especially of town-bred children, we have to deal with the liability induced by climate. All infants in this country, but in special and varying degrees the offspring of delicate, asthmatic, bronchitic, and gouty parents, stand in need of the help of preventive measures. If this were thoroughly understood and our practice regulated accordingly, a vast saving of life and health would be secured. The prophylactic plan suggested can be summed up in one word. It is a "hardening" plan carried out with vigilance and discretion; its essentials lie in the management of respiration and atmosphere, of temperature, of clothing, and of the skin.

Respiration and the atmosphere. — It is not sufficiently recognised
that the bronchial tubes and lungs are constructed for the air we live in, and conversely. Specially strong is the prejudice against night air, which in itself is exceedingly beneficial. The innocuousness, for the bronchial membrane, of the higher temperatures of atmospheric air needs no demonstration; the innocuousness of extremely cold air, though it is not usually brought home to us, is evidenced by the ease and comfort with which respiration is carried on in arctic temperatures. Much of the objection to night air is generally directed against the dampness of it; but moisture need not in itself be detrimental; indeed, as we have seen, it is often used as a remedy. Nevertheless any of the normal atmospheric peculiarities may cease to be beneficial and may be turned into a source of irritation by a systematic substitution of artificial atmospheres for that provided by nature.

The great prophylactic method is to see that infants and children live and sleep in the open air as much as possible during the day, and enjoy as much free ventilation from the outer air at night as may be compatible with prudence. The full measure of this fresh-air treatment may be attained by degrees only; but it should be persistently aimed at. In towns this rule is of much greater importance than in the country. The extraordinary amount of health enjoyed by the children of the poor, in spite of so much that is depressing, is in great measure to be explained by the out-door life they are obliged to lead in their dark streets and alleys.

The skin and temperature.—More serious still than the neglected training of the aerial mucous membrane is the neglected education of the heat-generating function in relation to the skin. An excessive amount of clothing by day and by night, with wraps round the neck and wool next the skin, excludes too completely the oscillations of the outer temperature which should act as stimuli to the cutaneous surface. Moreover, the constant moist heat which is thus maintained tends to make the skin delicate and to depress its power of reaction. Flannel underclothing is the best and safest for subjects too feeble to keep up their body heat; and it is an invaluable provision against unusual variations in the atmospheric temperature or in cutaneous action, as in athletics, campaigning, rapid journeys through extremes of climate; but its constant use is not part of the systematic training of the skin. In healthy children and adults it is as a rule superfluous at night, although indispensable for children suffering from rickets, restlessness in sleep, or enuresis. When it is worn during the day the outer garments should be made proportionately lighter. To pile up heavy outer clothing over thick flannel undergarments is bad hygiene, and cannot fail to weaken growing children.

Hygienic treatment of the skin.—Active means of promoting a vigorous habit of the skin should not be neglected. Massage is almost superfluous in children, whose life is perpetual movement. The chief indication is the sponge bath or the douche and rubbing. Few children will fail to take kindly to the cold bath if trained with sufficient tact to its use. As a rule, there will be no difficulty in obtaining the glow of
cutaneous reaction after the bath, by friction with a coarse towel. In some constitutions the cutaneous circulation is slow to recover itself, and some special modification of the bath is called for. An essential precaution is the application of plenty of warmth immediately before and immediately after the cold sponging. The child may be placed for a minute or two into a warm bath, transferred to another bath for cold or tepid sponging, and again put into the warm bath for an equally short time, before towelling. An alternative, and in some ways a better method is to sponge the surface rapidly with warm water whilst the child is standing in a warm foot-bath. After the cold sponging he is to stand again in hot water whilst the body is being rubbed dry. The latter method is extremely simple and very effectual. Adults also who otherwise might be debarred from the boon of the cold bath are in this way enabled to resort to it with perfect safety and with enjoyment. In nurseries a bright fire should be burning before the cold morning baths are given. The daily cold affusion is of the greatest value as a direct protective against "catching cold"; and its systematic use must be reckoned among the most powerful helps in training a habit of resistance, and of ultimate indifference to all ordinary bronchial or cutaneous impressions, in those whom inherent debility or inherited predisposition would otherwise have exposed to ever-recurring risks of bronchitis.

WM. EWART.

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BRONCHIECTASIS

Bronchial dilatation, when slight or limited to one tube, may escape clinical observation; usually it involves several of the cartilaginous bronchi, and then gives rise to unmistakable symptoms, constituting a clinical disease to which the name "bronchiectasis" is appropriated.

The name "bronchiectasis" is also in common use in descriptive pathology; but the affection is far from presenting in its anatomy that uniformity which we recognise in its clinical symptoms and signs. Walshe says: "The conditions of disease to which dilated bronchi may form an adjunct are: bronchitis, acute and chronic; emphysema; constriction of the tubes themselves; acute and chronic pneumonia; cirrhosis of the lung; phthisis, cancer, and chronic pleurisy with contracted side." So great are the differences between the various pulmonary lesions thus apt to be associated with it, that we regard bronchiectasis as a structural change which may result from a variety of morbid processes, rather than as a definite and independent pathological product.

Clinically speaking, bronchiectasis is a chronic affection implicating bronchi of good size; to this it is that the literature of the subject almost exclusively refers, and that the present article is chiefly devoted. In its anatomical sense the name is more comprehensive: it applies to the secondary and slighter dilatations as well as to those which are sometimes described as primary; and it belongs with equal right to air-tubes of all sizes. In a complete clinical nomenclature the affection as it occurs in the bronchioles should not be left out, and we should recognise a bronchiolar dilatation or bronchiolectasis, as well as a bronchiectasis; both varieties occur independently, and are clinically important. With a view to mark the distinction between them, which has not been much dwelt upon, they will be described under separate headings.

In another group, that of the secondary bronchiectases, tubes of inter-
mediate size are commonly involved. This variety does not possess the same clinical interest, and our references to it will be incidental only.

I. **CAPILLARY BRONCHIECTASIS, OR BRONCHIOLECTASIS** (including the so-called "Acute bronchiectasis").—This type of bronchial dilatation stands out in clinical contrast with the ordinary variety, while its distinct pathological features throw light upon the pathology of bronchiectasis in general. In its most striking form it occurs in children as an acute process; and as such it was described by Andral, Milliet and Barthez, and others as "acute bronchiectasis." The post-mortem recognition of a dilatation of the small tubes in connection with certain clinical symptoms noted during life led observers to infer that in other cases also, which presented the same symptoms but ultimately ended in recovery, the same lesions had existed without proving fatal; and it is upon this assumption, which is probable enough but not capable of demonstration, that rests the current belief that children may completely recover from acute bronchiectasis. Granting that recovery may be possible, it may in a proportion of the cases be but partial; and in these the dilatations persisting in some portions of the lung may lapse in the course of years into the common bronchiectasis of the larger tubes.

In the adult localised dilatations of bronchioles are frequent in chronic bronchial catarrh, and may follow an acute purulent bronchitis; but, so far as I have observed, they do not extend to the whole lung; and their accompaniment is not atelecstasy, but chiefly emphysema. Their value is rather that of a complication than of a disease. Since, however, their symptoms do not differ from those of a catarrhal bronchitis, and do not add largely to the fatality of the latter, it would be hard to say that acute dilatation of the smaller tubes may not occur more often in the adult, and more often be the origin of true bronchiectasis than is commonly thought.

In its chronic form bronchiolar dilatation is relatively of small importance. It is a local lesion secondary to the respiratory inactivity of a pulmonary district disabled by bronchial obstruction, or hampered by adhesions; in short, to imperfect expansion of the lung with resulting accumulation of mucus. Its most common seat is the apex of the lung in phthisis, where, although an old vomica may have undergone considerable contraction, the collapsed alveolar substance in its vicinity had failed, owing to surrounding fibrous changes, to expand again completely. Small thin-walled bronchi, distended with clear or purulent mucus, may often be seen in these partially aerated and inactive remains of healthy lung tissue.

The same change may, however, be met with in an opposite association, in emphysema due to chronic bronchial catarrh. Where the emphysema tends to become bullous the dilated bronchioles may take a share in the formation of the bullæ, and occasionally perhaps in the production of pneumothorax.

*The acute form* is of much greater clinical interest. In the majority of cases its mode of origin is tolerably obvious from the clinical
history. It is illustrated in the cases collected in Dr. Walter Carr’s paper on “Bronchiectasis in Young Children”; and it is well displayed in the drawings reproduced, by kind permission of the editors of the St. Thomas’s Hospital Reports, from Dr. Sharkey’s paper on “Acute Bronchiectasis.” In children the lesion is essentially the result of an acute catarrhal bronchitis and peribronchitis, with multiple and widely-diffused secondary collapse. As immediate factors, the bronchitis of measles and of whooping-cough probably contributes more cases than any other kind. The course of the disease and its clinical features are not very distinctive, as may be gathered from the brief account given by Dr. Sharkey of his two cases. The cases were not diagnosed as bronchiectasis during life.

In the first patient, set. 2 (Fig. 2), there was no previous record of illness except measles. The lungs after death were pale and curiously dotted with black pigment spots, hard to the touch. The centre of each of these was occupied by a small bronchus. The bronchioles were everywhere dilated, and scattered here and there were what appeared to be small miliary tubercles; but the other organs were free from tubercle. Microscopically acute peribronchitis was found, accompanied with extreme bronchiectasis, and a little, but very little, emphysema. No genuine tubercles were seen.

The other patient, a child aged 4, was under observation from May 7th to June 10th, 1893. He had always been healthy until cough began, two months prior to admission. Since then he had spit up thick phlegm, and had vomited three or four times a day; but he was able to attend school until admission. At that time he presented a dusky flush, rapid breathing, no marked dulness on percussion, no tubular breathing, but crepitations over the whole of both lungs. The pulse-rate was 136; the temperature 102.6; the respirations 44 per minute. The temperature gradually fell, but on June 3rd subcutaneous emphysema occurred; otherwise no material change took place till death. The lungs were found bulky, their surfaces thickly strown with soft, round, transparent, bladder-like elevations, the cavities of which were perfectly smooth, and either empty or full of frothy muco. Scattered through the lungs these small cavities, the largest of which was about the size of a pea, gave a worm-eaten appearance. The larger tubes were not perceptibly dilated or diseased, but there were numerous patches of broncho-pneumonia of small size, and here and there some collapse; but no tubercle. The microscope detected widespread acute bronchitis, peribronchitis, broncho-pneumonia and pulmonary collapse. The bronchioles were extremely dilated, and there was also considerable emphysema.

1 diagnosis of dilatation of the bronchioles cannot be made with any certainty; even in children; or even when, as in these cases, the change is general and extreme. At most its presence can be guessed at. Neither percussion nor auscultation can fasten upon any trustworthy sign, and the character of the expectoration does not differentiate the affection from severe catarrh. In its localised occurrence in the adult dilatation of the small tubes is still less capable of recognition.
Prognosis.—The acute puerile form, as shown by the cases narrated, is sometimes the result of a catarrh so severe as to be in itself fatal. In other cases, perhaps, the bronchiolar affection may be limited to a portion of the lung; and the catarrh getting well, the small tubes, may lose...
That this does occur is the view generally held; but, so long as a diagnosis of capillary bronchiectasis by physical signs is impossible, this must remain an unproved though a plausible opinion. Considerable likelihood has recently been added to it by the successful results obtained in cases of bronchiectasis in the adult.

The treatment of an affection incapable of diagnosis cannot be
laid down with any definiteness. In the chronic form none may be needed, the general symptoms being themselves chronic and sometimes unimportant. In the acute affection the presence of the bronchitis and of the catarrh supplies all the important indications; and these are sufficiently dealt with elsewhere. The great object in bronchitis being to prevent stagnation in the bronchioles, of which this form of dilatation is one of the results; the treatment of both diseases is practically identical.

II. Bronchiectasis.—Morbid anatomy.—Since the time of Laennec, to whom we owe the first anatomical and clinical account of the disease, three main varieties of dilatation have usually been described: (i.) the regular or cylindrical, (ii.) the fusiform, and (iii.) the globular or sacculated. A modification of the globular is the bead-like variety, in which a tube may present at intervals a normal calibre between successive distensions. Sacculated dilatations, with that exception, are terminal. The cylindrical expansions, on the contrary, affect the tubes as they pass towards the periphery. If a further dilatation should occur at their peripheral end, and cause the latter to become bulbous, the fusiform variety is brought about.

The largest and most extensive bronchiectases are found in more or less fibrotic lungs. Dilatations occurring in emphysematous surroundings are usually either fusiform or bulbous dilatations of single tubes, or cylindrical expansions of sets of smaller bronchial tubes which may be filled with catarrhal secretion.

Congenital bronchiectasis, the varieties of which constitute a distinct group, may be regarded as a malformation, or as resulting from some intra-uterine disease, perhaps syphilis. Usually one lung only is affected, and may present a large cyst with a central space branching into a peripheral set of intercommunicating secondary and tertiary cysts, with serous contents. Instances of this kind have been described by Grawitz, by Kessler, by Meyer, and by Fränkel. In another variety described by Grawitz, numerous separate cysts are formed on the bronchi of the third and fourth order; some of them communicating with the bronchial lumen, others being entirely closed. Goitre was found associated with this malformation.

In the atelectatic bronchiectasis described by Heller there is an abnormal growth of the bronchial cartilages, together with remnants of unexpanded, non-pigmented fetal lung tissue; and the epithelial lining is not of the columnar ciliated, but of the pavement type. Cases have also been described by Gairdner, by Francke, by Herxheimer, and others.

Lastly, congenital bronchiectasis may be due to a dermoid growth within a bronchus. An almost unique specimen, now in the Museum of St. George's Hospital, was exhibited by Dr. Cyril Ogle before the Pathological Society on March 2nd, 1897. The patient, a male aged twenty-eight, had suffered intermittently for five years with cough and hemoptysis, and ultimately died from profuse hemorrhage, after a period of hectic temperature, fetid expectoration, and physical signs suggesting empyema or bronchiectasis; both of which were found after death. The
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vessels may persist for a long time in the trabecula, the bronchi—even those of large size—which traverse the diseased region are laid open and removed by ulceration at an early stage.

In the emphysematous tissue surrounding very chronic and practically healed lesions of the apex it is not uncommon to find unimportant dilations of the peripheral air-tubes due to a rarefaction of the lung substance; these, however, are hardly to be dignified with the name bronchiectasis.

The changes in the mucous membrane and in the outer bronchial coats.—So long as the mucous membrane escapes destruction—and it is remarkable how long it will remain intact—it presents the signs of catarrh. In its later stages, however, it loses the velvety look, and assumes rather a smooth and shiny appearance consistent with atrophy of the epithelial layer. Most probably in all cases the atrophic changes prevail; although in some they may be limited to the internal coat, the adventitia taking on an inflammatory action which explains the thickening described as the alternative change. In Walsh's words, "The walls of such dilated portions of tube, commonly thick, and exhibiting the several characters assigned to tubes affected with chronic bronchitis, are, on the contrary, in rare instances thin and almost transparent." In general the instances of thickened bronchial membrane are those in which the inflammatory process extends around the dilated tubes into the pulmonary and interstitial tissue; whilst the bronchiectases with thin walls belong to the emphysematous group.

The condition of the mucous membrane differs much in the several varieties and stages of the disease; it is swollen and congested in the acute form (as in the acute cases of childhood), and in those chronic cases which remain free from much accumulation; congested and atrophic in cases of an opposite process; and, lastly, sometimes ulcerated or even gangrenous in the later stages of extensive retention, when septic inflammation has supervened.

Hanot and Gilbert have connected the occurrence of hæmoptysis in bronchiectasis with the marked alterations described by them in the blood-vessels, which may form in the submucous tissue an extensive cavernous network, interspersed with numerous minute aneurysms.

According to Professor Hamilton the basement membrane of the original bronchus seldom gives way, but becomes stretched and attenuated. "On the basement membrane stratified columnar epithelium in a wonderful state of preservation may sometimes be found."

The changes in the surrounding pulmonary tissue.—As stated by Walsh, "The surrounding tissue is either slightly condensed by pressure, hardened by chronic pneumonitis, rarefied by emphysema, or perfectly natural."

Ulceration occurring in a sacculation is prone to set up fatal pulmonary gangrene. This was observed in twelve cases out of twenty-four by Rapp: in three out of forty by Barth; and in five out of fifty-four by Biermer. The gangrene, as in an isolated case mentioned by Lebert, may perforate

1 Marfan devotes a special chapter to "gangrene of the bronchi," which he regards as distinct from pulmonary gangrene and from purpuric bronchitis.
a branch of the pulmonary artery. Perforation of the pleura would probably be less rare than it is but for the adhesions which so commonly exist and check the production of pneumothorax and of subcutaneous emphysema. Both these conditions have, however, been observed.

Sir T. Grainger Stewart has described the process of absorption by which bands are left stretching across bronchiectatic cavities; or the latter may become multilocular, as often seen at the pulmonary base.

Inflammatory changes in the pulmonary tissue in the vicinity of the lesions are common. Acute pneumonia was recorded in twelve cases by Biermer, and in five by Lebert. Some inflammation also extends to the air-tubes in general. Hypertrophy of the bronchial cartilages, and a calcification of the walls of the dilated tubes—which in the bovine species is stated by Biermer not to be uncommon—have been described in isolated cases.

A cystic form of bronchial dilatation has sometimes been described (Biermer, Briquet); the cysts, which average the size of a walnut, being associated with a bronchial stenosis situated higher up. The contents may be serous, mucous, caseous, or even calcareous.

The secretion found in the dilated bronchi at different stages varies in its factor, and in the proportion of its fluid and of its solid constituents. Among the latter may be found: (a) recent mucus; (b) small casts, described by Dittrich and by Grainger Stewart, sometimes presenting epithelial flakes; (c) stale, opaque mucus undergoing granular and fatty degeneration; (d) micro-organisms of putrefaction (including sometimes sarcina and leptothrix pulmonalis, to which is due the purplish colour reaction of the bronchial casts on the addition of iodine, etc.), but no bacilli of tubercle. Occasionally the contents are blood-stained. Very frequently, though not always, crystals of the fatty acids and of cholesterol are found, especially in the fetid stage. Calculous concretions (Stokes, Dittrich) have also been observed.

Pathological changes in distant organs.—Various accidental complications have been described, such, for instance, as cancer, which Barth recognised in 8 out of 43 cases. The associated changes special to the disease are chiefly those connected with the obstructed circulation through the lungs: secondary dilatation of the right side of the heart, and venous congestions in the portal and in the systemic circuit. Valvular lesions may coexist, but do not appear to be traceable to the disease; pericardial adhesions sometimes occur as an extension of the pleuro-pulmonary fibrosis. The liver is almost always congested; it may present fatty change, and is sometimes lardaceous. Lardaceous degeneration also occurs in the kidney; and catarrhal nephritis has been recorded. Septic abscesses may be set up in various situations; one of their most common sites is the brain.

An articular affection, analogous to gonorrhreal synovitis, or to that sometimes following dysentery, has been described by Gerhardt in two cases of bronchiectasis, and is regarded by him as secondary to the bronchial trouble.

We should also mention the skeletal changes, not limited to this
disease, described by P. Marie and by Souza-Leite under the name of Hypertrophic Pulmonary Osteo-arthritis, and previously noted by Bamberger. In extreme cases there may occur, in addition to the usual clubbing of the finger-ends, an enlargement of the joint ends of the phalanges and metacarpals, of the long bones of the arm, and even of the vertebrae. Similar changes are also traced in the bones of the lower limb [vide vol. iii. p. 153].

Bamberger believes that the changes in bronchiectasis constitute a separate variety distinguished by the painful swelling of the epiphysis, and by the condensation occurring in its spongy substance as well as in its shell of hard bone.

**General and clinical causation.**—The insidious beginnings and the chronic course of bronchiectasis are not favourable to a study of its causes. Statistics of the disease at various ages can only deal with approximations. Lebert, in a series of 83 cases, found 47 per cent occurring before, and 53 per cent occurring after the age of 40:

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The congenital dilatations are exceedingly rare.

The male sex is more often affected than the female, according to Trojanowski and Bamberger; but other authors (Biermer and Willigk) have traced no difference. Occupation does not influence the production of the disease in any direct way, though it may act indirectly by setting up pulmonary and bronchial changes favouring a dilatation. Depressing circumstances of all kinds might also have an indirect effect.

**Clinical antecedents.**—We have no proof that the change ever arises spontaneously during extra-uterine life. In children we are able to trace its acute form to bronchitis. Fatal cases of this kind furnish us with the only direct evidence in favour of a definite causation from acute inflammatory disease; but clinical observations, although less conclusive, lend their support to the same view. When not traceable to an acute attack, dilatation is probably secondary to some chronic bronchial or pulmonary affection, and the precise time of its onset becomes difficult to determine.

As regards the immediate etiological factors Lebert's results are probably trustworthy. In a quarter of his series there had been previous emphysema; in another quarter an acute pleurisy or an acute pneumonia had preceded the disease; and in a large number the history was one of long-continued bronchitis with intercurrent acute attacks (Wilson Fox). Thus, bronchitis in all its forms, but especially when complicated with spasmodic cough, as in whooping-cough (Laennec) and in asthma (Hyde Salter), contributes a well-marked etiological group; pulmonary diseases,
whether acute or chronic, rarefying or condensing; forming a second group; and pleuritic affections a third. A fourth group is that in which a temporary or permanent narrowing of a large bronchus, as by an aneurysm, has led to increased strain or to accumulations within its subdivisions.

The relation which the bronchial affection may bear to tuberculous disease has been much discussed. Some, including Rokitansky, have regarded the two diseases as almost incompatible, and as mutually protective. Nevertheless, true bronchiectasis may occur in the subjects of chronic tuberculous disease; for instance, at the base of a lung with an indurated apex. And, conversely, sufferers from chronic bronchiectasis may end in tuberculosis, though this is rare.

Wilson Fox suspected that the fibrotic induration around the tubes was probably tuberculous in its origin; the other tuberculous deposits in the same lungs having been slight and obsolescent: but this opinion does not appear to have had the support of any direct evidence.

Biermer, who quotes Trojanowski as reporting tuberculosis in 21 out of a series of 68 cases, could find only 3 in his own collection of cases. As pointed out by Wilson Fox, discrepancies of this magnitude can only be explained on the score of some confusion between tuberculous lesions and those due to bronchiectasis.

Pathological etiology.—The history of the subject is a record of hypotheses as varied as the associated intrathoracic conditions; but they may be briefly classified as attempting to identify the causation (1) with changes limited to the tubes themselves, (2) with changes in the pulmonary tissue, (3) with changes in the pleura, or lastly (4) with a combination of the bronchial, pulmonary and pleural changes.

Some cases carry their own explanation: cicatricial stricture, lateral pressure from aneurysms or morbid growths, internal obstruction due to tumours, and particularly the impaction of foreign bodies are occasional causes of bronchiectasis; but those needing elucidation form a much larger group.

Laennec regarded the dilatation as due to an accumulation of mucus. André accepted this view only for the bead-like form, and attributed the other dilatations to a process of hypertrophy analogous to that of other hollow organs; this was also in part the view of Louis. Rokitansky, and subsequently Hasse, assumed a stenosis of the larger and an obliteration of the smaller bronchi, with compensatory dilatations elsewhere. Stokes and Williams traced the production of dilatation, under stress of cough or of accumulating secretion, to impairment of elasticity and of muscular contractility by inflammation. Atrophy of the bronchial muscles has been described by Bamberger, by Trojanowski, by Lebert, and, as a primary and probably constitutional defect, by Sir T. Grainger. Stewart. Lebert also suggested that dilatation might be due to atony dependent upon defective innervation. Various other pathologists (Beau, Maissiat, and Mendelsssohn) have insisted on the share taken by cough in the production of dilatation.

Wilson Fox considered all forms, except those secondary to a con-
the view that bronchiectasis is a progressive deterioration due to an innate local delicacy, independently of disease.

In disease, mechanical factors arise which are entirely foreign to the natural play of the organ, and which do not necessarily seek out the weakest part. To these belong, within the tubes themselves, an accumulation of mucus and the antecedent or the resulting degenerative changes in the bronchial wall.

A second influence is that of changes induced in the lung tissue. In a rather large proportion of cases bronchiectasis is accompanied by more or less emphysema. Much of this is clearly a result rather than a cause, since the ordinary vesicular emphysema does not carry with it any accessory bronchiectasis.

Another frequent accompaniment of bronchiectasis is pulmonary collapse. When occurring unevenly, at one side of a bronchial tube, this may act as one of the agents of dilatation. Not only in advanced bronchiectasis do we often observe a proportionate amount of condensing fibrosis of the lung, but in any recent dilatation, such as that witnessed in the infant after bronchitis or whooping-cough, the incipient bronchial bulgings occur side by side with considerable lobular collapse.

A further set of structural changes contributing, in a large proportion of the cases, to faulty allotment in space, are those of the pulmonary stroma, which includes the subpleural, the perilobular, and the interlobular systems.

As to the general mechanism of the dilatation we must again look for some elementary factor common to all varieties; and this we find in "obstruction," understood in the broadest sense of the word.

In the alimentary tract and in most animal tubes the obstruction is invariably situated forward, beyond the dilating segment. In the bronchial tract no such local restriction obtains. Neither is the nature of the obstruction necessarily limited to stenosis or to impaction. Owing to the alternating direction of the respiratory air-currents, an obstruction may lead to dilatation either on its proximal or on its distal side. Again, the dilating force is not usually, as in other tubes, the pressure of an accumulation within the dilating bronchus. This mechanism may occur in the bronchial system: an instance in point is the thin-walled saculation, completely filled with stiff gelatinous mucus, sometimes found beyond a bronchial stenosis. But much more often the obstruction has its seat on the distal side of the dilatation and is not a bronchial stenosis, but a terminal occlusion of a respiratory district of the lung; and the dilating force, far from being exclusively due to the pressure of an internal accumulation, is then applied to the outside of the tube; it is an aspiring, not a forcing pressure.

If we bear these elementary data in mind we shall find that the details of the problem work out. Thus, whereas in the normal state each pulmonary constituent preserves its relative position and its allotted space, the local failure of any individual constituent to perform its
respiratory function would interfere with the perfect adjustment of other parts during the phases of respiration. How readily bronchiectasis might result from this disturbance will be seen from a consideration of the forces which normally protect the weaker non-cartilaginous tubes against the dilating influences of atmospheric pressure. The elasticity proper to the inflated pulmonary tissue through which they pass tends to widen them; but this tendency is counteracted by the inspiratory elongation of the lung, and probably never goes farther in health than to ensure their patency, thus acting in the depth of the lung in lieu of a cartilaginous armature. On the other hand, both during inspiration and during expiration, the small tubes receive lateral support from their closely fitting environments. Let this support be withdrawn at any one spot by the persistent inspiratory inactivity of one of the adjacent lobules, even though this were merely a delay in the fulfilment of inspiratory inflation, then the imperfectly resisted intrabronchial pressure would gradually bulge out the yielding wall into the space rendered available, and thus establish the first stage of a progressive dilatation. Or, to put the matter more clearly, the inspiratory traction made by the chest wall, if it should fail to expand an obstructed lobule, might be transmitted to the delicate air-tube adjoining the latter, and might dilate it.

Owing to the solidarity existing between all parts of the lung, this encroachment of bronchial space into the vacated pulmonary space may occur at a distance from the original collapse. The same mechanism might therefore be concerned in some measure in the production of almost every variety of bronchiectasis. Its more strictly local operation is probably alone concerned in the early stages of the affection when the pulmonary tissue is still free from induration. In some instances bronchiectasis remains permanently uncomplicated with any pulmonary fibrosis, or with any peribronchial thickening. It is in these cases that the bronchial membrane preserves its delicate and transparent thinness. The plug of semi-gelatinous mucus which sometimes fills simple dilatations of this kind in the midst of soft spongy lung tissue suggests the idea that the mucus itself was originally the obstacle to the free inflation of the collateral lobules, whilst its accumulation eventually assisted in producing the distension.

The progressive increase in the dilatation may conceivably be brought about by the various mechanisms assumed by the so-called inspiratory and expiratory hypotheses; although much that has been advanced in connection with them is lacking in strict proof. Thus:—

(i) The inspiratory hypothesis of Laennec asserts that the abnormal inspiratory effort preceding cough throws damaging stress upon the weakened parietes of the bronchial tube. In the diagram (Fig. 4) which illustrates this supposed agency, if we imagine the shaded zone to remain unexpanded, the arrows would represent the inspiratory traction thus transferred from the alveolar to the bronchial walls.

The same explanation has been applied to the condition which may result from a proximally situated stenosis, when the impeded removal of
the products of catarrh from the terminal districts has led to an irregular lobular collapse with consequent disturbance of the balance of pressures.

(ii.) *The expiratory hypothesis* has also been pressed into the service of bronchiectasis as well as of emphysema. Were it not that one of the chief functions of man in earning his bread by manual labour is the performance of muscular strain with closed glottis, and that his organs are specially constructed for that purpose, the wonder would be that the prevalence of bronchiectasis and emphysema is not universal.

As a fact, nothing gives way within our visceral cavities under the high pressures due to muscular strain so long as every part is sound and works true. The extent to which we are dependent for this immunity upon a perfect distribution of pressures is illustrated by some of the delicate valvular membranes of the heart which could not perennially resist the stress to which they are exposed, were not the pressure exerted upon one of the two surfaces neutralised by equivalent pressure or support on the other. So must it be also with the delicate bronchial membranes. The range of pressures to which they are exposed is not so great, but their risk is multiplied by the number of their subordinate districts. A loss of the even balance between the intra- and the extrabronchial pressure occasioned by imperfect inflation of any of the latter might in delicate and predisposed subjects cause the bronchial wall to yield, and to suffer progressive dilatation.
Cough is a special instance of muscular stress; it is often complicated by the mechanical influence of the secretion which excites it. The diagram (Fig. 5) illustrating the mechanical theory of expiratory pressure will also serve to explain this point.

The cough which may be powerless to dislodge and evacuate the contents may yet propel some of them far enough to cut off the dilated chamber from the main bronchial channel. The moment represented is that of the explosive expiration, when the air accumulated under high pressure leaves the chest without any further hindrance. Alone in the dilated tube the pressure, indicated by the curved arrows, will remain at that moment nearly as high as during the period of closure of the glottis; and its dilating effect is but feebly counteracted by the released elasticity of the immediately surrounding lung tissue. Slowly, with the ensuing inspiration, the plug may be sucked in again; and this suction is the most likely explanation of the long-drawn, semi-musical, or croaking rhonchi and râles of bronchiectasis.

The practical results of a recurring valvular obstruction of this kind would be not only a continued fulness of the dilatation, whilst the surrounding tissues are being relieved of much of their air, but a maintenance within it of the highest air-pressure at the time when the air-pressure in its vicinity is at its minimum. Neither should we lose sight
of the possible injection into the tributary bronchioles and lymphatics of some of the bronchiectatic contents.

Most cases may begin and progress after the mode suggested; but, except in fatal cases of bronchitis and whooping-cough in children where these etiological relations are well displayed, an opportunity of examining the lung at this stage is not often afforded; and ulterior changes of a very different kind usually obscure more or less completely, the original mechanisms.

The influence of catarrh seems entitled to be regarded, as it has been by most writers since Laennec, as the chief and earliest etiological factor of bronchiectasis. The inflammatory softening and weakening of the bronchial wall, the changes in its muscular and fibrous coats, whether in the direction of atrophy or of overgrowth, are all possible accessory agents; but the special action of catarrh consists in the mechanical plugging of bronchioles. When a bronchiole becomes occluded the amount and the pressure of the air within its district are rapidly altered, and the balance of pressures will be disturbed to the special detriment of the tube from which the bronchiole sprang. If the pressure can be speedily readjusted by collateral expansion in the vicinity any strain or dilatation thus induced will be corrected. Failing this adjustment, however, the existing catarrh will aggravate the dilatation by a tendency to accumulation and by the impairment of the respiratory mechanisms of relief.

The successive obliteration by catarrh of many tributary bronchioles is probably the mode of extension of bronchiectasis. The greater the stretching of the dilated bronchial membrane and the accumulation within it, so much the greater will be the number of collateral bronchioles obliterated by stretching or by plugging, and so much the greater the extent of the resulting atelectasis.

A direct influence aiding the dilatation is that of any impairment of the muscular coat, whether in its structure, as in the atrophic fibrosis described by Lebert, or the simple atrophy of Grainger Stewart; or in its function, as in atony from defective innervation, or from insensitiveness of the mucous membrane.

Indirectly, the process of dilatation might be favoured, as in pertussis and in acute bronchitis, by the opposite condition of bronchial spasm, since this would lead to a narrowing and to a more ready plugging of the smaller tubes.

The influence of interstitial pneumonia and fibrosis.—In whatever way it may have arisen, a saculation of a small bronchus is fatally exposed to an accumulation of secretion during periods of catarrh, and to irritation not only within its own terminal divisions, but probably, by overflow and by inhalation, in collateral lobules also. This is the beginning of an interstitial pneumonia, the ultimate result of which may be a conversion of the pulmonary substance into structureless fibrous tissue. The loss of expansive power is progressive, and the shrinking of the chronically inflamed parenchyma favours the encroachment of the saculation; whilst the implication of the lymphatics of the lobule causes an extension of the
changes along the perilobular system. In this way the pulmonary degeneration is promoted along two lines, by intralobular and by perilobular agencies. How far it may extend will depend upon the varying ability of the remaining pulmonary tissue by its increased expansion to replace some of that which has atrophied. Dense adhesions would largely interfere with this compensatory process.

The influence of pleuro-pneumonic fibrosis.—The close relationship existing between the pleura and subjacent stroma and the lymphatic system of the lung explains the influence which agglutination of the pleural surface exercises on the course of the interstitial pneumonia, and on the etiology of bronchiectasis. Extensive pleuritic thickening at the base, with obliteration of the groove and agglutination of the surface of the diaphragm, cripples the lung. The respiratory function of the base is almost entirely lost, or can be carried on only by considerable mechanical effort on the part of the diaphragm, and of the inspiratory muscles; an effort which must tell on the pulmonary tissue as a constantly recurring and irritating traction. The lymphatic circulation may also be impeded. The result is usually a considerable shrinking of the side affected, and a compensatory hypertrophy of the sound lung, with great distension of that side of the thorax.

The process which has just been sketched is essentially that originally described by Corrigan under the name of “cirrhosis of the lung.”

The influence of stenosis.—Dilatations are by no means the invariable result of bronchial stenosis. When a bronchiectasis occurs beyond the stenosis its mechanism is generally held to be analogous to that of emphysema from a partial obstruction of tubes, which allows a slow entrance, but unduly delays the escape of air. Syphilis, being a well-known cause of bronchial stricture, should be allotted a place among the recognised factors of bronchiectasis. It is not improbable that in some cases the occurrence of a late ulceration of the dilated tubes may be due to the same influence.

Hoffmann believes that sufficient attention has hardly been paid to the probably frequent origin of bronchiectasis from _inhalation of solid particles_, and he refers to the experiments of Cohn which show that dilatation occurs not beyond but at the seat of impaction, around the impacted foreign body.

Lichtenheim’s experiments go to prove that total closure of a bronchial tube leads within twenty-four hours to a complete atelectasis of the pulmonary district, with purulent accumulation within the tubes. After several weeks the latter become more or less dilated, the surrounding tissue being completely compressed by the distended bronchi, or expanded by collateral emphysema.

Beyond any valvular obstruction micro-organisms, which easily penetrate through the stenosis, may set up fermentation, and the secondary results of putrid decomposition will follow.

Symptoms.—The severity of the disease varies greatly in different individuals and at different stages in each. Its course and its symptoms
are largely determined—(a) by the mechanical factors, such as induration or persisting elasticity of the surrounding tissue, position of the dilatation, its single or multiple character; (b) by constitutional factors special to the individual or to phases of his general health; and (c) by climatic and atmospheric factors, including not only temperature and humidity, but also purity of air, in the sense of relative freedom from septic germs.

It has already been stated that in exceptional instances bronchiectasis may be latent for some time after its commencement: in a few cases also there may be periods of quiescence during which it might pass unobserved. These are the milder forms, of a catarrhal and emphysematous type—non-indurative, non-Septic, non-ulcerative, progressing but slowly, and compatible with relative longevity. All cases are liable to exacerbations in the symptoms, to occasional or periodical increase in the expectoration, to recurring intervals of fetidity of the sputum, and to intercurrent attacks of general bronchitis or catarrh.

*Constitutional symptoms.*—For long periods the flow of expectoration, sometimes even when fetid, may proceed without making any obvious impression upon the general nutrition or functions; but these are gradually involved as the diminution of respiratory surface and consequent loss of energy advance; and ultimately the system is contaminated by the septic matters inhaled, absorbed, and swallowed. The constitutional symptoms set in at different stages, and at first may not be permanent, but coincide with transient periods of factor of the sputum. In the worst forms these deteriorations are lasting. Sooner or later the pulse and respiration become permanently accelerated, and the temperature moderately hectic, or at the least remittent, with an evening rise to 101° or 102°, and in a few cases with associated night-sweats. Diarrhoea may be among the septic symptoms, and sometimes vomiting also. Vomiting as a mechanical result of cough is not so common in bronchial dilatation as in phthisis.

Failure of cardiac energy lies at the root of the final cachexia. In addition to the previous lividity and cyanosis edema supervenes, and the patient becomes a bed-riden invalid. At this stage, or prior to it, intercurrent albuminuria may be observed; or in association with lardaceous disease it may become permanent. Various complications may cut short the gradual process of exhaustion; low pneumonia, putrid bronchitis and gangrene, septicemia or pyaemia, cardiac or renal disease, and cerebral abscess are among the most common. In the more favourable cases, especially when helped by the advantage of climate and treatment, the sufferers may live with their trouble for years, and die from other causes. Those who reach a relatively mature age are more and more exposed to catarrh and emphysema with their attendant symptoms, and the disease, whether directly or through its complications, is usually responsible for death.

*Pulmonary symptoms.*—Under this heading we must briefly review the changes in the respiratory function—the cough, the expectoration, and haemoptysis.
Dyspnoea.—There is often a cardiac element in the dyspnoea observed in bronchiectasis. Much cardiac and nervous depression is induced at times by septic absorption from the bronchial tubes and through the breath, especially in ulcerative cases. As a rule, during the major part of the clinical history the dyspnoea is not excessive; but it varies much with the degree of emphysema or of fibrosis, and with the amount of intercurrent catarrh. In the ultimate stages dyspnoea becomes a prominent feature.

Cough.—A leading peculiarity of the cough of uncomplicated bronchiectasis is its intermittence. It would seem as though the sacculated membranes lost their sensitiveness, and that cough were excited only when the tide of accumulation reaches the level of some healthier part of the bronchial tubes. It is often observed that for long periods, during which a patient preserves the posture which acts as a protection, no cough is set up; but change of position will bring on severe spasmodic cough and profuse expectoration. The severity of the cough and its paroxysmal character are explained by the irritating quality of the secretion which has to be forwarded through the sensitive upper passages; and also by the difficulty, or impossibility in some cases, of complete relief on account of the position of the sacculations. Whereas a partial emptying of the surplus of the bronchial contents is comparatively easy, nothing short of an inversion of the patient could empty some of the ultimate dilatations, especially when surrounded by fibrous tissue. The creasote inhalation method introduced by Dr. Chaplin has demonstrated that the factor of this residual material is much in excess of that of the tidal output, a point which should be borne in mind as of the utmost importance in treatment.

Expectoration.—The sputum in bronchiectasis varies considerably in amount and in character. Sometimes it remains sweet and almost purely mucoous for long periods; more usually it is muco-purulent throughout. In most cases it becomes fetid at times; or this may be the habitual condition. Very often, when ulceration has taken place or after severe paroxysmal cough, it is slightly blood-stained.

A third of a pint or half a pint is not an unusual daily quantity; but this amount is often much exceeded. The way in which the expectoration pours out of the mouth in some cases is almost distinctive, though the same peculiarity may be observed in phthisis. At intervals the expectoration may be much lessened or completely absent. Complications, especially bronchitis or pneumonia, commonly reduce the amount.

The sediment deposited by the expectoration, which may separate into an upper frothy mucus, and a lower puriform layer with an intervening watery layer; presents, besides bronchial epithelia, numerous pus-cells, granular débris, bacteria and vibriones, fatty acid crystals; and occasionally sarcoine, leptoethrix, Dittrich’s plugs, and Charcot-Leyden crystals. The presence of elastic fibre would be a proof of ulceration. The factor is apt to be great, but it is occasionally more marked in the breath than in the sputum.
Hæmoptysis was among the symptoms described by Kenney. Walshe failed to observe hæmoptysis except in the presence of mitral disease or of tubercle. Liebert observed hæmoptysis, of varying degrees but decidedly more marked than that which belongs to ordinary pneumonia, in one-sixth of his cases. Biermer reports one case of fatal hæmorrhage in non-tuberculous ulcerative bronchiectasis. Wilson Fox, who quotes these authors, refers to it as not being rare. It may occur early and independently of any ulceration. On the whole, it is to be regarded rather as a frequent complication than as an invariable symptom.

The respiratory symptoms vary with the degree of the pulmonary atrophy. Among them are to be noted frequency of breathing and dyspnœa on exertion, and, in unilateral cases, inability to lie on the sound side.

Physical examination of the chest.—Inspection.—There is no distinctive chest shape peculiar to bronchiectasis; and the thorax does not present the characteristics of phthisis, even though one side may be much retracted. Whatever amount of flattening may be present locally, this is compensated elsewhere by active thoracic expansion. The immaturity of the apex in the majority of cases and its compensatory expansion, coupled with the fulness of the neck, establish at first sight a distinction from the ordinary case of phthisis. Often, on the other hand, the deformity peculiar to emphysema may be more or less fully established. The unilateral character of the group of cases described by Corrigan as cirrhosis of the lung is usually made obvious by the cardiac displacement, and by the extreme disproportion between the size and the respiratory movements of the two sides of the chest. But in some unusual cases, owing to considerable encroachment of the sound lung across the middle line, the thorax on the side affected is much less collapsed than the lung which it contains. Cases of this kind are deceptive, and need, for an accurate determination of the size of the lung, a very careful percussion of the boundaries of the cardiac dulness. I have described a case of this sort. This cirrhosis of the lung without thoracic deformity is much less readily distinguished from phthisis or from chronic bronchitis than the usual form.

Among the bilateral cases the emphysematous variety is to be diagnosed from phthisis, on mere inspection, by the dusky and congested complexion, the prominent veins and deeply coloured lips, the high, deep, and broad chest, and the relatively good nutrition.

In the remaining groups the diagnosis may be assisted by a knowledge of the following points:—(a) A solitary bronchiectatic lesion is seldom localised at the apex; this is the customary site for the tuberculous lesion. (b) The supraclavicular area is usually not implicated in any dulness due to bronchiectasis; it is invariably implicated in the apex dulness of phthisis. (c) In phthisis, as pointed out by Stokes, consolidation precedes, excavation follows: in bronchiectasis this is otherwise. And again, extension of the excavation is peculiar to phthisis (Stokes), whilst a stationary size belongs to bronchial dilatation (Walshe). (d) The
almost daily alternations between the signs of fulness and those of vacuity greatly help the diagnosis of sacculation. This peculiarity is usually absent or inconstant in excavating phthisis. (e) The normal site for tuberculous disease is the apex; it hardly ever involves the base. The site of predilection for bronchial dilatation is the base; but bronchiectasis also favours the middle and lower third of the back and may affect various other situations without any hitherto ascertained regularity of order; it is specially uncommon in the district of the vertical bronchi ascending to the apex. (f) It is unusual in phthisis for multiple excavations to form in the same lung with the intervention of sound pulmonary substance, except in the situations described in the Goulstonian Lectures for 1882, and by Dr. J. Kingston Fowler in his Dictionary of Medicine. Even these secondary deposits are commonly almost continuous with the upper zone of disease. In multiple bronchiectasis a truly sporadic arrangement is the rule. (g) Unilateral indurative tuberculous phthisis invariably excavates and condenses the apex first, even if later it should extend downwards. The fibroid change associated with bronchiectasis originates as a rule at the base and spreads upwards. (h) The displacement of the heart towards the diseased side of the chest in the usual cases of unilateral phthisis follows an oblique direction upwards; a horizontal displacement is exceptional and suggests some complicating basic pleural factor. In unilateral bronchiectasis the displacement is, practically speaking, always horizontal; not only by reason of the basic origin of the disease, but largely also owing to the lowering of the diaphragm on the sound side, with extension of the cardiac beat into the epigastric notch.

Attention to these general guides may often prove of greater value than a close search for points of difference in the auscultatory and percussive sounds.

Percussion in advanced cases may yield different results in the same chest at brief intervals of time, according to the amount of retained secretion; and this variability is perhaps the most distinctive feature obtainable by the method. If in a chest otherwise resonant patches of dulness be found scattered in the middle and lower third, and particularly over the back, and if some of them yield a cracked-pot sound, a strong suspicion of bronchiectasis will arise. The high-pitched, the tympanitic, the splashing, and other varieties of percussion note which have been described cannot be expected in every instance. Much emphysema may almost preclude a diagnosis by percussion alone; although with a previous knowledge of the existence of sacculations their site could in most cases be made out by an expert percussor. The strong element of dulness in the fibroid variety of the disease, coupled with the boxy note obtained over the cavities when empty, is a much more definite guide; although the diagnosis from a basic cavity of tuberculous origin would still have to be made.

Auscultation, although not always capable of establishing a diagnosis between slight bronchiectasis and bronchial catarrh, seldom fails to
identify advanced dilatation, from a joint observation of the respiratory sounds and of the râles.

As regards the respiratory sounds, the peculiarity of the emphysematous variety of bronchiectasis is the intimated blending of the tubular with the vesicular breath sounds; the fibrotic variety is distinguished by the local absence of the latter.

The râles occurring in small dilatations, and in those which are mainly cylindrical, do not differ from ordinary catarrhal râles of medium and of large size. A distinctive character belongs to those produced in the sacculations. The sound, which is best described as "croaking," is partly due to the valvular action of the viscid and confluent secretion, and partly to the free communication and continuity subsisting between the sacculations and the corresponding bronchus. The undiminished length of the latter, and the branches which open into it above the terminal sac, are probably additional factors. An explanation of the mode of production of this sound is suggested above in connection with Fig. 5. The croaking sound is most distinctly produced in sacculations surrounded with more or less spongy tissue. In the fibrotic variety the solid medium through which it is conducted to the ear imparts to it a more metallic character.

It is unnecessary to dwell upon the common catarrhal sounds, the sibilis and the rhonchi, which may spread over the lung as a result of general bronchitis. They may complicate the diagnosis by veiling to a certain extent the diagnostic sounds which have been described, although they seldom mask them entirely.

The voice sounds sometimes supply definite data. Bronchophony and eso-bronchophony are yielded by the extensive and multiple sacculations of a partly cirrhotic lung, and sometimes by those not surrounded with fibrous tissue, if sufficiently large and superficial. Hollowness of the voice sound would, however, disappear if the cavity were to fill completely. The vocal fremitus varies considerably in different cases, the pleura being unaltered in some, in others greatly thickened.

Diagnosis.—The diseases most likely to be mistaken for bronchiectasis are the various forms of bronchitis and phthisis. Less commonly the difficulty may be to distinguish it from emphysema, pulmonary gangrene, and cancer.

When originating in a general bronchitis, dilatation, in its earlier stages, can only be inferred. Subsequently factor of the sputum necessitates a diagnosis from fetid bronchitis or bronchorrhoea; and, apart from any previous knowledge of the case, the distinction may be extremely difficult if a general catarrh should coexist. In the absence of the latter, dilatation would be known by the localisation of the large râles in the situations which present some alteration of the percussion note; and the same observation would also be a help in the more complicated condition. Again, the mode of the expectoration, even more than the nature of it, might throw light on the case; although in fetid bronchorrhoea the expulsion of the bronchial contents is often sudden and paroxysmal.
Pulmonary gangrene, occurring in aged or broken-down subjects and preceded by a history of chronic bronchial catarrh, would suggest bronchiectasis culminating in ulceration. Most commonly the onset of pulmonary gangrene is sudden and marked by extreme prostration; that of gangrenous ulceration of a bronchiectasis is gradual. As pulmonary tissue is expectorated in both cases, our guides must be the clinical data and the clinical history. But commonly in bronchiectasis a gangrenous odour occurs apart from any tissue necrosis; and a fruitless search for elastic fibre would strengthen any direct evidence of bronchiectasis otherwise obtained, and any negative evidence as to the existence of broncho-pneumonic or tuberculous processes such as lead to gangrene.

The intra-bronchial ulceration of an empyema may closely simulate bronchiectasis. Its presence will be sufficiently indicated by the history of an absence or insignificant amount of expectoration prior to the bursting; and of the considerable relief given by the latter to the cough, dyspnoea, pain, and thoracic deformity. The expectoration of an empyema is usually distinguishable at first sight, by its freedom from mucus, from that of bronchial dilatation. According to Biermer, it contains crystals of cholesterol and of haematoidin. In any special case a physical examination of the chest would probably remove any lingering doubt.

Prior to the discovery of Koch's bacillus the diagnosis from phthisis had to be made almost exclusively from physical signs, and was often very difficult for persons unfamiliar with the physiognomy of bronchiectasis. A microscopical examination of the sputum now decides the question. Nevertheless, the other elements of diagnosis—(a) the clinical history, (b) the general clinical state and aspect, and (c) the physical signs—are too important to be neglected.

(a) In most cases phthisis can be traced back to characteristic beginnings, the constitutional effects of the invasion being out of proportion to the pulmonary symptoms existing at that time. This is not the history of bronchiectasis, which begins with a definite bronchial affection, or with a pneumonia or a pleurisy; the worse constitutional symptoms being relegated to the late stages. Again, when the patient's affection begins with a profuse hæmoptysis the probability of its tuberculous nature is great.

Moreover, the duration and the progress of the two diseases are strikingly different. Cough and expectoration of many years' standing, in a subject not markedly marasmic, would not be features of the common phthisis; though we should not forget that unilateral phthisis may, and often does, run an exceedingly protracted and mild course. In such a case the signs would be unmistakable and strictly apical, and therefore unlike those of bronchiectasis which, when single, hardly ever implicates the pulmonary summit.

(b) Between ordinary pulmonary tuberculosis and ordinary bronchiectasis a very marked contrast in the general clinical appearances is at once
perceptible. In the ultimate stage of pulmonary consumption there is no difficulty in the diagnosis; the patient carries it written large in every feature. At a rather earlier period in the complaint, when doubt might be possible, the same peculiarities are apparent, although not yet so manifest as to strike the superficial observer. They are briefly these—wasting of the subcutaneous fat in general, and in particular of the fat of the orbit and of the cheek; wasting of the muscles; visible loss of energy; pronounced anaemia, in the strict sense of the word, namely, reduction in the total amount of the blood, the patient being bloodless and withered. These are not features of bronchial dilatation, uncomplicated with tubercle, at a like interval after the beginning of the affection: emaciation usually exists, but it is not extreme; there may be slight anaemia also, but it does not confer the characteristic wan look of phthisis. The hollow orbit, with undue exposure of the sclerotic, the sunken cheek with projecting malar eminence, and the thin, drawn lip are all conspicuously absent. Instead of these bronchiectasis often presents outward peculiarities of its own; a certain fulness of the eye, of the lip, and of the features, and a slight duskyish of the complexion suggestive of congestion rather than of anaemia: and the veins, the jugulars in particular, are commonly conspicuous, if not turgid. On analysis these peculiarities will be found correlated with the state of fulness of the right side of the heart, which in advanced phthisis is never surcharged, in spite of the great obstacle to the pulmonary circulation. In short, the bulk of the blood is not reduced in proportion to the pulmonary destruction, as is the case in phthisis. For the same reason also the depressed and devitalised aspect peculiar to phthisis is not noticed in this disease.

Another striking peculiarity is the unusually bulbous expansion of the finger-tips, associated with a very marked incurvation of the nails. In phthisis the nails are aduncate, but the finger-ends are seldom much clubbed; nay, the pulp of the finger is often wasted.

Prognosis.—The spontaneous cure of acute bronchiectasis, such as it occurs in the growing lung of infants, cannot be expected at a later age; and a restoration of the damaged lung is impossible. In rare instances, where the dilatation is single, and where it is no longer the seat of catarrh, as in the exceptional case of the cicatrical closure of its bronchus higher up, the disease may become obsolete. Lebert quotes a case of Bamberger's, in which the formation of an external fistula eventuated in a cure; and a similar result might be hoped for from the surgical treatment of a solitary dilatation. In the great majority of chronic cases, so long as the original conditions persist, the disease, if left to itself, is inevitably progressive; and therefore less likely as time goes on to be permanently relieved. The most favourable achievement to be gained by treatment is often no more than a relative quiescence of the trouble; or a reduction in the rate of a progressive destruction of the lung.

As regards duration of life, the great diversity in the kind, degree, and multiplicity of the lesions, and of their bronchial, pulmonary, and
pleural complications, must establish a wide difference between the chances of this some idea is given by the figures obtained by Lebert in a series of fifty-two cases.

The period of survival was:

- Of one year: 7.7 per cent
- Of one to two years: 30.7 per cent
- Of three to five years: 15.5 per cent
- Of six to ten years: 25.0 per cent

Apart from all other difficulties, an exact determination of the extent and number of the lesions is so unlikely to be attained by physical examination, that the physician's forecast in the individual case must be based on very broad considerations: such as the age, temperament, antecedents, energy, nutrition, and general circumstances of the patient; the unilateral or bilateral character, and the cirrhotic, emphysematous, or stenotic type of the affection; the presence or absence of heart, kidney, or liver disease; the present and the previous state of the expectoration, and the effects of treatment on the catarrh.

Often enough, when all has been taken into account, great uncertainty must still surround the prognosis, and it will be wise not to venture upon too precise a statement of the probabilities. In the future much more may be expected from an improved diagnosis, and from the earlier adoption of improved preventive, palliative, and curative measures, than from surgical interference, which is not likely to prove more successful than in the past.

The worst prognosis will probably always belong to the bilateral cases and to the unilateral cirrhotic variety, especially when associated with some defect of the other lung or pleura. Hæmorrhage is occasionally a fatal complication; it is apt to be profuse in cases of valvular disease or of secondary cardiac dilatation. The occurrence of perforation and pyopneumothorax, or of ulceration with the attendant dangers of gangrene, of putrid bronchitis, of pyæmia, and of septicæmia, would justify a grave prognosis. Mere factor of the expectoration is not in itself an alarming sign.

In those most favoured cases which remain free from all serious complications life may not be greatly shortened.

Treatment.—The acute bronchial dilatation of early childhood, depending upon temporary impairment of the expansion of lobules, and of the pulmonary and bronchial elasticity, is capable of spontaneous recovery. The general principles on which such cases should be conducted are too well known to need comment.

Inveterate bronchiectasis, though not, strictly speaking, curable, is often susceptible of considerable amelioration. The extent to which positive results may be hoped for must largely depend upon the extent of the bronchial lesions, and especially upon the condition of the sur-
rounding tissue; the most unpromising cases being those in which ulceration or considerable fibrosis has already taken place.

In addition to the therapeutic measures specially intended for the pulmonary condition, we shall consider those meant for the relief of complications and for the improvement of the constitutional state.

The constitutional treatment, an essential adjunct of the pulmonary treatment, need not detain us long, since its climatic and hygienic aspects are included in the account to be given of the latter. It cannot be regarded as curative, nor even as being aimed at the cause of the affection; but it undoubtedly promotes the patient's chances and the results to be obtained from symptomatic treatment. The only instances in which it might claim to be in any sense specific are those in which the disease has been traced to syphilis, and in which mercury, a drug possessing also general advantages as an antiseptic, should have a trial. Iron, quinine, and cod-liver oil perseveringly administered, with intervals of rest and interludes of hepatic treatment, are still, so far as we know, the best means to the end of strengthening both fibre and function. Syrup of the iodide of iron in liberal doses, or the hypophosphites of calcium, of sodium, and of iron also freely administered, are remedies specially adapted to counteract the exhausting effect of catarrh on the serous and glandular elements. A liberal, varied and nutritious diet, and a moderate allowance of burgundy or of port wine are indicated. Much general tonic effect may also be obtained by systematic treatment of the skin and by salt-water baths—subjects to be discussed presently. Neither should we lose sight, in cases showing a tendency to venous stasis and to cardiac dilatation, of the great value of derivative, alterative, and mildly hepatic treatment. Much might be effected in early stages by hygienic and medicinal measures of this kind; but too often the opportunity of recommending them is not afforded until it is almost too late for their successful employment.

The treatment of complications.—As in other chronic affections, medical advice may at first be called in for the treatment of aggravated symptoms, of complications, and of emergencies. Among the latter, haemorrhage—fortunately rare in its worst form, that of ulcerative perforation of an arterial branch—calls for immediate action, and must be treated on the usual principle of reduction of blood-pressure, by subcutaneous injections of morphia, by calomel by the mouth, and by an enema of glycerine (not of a large bulk of fluid); all of which should be administered as soon as possible.

The febrile exacerbations of the bronchial catarrh, the complications of pneumonia and of pleurisy, the severe symptoms attendant upon absorption of septic material, and the occurrence of ulceration, with threatenings of gangrene, will need measures adapted to each event. In all of them a supporting plan of treatment will be necessary, and, in those last mentioned, stimulants, both medicinal and alcoholic, must be freely administered.

The special treatment of the respiratory organs should be guided by
the following indications: (i.) the emptying of the cavities; (ii.) the relief of the factor; (iii.) the reduction of the catarrh; (iv.) the protection of the membrane from further irritation; (v.) the diminution of the size of the dilatations and (vi.) the improvement of the respiratory function in general. Until recently these indications have been very imperfectly fulfilled. The methods employed have acted as palliatives, but their inability to check the progress of the worst cases has been one of the reproaches of medicine, and has led within recent years to a desperate resort to surgical measures, the hopelessness of which has now been made apparent; and indeed was almost foretold in the anatomical peculiarities of the affection.

As regards the emptying of the dilated tubes, sufferers often discover at an early stage the value of posture as a mechanical aid to the bronchial outflow. With the majority, lying down or turning to one side or to the other will bring on more or less cough and expectoration; but in others, when the dilatations are situated at the back, it is the change to the sitting posture which induces the paroxysm of cough. In this disease, even more than in phthisis, lowering the head, either over the edge of the bed or whilst standing, will allow the accumulated secretion to gravitate out of the acculations and into the receiver. Some patients are in the habit of practising this method of relief. Its regular employment should be suggested whenever no contra-indications exist. In the case of multiple bronchiectasis systematic treatment should also include, unless there be good reason to the contrary, the yet more effectual resort to an emetic; and it is well to administer, for two days prior to this, repeated doses of an expectorant mixture containing ipecacuanha, small doses of vinum antimonials, and iodide of potassium,—a mixture to be subsequently continued until a second emetic shall have been taken at an interval of a few days. The object is to wash out the stale secretion by a more abundant flow of watery mucus. Much will have been gained if this result can be secured.

For the relief of the factor two methods have hitherto been adopted alternately or combined: (a) the inhalation, and (b) the internal administration of deodorising and antiseptic agents.

(a) Inhalations as a rule fail to influence the bulk of the accumulations, though they may reach the uppermost layers. A noteworthy exception must be made in favour of those inhalations which set up cough and copious expectoration.

Theoretically, oxygen was expected to fulfil a double purpose, as an aid to respiration and as a disinfectant; but it has really proved of little service, partly perhaps because of its tendency to diminish rather than to increase the activity of the respiratory movements. Some relief may be obtained from the inhalation, from a jug, of vapour impregnated with thymol, eucalyptol, wood fir oil, or other antiseptic.

Inhalation may also be practised with the dry inhaler, through which air is drawn over a sponge or a quantity of cotton wool steeped in the
solution to be used. Since only those constituents are inhaled which are volatile at the ordinary temperatures, substances such as carbolic acid, creasote, tar, terebene, and others can be used fairly concentrated. Iodine can also be used with proper precautions.

Lastly, inhalation may be conducted on the principle of the spray. Steam sprays, at one time much in use, have their drawbacks, but in some respects are convenient: they may be made the vehicle of a great variety of medication. The complication of steam is avoided in the mechanical spray-producers which "atomise" the solutions to be inhaled, by forcing them through the minute orifice of the outlet with a jet of compressed air worked by an india-rubber hand-ball. In this case the solutions are not diluted by steam, and must be prescribed of an appropriate strength. The dripping and dampness inseparable from the steam are avoided; and the nozzle of the instrument can be introduced into the nose or mouth, thus almost ensuring actual inhalation of a large proportion of the remedies. The finest subdivision is obtained—as in Oppenheimer's "nebuliser"—by combining strong pressure with smallness of orifice. The latter condition unfortunately limits the supply of the medicated atmosphere.

(b) The internal administration of creasote, tar, terebene, the essential oils, the oleo-resins, and the balsams has long been in use. Only of late years, however, have the improvements in pharmaceutical detail enabled efficient doses of the more powerful of these agents to be taken with comfort. Copaiba, tar, and especially thymol, eucalyptol, guaiacol, and creasote, can be administered in the shape of capsules at frequent intervals throughout the day; and, by the persistent action kept up on the respiratory mucous membrane, may be of great benefit. Fifteen centigramme capsules of myrtol, taken every two hours throughout the day, are well spoken of in Germany; and are worthy of trial in cases in which none of the measures about to be described can be carried out.

The fault of most of these methods is their inadequacy; they do not deal with the evil at its chief seat in the depths of the lung. A new era in the prognosis of bronchiectasis has happily been opened up by the more thorough methods associated with the names of Dr. Vivian Poore, Sir T. Grainger Stewart, and Dr. Arnold Chaplin; these methods consist respectively in the internal administration of garlic, in the intralaryngeal injection of disinfecting solutions, and in the systematic inhalation of the vapour of coal-tar creasote.

(1) Dr. Poore's method is based upon the penetrating properties of some of the volatile constituents of garlic, and upon their stimulating and antiseptic as well as odoriferous virtues. Garlic probably acts as a general tonic as well as a local stimulant. Its local effect is produced at the surface of the mucous membrane by exhalation; but the fact that the smell of garlic is also given off by the skin suggests that the constitutional influence of the drug may be widespread and important.

The favourable results reported by Dr. Poore in his work on *Nervous Affections of the Hand and other Studies* were obtained from the continued
administration of sufficient garlic to render the odour permanent in the breath. In the cases to which he refers the original factor of the expectoration was replaced by a pungent smell reminding one of that of syringa. The discharge was greatly diminished; and a remarkable improvement took place in the health, in the strength, and in the weight of the patients. The treatment is generally well borne, and, if the remedy be taken with meals, patients submit to it without much inconvenience. A clove of garlic is chopped up and mixed with the beef-tea, or preferably enclosed in gelatine capsules. I have administered as much as eight capsules daily, each containing thirty grains of chopped garlic. An extract might also be used. Dr. Poore suggests that sulphide of allyl, which is contained in the essential oil of garlic, is probably the remedial agent. The oil of allyl has an exceedingly penetrating smell. It should be taken immediately after meals. I have prescribed it in three-minim capsules three times a day; but this dose is too large, and soon disagrees. Capsules containing half a minim of the oil will be found more convenient. The remarkable results obtained by this method are not limited to cases of bronchiectasis, but have also been obtained in phthisis. The chief theoretical objection to the treatment by garlic is that, whilst it provides for the disinfection, it does not ensure the complete clearance of the dilated bronchi, nor directly assist their contraction.

(2) Intratracheal injection, suggested years ago, and experimentally tried in animals, was first performed with the hypodermic syringe,—a valuable method in some cases, and was described by Dr. Sehrwald.

The idea was applied in a practical form to the treatment of pulmonary affections by Rosenberg, Colin Campbell, Jamieson, Downie, Byrom Bramwell, and by Sir T. Grainger Stewart, who treated with great success by the intralaryngeal method an inveterate case of fetid bronchiectasis in which all other measures had failed. The treatment consisted in injecting twice daily into the trachea (through the glottis) one drachm of a solution of 10 parts menthol and 2 parts guaiacol in 88 parts of olive oil. The injections were continued for a considerable time with benefit.

This method, which has now been fairly tried and seems likely to lead to important results, is the only one which aims at disinfecting the secretions in the lung by fluid admixture with the disinfectant, and at treating the mucous membrane locally by soluble applications. What proportion of the injection may reach the dilatations will depend upon circumstances not easily controlled, but chiefly upon the posture adopted by the patient at the time of the operation and afterwards. At any rate, the effect on the bronchial mucous membrane must be widespread and decided, and, with a systematic use of the treatment, would finally extend to that of the diseased mucous membrane also. The possibilities opened up by this therapeutic innovation are obviously great, and its applicability is not restricted to the disease under discussion, nor to the stated formula. Chronic bronchial catarrh, fetid or putrid bronchitis, and
bronchorrhea, especially of the purulent variety, are suited for its adoption.

Although in common with those who have tried this method I have wondered at the facility with which the pulmonary lymphatics dispose of the injected solution, we are left too much in the dark as to the destination of the latter. It is much if, by carefully directing the nozzle of the syringe and adjusting the patient's posture, we can ensure the treatment of one lung rather than of the other; but we are unable to control the injected fluid in its course down the tubes. In the majority of cases nothing but good happens. In tuberculous cases, however, there may be some misgiving as to the possible dissemination of the infection from the upper into the lower lobes. From personal observation I regard the use of the method in cases of phthisis with grave suspicion; and it has been my regret to witness, after its repeated use, the appearance of persistent rales, of bronchial engorgement, and of catarrhal pneumonia at both bases in a case which ended fatally.

(3) No objections of this sort can be urged against Dr. Arnold Chaplin's creasote method, which both theoretically and in its results is the only one hitherto claiming to be strictly rational and thoroughly adequate. Its principle is to obtain an amount of coughing sufficient to squeeze out every remnant of the noxious secretion, and to keep up local disinfection by inhalation for a sufficient time, and in sufficient strength, to enable the mucous membrane and the lung itself to be completely purified. These indications once fulfilled, nature will do the rest. Living in an atmosphere of the disinfectant would carry out an important part of the treatment; and Dr. Chaplin originally noted the tradition, which exists among workmen constantly employed in an atmosphere of creasote, that the fumes "clear the chest of phlegm," and confer an immunity from "asthma" and consumption. But in bronchiectasis the object is to bring about a complete expectoration of the bronchial contents; and with this view the creasote atmosphere has to be made almost intolerably strong, so that it can be inhaled for short periods only. This concentration of the vapour is the irksome side of the treatment; but any objections connected with the hardships of the method will, after a brief trial, be laid aside when the patients have experienced the remarkable relief afforded by its use. In addition to the intense cough, which has the advantage of leading to inhalations of the disinfecting agent proportionately deep, the discomforts are chiefly the irritating action upon the other mucous surfaces and the eyes, the strong smell which clings to the hair and clothing, and the diffusion of the smell into the surrounding space. So pervading is the odour that it may be complained of even within neighbouring houses, and it is desirable to provide an entirely separate inhalation chamber at some distance from the doors and windows of other buildings. The remaining difficulties are met by loosely plugging the nostrils with cotton wool, by wearing over the eyes watch-glasses framed in bandage or sticking-plaster, and by covering the garments and the head with oiled silk or mackintosh.
BRONCHIECTASIS

The inhalation chamber should be of small size, 6 or 7 feet wide by 8 feet high, and should be made as air-tight as possible, with cotton wool or tow, in order to obtain a dense creasote atmosphere. In vaporising the creasote proper care must be taken to prevent a conflagration. A fair-sized metallic evaporating dish is the best, and into this it is convenient to place some dry sand. Some more stable support than the common tripod should be used, and gas flames must be avoided.

At the first sittings the patient may with advantage enter the room before the spirit lamp is lighted under the dish containing the sand and creasote; but subsequently time may be saved by vaporising beforehand some of the creasote. The duration of the exposure is gradually increased from a quarter of an hour to an hour or more. The residual phlegm dislodged by the searching cough is exceedingly offensive; but the factor is partly covered by the strong creasote odour. The treatment, unless contra-indicated, is to be continued daily until little is coughed up in the chamber, and until no expectoration is brought up spontaneously the next morning. In an average case this will imply a treatment of from four to six weeks.

In itself the adventure is a valuable respiratory exercise. Whilst the cavities are being cleared and disinfected collateral expansion of the lung is induced by the cough, and the gradual contraction of the sacculations is promoted. A remarkable improvement takes place in the general health and strength, as well as in the respiratory capacity. In the seven cases originally reported by Dr. Chaplin excellent results were obtained. Notes of equally successful cases have been kindly communicated to me by Dr. Devereux of Tewkesbury. A full account of one of his cases has been published by Dr. Brig Dobell. Through the kindness of Dr. Dobell and of the Editor of the British Medical Journal the temperature chart of this case is reproduced on the following page as a striking illustration of the reduction of temperature which is obtained in pyrexial cases.

A case of inveterate bronchiectasis under my own care is for the present cured. In another the relief seemed to be complete, but was followed by a relapse which did not yield thoroughly to a second course. Reduction of temperature and of the expectoration and general improvement were also observed in a third patient with fibroid disease of the lung and profuse offensive expectoration; but the treatment has been temporarily interrupted, whilst these pages are being written, because of an intercurrent aggravation of the catarrh, due perhaps to irritation by the fumes.

The freedom from risk and the brilliant results hitherto secured in most of the cases reported leave us no choice: so long as we have no better method, every sufferer should have the benefit of a trial of the inhalation method. A combination with it of the administration of garlic, whilst adding a fresh therapeutic infliction to a trying treatment, would probably curtail the duration of the latter. Lastly, for cases not yielding sufficiently good results the intralaryngeal injection method would still be available.
Chart 1.—Reproduced from Dr. Brian Dobell’s report of Dr. Devereux’s case of bronchiectasis, showing the influence of the creosote inhalation treatment upon the pyrexia.
The prospect of a permanent cure will be greater the earlier the creasote treatment can be applied. Some of the inveterate cases which have long resisted all other remedies may fail to end in a complete recovery, and may need repetitions of the course. But their number will decrease as the method comes into more general use. Indeed, it is not improbable that in the future, when cases are treated sufficiently early, bronchiectasis may cease to be regarded as an incurable disease.

Surgical treatment.—An attempt was made some years ago to treat the lesions by injecting weak solutions of carbolic acid, of iodine and of other antiseptics through the chest wall into the surrounding pulmonary tissue. No good results were obtained by this method, which has since then been almost forgotten.

Treatment by incision and drainage was proposed and attempted as a last resort a few years prior to the recent advances. Although this may now be regarded as a closed chapter in the history of pulmonary therapeutics, it calls for a few retrospective remarks. The few cases of operative interference which have been reported in this country and elsewhere are not encouraging. Hofmockel, who gives a review of eighty cases of operation for abscess, for gangrene, or for bronchiectasis, finds that the worst results were obtained in the cases of bronchiectasis.

A disastrous experience has shown that success can be looked for only where a single dilatation exists. These are, however, the cases in which the symptoms are least urgent as well as least refractory to the ordinary measures. Where help is most needed—in the instances of multiple sacculations—surgery is doomed to failure. To attempt multiple incisions is to multiply the risks of septic infection of the pleura; and to open only one of the sacculations is not only to leave a great part of the disease unrelieved, but to place its remaining foci in a worse position than before, by weakening the expiratory mechanism of cough owing to the direct leakage of air, and to the unavoidable interference with the freedom of the thoracic movements.

The mechanical hygiene of respiration and the climatic treatment may be considered under one heading. They are both necessary adjuncts to any method of treatment, although in the future their relative importance will probably be less than it has been heretofore.

For the control of the catarrh and for the protection of the mucous membrane from further irritation we had until recently looked with greater confidence to the effect of climate than to medicine. The great indication was to strengthen the clogged and sodden mucous membrane by constant contact with the purest air, whilst invigorating the system by prolonged residence in a warm and equable climate, where patients might live in the open. The dry and stimulating climates to be found on the table-lands of South Africa, in South California, on some of the slopes of the Andes, or at high elevations in islands, as in the West Indies, or even in the Mediterranean, as at Ischia or Capri, are specially indicated; and along the shores of the Mediterranean there is a large selection of suitable sites. For a fuller discussion of this part of
the subject the reader is referred to the chapter on "Climate and Disease" in the first volume of this work.

Patients who at a sufficiently early date adopt and adhere to this thorough treatment by climate might hope for a permanent arrest of their catarrh, and, thanks to the increasing pulmonary expansion due to open-air life, might ultimately secure a degree of improvement almost equivalent to a cure. For this happy result a life-long treatment is now less indispensable, nor need we expatriate our patients. The climatic treatment is henceforth, as in the case of other diseases, an after-cure. A suitable climate for the winter retains its importance; but its selection is no longer limited as formerly when the consequences of any incidental catarrh were much less within our control. We might, for instance, without serious risk, in the case of some convalescents not advanced in years, recommend the dry, cold atmosphere of the Alpine winter and the outdoor life and physical exercise, which are not the least of the advantages of the Alpine cure; whilst for those unable to travel our home resorts and seaside places afford eligible climates, among which Thanet, Folkestone, Eastbourne, and Brighton deserve special mention.

Warm sea-water baths may be of considerable value. For some patients a stronger effect might be sought from the artificial Nauheim salt-water baths. In any case the temperature and the duration of the bath must be adapted to the individual. An important part of the balneal treatment is the tepid, and ultimately the cool or cold affusion terminating the bath, followed by strong friction of the surface.

Among the medicinal springs the sulphurous thermal waters enjoy a deserved reputation in the treatment of this affection. Harrogate, Moffat, Challes, Aix-les-Bains, Eaux Bonnes, Eaux Chaudes, Cauterets, Bagnères-de-Luchon, and a variety of other spas might be visited with profit; but for patients unable to leave home a substitute may be found in tonic baths combined with the internal administration, for recurring periods, of some preparation of sulphur.

At most of the foreign health stations and at some of our own various hygienic measures are recommended in addition to the use of baths or waters.

Among them special value attaches to the following:—

(a) The inhalation of an oxygenated and terebinthinated atmosphere;
(b) systematic exercise, at first passive only, of the thoracic muscles and of the abdominal muscles, including the use of dumb-bells or clubs, and a variety of postural exercises; (c) systematic respiratory gymnastics, such as deep inspirations followed by deep expirations in various attitudes, reading aloud or singing; (d) general massage and passive resistance movements followed by brisk rubbing. An improved circulation through the skin and a general bracing of its nerves are special objects of this form of treatment; another is the tonic effect on the right heart and pulmonary circulation, and the help which the mucous membrane may derive by sympathy from a healthier cutaneous surface, and from its improved reaction to atmospheric influences.
The importance of these systematic methods lies in the regularity with which they can be enforced; but the benefit they can confer might equally well be secured by a perpetual outdoor life in a really suitable climate, and by progressive exercise gradually pushed to the extent of slight breathlessness.

The contraction of the accumulations and the general improvement of the respiratory function, which are the final aims of our treatment, are directly promoted by all the measures which have been detailed; and in none of the ordinary cases, nor even in fibrotic cases if one lung be perfectly sound, need we despair of their partial attainment.

Wm. Ewart.

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PNEUMONIA

Definition — Nomenclature — Historical sketch — Clinical course — Symptoms and physical signs — Diagnosis — Complications and sequelae — Secondary Pneumonia and other Clinical Varieties — Morbid Anatomy — Histology — Bacteriology — Pathology — Etymology — Prognosis — Treatment — Statistics of four hundred and thirty-five cases — References — Catarrhal Pneumonia — Chronic Pneumonia.

SYNONYMS. — Peripneumony, περιπνευμονία, Hippocrates and later Greek writers. 1 Pneumonia, πνευμονία (Attic form πλαυμονία, used by Plutarch). Peripneumonia vera (Sydenham), as distinguished from Peripneumonia nolha, "obstruction of the lungs by a heavy, viscid pituitous matter," that is, in modern nomenclature, bronchitis. Pneumonic fever (Huxham). Pleuro-pneumonia; Acute or Siharic pneumonia; Lobary pneumonia; Croupous or Fibinous pneumonia; Pulmonary fever.

Definition.—A febrile disease, running a short course, with a special form of acute inflammation of one or both lungs.

The disease which is now understood by the name Pneumonia, when stated without qualification, has been a common acute disorder in all historical times, in all climates, and at all periods of life. It is one of the most striking and characteristic of maladies in its symptoms and physical signs, and not less so in its morbid anatomy. Other forms of acute inflammation of the lung bear the same name; but they bear it with a difference: they are, or should be, always distinguished as broncho-pneumonia, lobular, or catarrhal, hypostatic, tuberculous, or pyæmic pneumonia. These all differ pathologically and clinically from the disease now under consideration; and no less different is a chronic interstitial inflammation of the lungs—also known as iron-gray or slaty induration—which is sometimes called chronic pneumonia. It would be well if the historical name pneumonia could be restricted to the acute disease with lobar hepatisation; and the other inflammatory affections of the lung be named pulmonary catarrh, pulmonary abscesses, and cirrhosis of the lung.

History.—Before morbid anatomy was studied, or physical diagnosis invented, acute inflammations of the chest, whether affecting the parietal pleura alone or the lung with its pleural covering, were described under the name peri-pneumonia; that of "pleurisy" being applied to the sharp characteristic pain in the side which accompanies both diseases. Charlemagne is said to have died of a "fever, with a pain in the side which the Greeks call pleurisy." We now recognise the pain as due to inflammation of the pleural membrane, and the name of the symptom is applied to the anatomical change which it accompanies; pleurisy always accompanies acute lobar pneumonia, although it is often present independently; and the name pleuro-pneumonia is therefore superfluous.

The characteristic clinical features of pneumonia were identified with solidification of the lungs by Morgagni. Baillie described them as sometimes converted into a solid mass very much resembling liver (hepatisation). But Laennec, Cruveilhier, and Rokitansky completely described the anatomy of the disease. The diagnosis of pneumonia by auscultation was one of the most important results of Laennec's great discovery. The chief steps since made in advance have been the proof by Addison that the exudation of pneumonia is not into the "interstices" of the lung, but into the air-vesicles themselves; the distinction between fibrinous or lobar, and catarrhal or lobular pneumonia, which is due to Rokitansky; and the discovery of a specific pathogenetic microbe, which has been the work of numerous observers.

Clinical features of the disease.—Onset and early symptoms.—The symptom which commonly marks the onset of pneumonia is a feeling of chilliness, a fit of shivering. When this initial rigor has passed off, the patient often feels prostrate, with headache but without the severe lumbar pains which mark the onset of some acute diseases. He becomes thirsty, the skin is hot and dry, and the pulse is quickened; the tem-
perature rises rapidly from the beginning, and by the evening of the first day often reaches $103^\circ$, or in children $104^\circ$.

When twelve or more hours have passed from the onset of the disease the patient's aspect is characteristic; the face is flushed, the eyes bright and watchful, the expression that of constraint and apprehension. He usually lies on his back without marked orthopnea. Breath is short, frequent, and shallow, deep and efficient respiration being hampered by a sharp pain in the side; the pulse is quickened, full, and of increased tension, but varies less from the normal than the temperature, and still less than the breathing. The skin is dry and pungent. Except scarlatina, and ague in its second stage, there is scarcely any disease which gives such a sensation of burning heat to the hand of the observer, a character probably due to the absence of perspiration.

Not infrequently an eruption is seen upon the face, which consists of a group of small, clear vesicles on a reddened patch. This *herpes labialis* is most commonly seen on the upper lip in the neighbourhood of one or the other nostril; but it may be seen on the lower lip at the angle of the mouth, or upon any part of the cheek, chin, or jaw; hence it is sometimes called herpes facialis. Although frequent enough to form a characteristic feature of pneumonia, it is probably not present in more than two-thirds of the acute idiopathic cases we are now describing; and it is the exception instead of the rule when pneumonia is secondary to another disease. Moreover, exactly the same eruption may occur with slight pulmonary catarrh, or bronchitis without bronchopneumonia, or with a mere cold in the head. Indeed some persons have an attack of *herpes labialis* with almost every accidental catarrh. It is occasionally seen in other febrile attacks also; or it may even follow a rigor without subsequent fever. The little vesicles cause no irritation or pain; they become purulent and dry up, leaving their dark crusts, which drop off and leave no trace. The eruption lasts from ten days to a fortnight.

As in other febrile disorders, there is complete loss of appetite, with constipation. By the second or third day the tongue is thickly plastered with white fur, particularly if the patient is kept on a diet of milk. It closely resembles the appearance of the tongue at the beginning of acute rheumatism and of scarlatina.

Physical examination of the chest during the first few hours will often determine the nature of the case by the presence of a pleuritic rub, or a small crepitant râle; and towards the end of the first twenty-four hours, or on the second day, consolidation of a part of the lungs will be recognised by its physical signs. These will be conveniently considered together after the account of the external symptoms has been completed; but it must be stated here that the evidence of physical changes in the lungs, derived from auscultation, is occasionally absent on the first, the second, and even the third day. In these cases there is no reason to suppose that the local change is present without its appropriate signs;
nor are we aware of any such case in which the opportunity occurred of ascertaining the state of the lung; in the absence of such direct observation, we are justified in believing that the pneumonic fever with all its characteristic symptoms may precede the pneumonic hepatisation not only by hours but by days.

Clinical course.—The temperature, after an abrupt and rapid rise on the first day of the disease, continues at the degree then reached, or rises somewhat higher; the mean range being from 103° to 104°. In severe cases it may reach 105° and upwards; while in slight cases, particularly in children, it is often a degree lower. The same is true, even of severe cases, in elderly people. The moderation of the pyrexia, not uncommon in children, depends on the rule that the mildest cases of pneumonia, as of enteric fever, occur in children; the lower temperature in elderly patients on the still more general rule that the same degree of febrile disturbance, as measured by other symptoms and by local changes, will produce a higher temperature in a child or young adult than in an aged patient. The morning remission and evening rise, which are rarely quite lost in any case of pyrexia, are present in pneumonia; but they are less marked than in enteric fever, and still less than in suppurative fever. Hyperpyrexia is not an uncommon event; some cases indeed, and these not always the most severe, in their subsequent course, manifest a very high temperature on the first or second day. The pulse usually lags behind the rising temperature, and the respiration follows rather the extent of the local changes in the lungs than the course of the fever. (Appendix, p. 134.)

Sometimes at the very beginning of the attack, sometimes later on the first day or in the course of the second, the patient feels a sharp pain like a magnified stitch in the side. This pain, independently of auscultation, tells us which lung is affected; for although the pain is pleuritic, the pleurisy is part of the pneumonia. It is usually referred to the front of the axilla an inch or so outside the nipple; sometimes to the post-axillary line a little outside the angle of the scapula; occasionally it is felt in the mid-axilla, and still more rarely towards the base of the lung. A friction sound can usually be heard at the seat of pain; but inspection of a pneumatic lung after death shows that the pleural inflammation is more extensive than the point on the chest-wall to which the pain is referred. It is certainly rare to hear a rub under the clavicle, or above the scapula in cases of apical pneumonia; and the same remark applies to the pleurisy which so frequently accompanies tuberculous disease of the apex.

The pain felt on taking a deep breath makes the breathing shallow and hurried, without dyspnoea; and the cough is short, dry, and restrained by the patient.

The urine, from the first day onward, shows the characters of pyrexia in a marked degree. It is like that of rheumatic fever, of quinsy, scarlatina, or typhus; scanty, high-coloured and very acid. The lithates of soda and potash are deposited as it cools, partly from want of water to
dissolve them, partly from a strongly acid reaction, and partly also from an increase in the output of uric acid. The daily excretion of urea is also larger than in health.

The inorganic salts excreted in the urine—the phosphates, sulphates, and chlorides, and particularly the chloride of sodium—are diminished as in other febrile disorders; but in a much greater degree. When nitrate of silver with excess of nitric acid is added to the urine of pneumonia, it is not uncommon to see an opalescence only in place of a thick opaque precipitate. This diminution of the saline constituents of the urine does not depend merely on the patient’s diet, as experiment has proved, but is probably due to the abundant exudation of salts into the lungs. Hepatised lung is found to contain considerably more saline constituents than healthy lung, and during convalescence there is excess of salt in the urine.

Another feature of febrile urine in general is particularly frequent in pneumonia, namely, the presence of albumin. This has been observed by different authors in one-third, one-half, or two-thirds of the cases. It is probable that even the last estimate is below the truth, and that some amount of albumin is present in almost every case of primary acute pneumonia.

The sputum which the patient coughs up is characteristic. It is not abundant, not very frothy, and is unmixed with saliva or with pus. It consists of clear, tenacious mucus with a few air-bubbles, and is more or less deeply stained with blood. It hangs on the patient’s lips and clings to the vessel in which it is received. The colour, when most characteristic, deserves its traditional name of “rusty”; it is a bright orange-brown, resembling the burnt sienna of the colour-box. When the sputum is abundant and thin it loses its bright chestnut colour, and has been aptly compared to the juice of dried prunes when stewed. Under other conditions the reddish tint is lost and is replaced by a greenish yellow; the sputum is then compared in colour to greengages. When the amount of blood is scanty, a bright lemon colour is no less characteristic than the more usual rusty sputum; when, on the other hand, it is excessive, the yellowish brown is replaced by a more decided red, and in some cases pure blood is spat up. This haemoptysis is sometimes very free, and denotes, we may presume, unusually intense congestion or unusual fragility of the pulmonary vessels. Whatever its immediate cause, it does not appear to have any unfavourable import, and certainly it does not point to subsequent tuberculous disease. But the “prune-juice” and the “greengage” varieties of pnemonic sputum are justly held to be of graver significance.

On microscopic examination, beside transparent structureless mucus with a few small air-bubbles, the sputum of pneumonia is found to contain abundant blood-diges, a few leucocytes, and frequently minute casts of the smallest bronchioles. When treated by appropriate staining, the pneumococcus or diplococcus described by Frankel—an oval or rounded organism with a thick transparent capsule—is revealed; some of them are
separate, some in groups of three or four; but almost every slide will show several pairs united two and two in characteristic fashion.

The rusty sputum has of all the symptoms of the disease the best right to be called distinctively pneumonic. Its peculiarity is due to the hemorrhagic quality of the inflammatory exudation itself, which may be compared in this particular to that of acute glomerular nephritis. As in the smoky urine of this disease so in the rusty sputum the blood-discs have been poured out uniformly and continuously from the first, and are therefore more equally distributed than when hemorrhage is added to inflammation as a subsequent event. Accordingly we do not find the characteristics of “rusty” sputum in the hemoptysis of phthisis, of aneurysm, of purpura, or of laryngeal or tracheal ulceration. Nor is it, as a rule, to be seen in cases of cardiac disease with consecutive pulmonary hemorrhage. In these cases the blood is more separate from the sputum. When, as sometimes happens, characteristic rusty sputa are observed in the course of heart disease, it is probable that the hemorrhage is complicated by local consecutive hepatisation. This is often recognised during life, but still more often it is ascertained after the patient’s death.

It is well known that children do not expectorate; hence we lose in these cases the important help derived from inspection of the sputum. Even as late as the age of eleven and twelve years the patient may be unable or unwilling to bring up the phlegm. When a child vomits after coughing we may sometimes observe characteristic rusty sputa in the basin. Old persons, as a rule, are like children in this respect; they seem unable to expectorate the sputa.

The nervous system is less frequently and severely affected in patients suffering from pneumonia than in most cases of typhus or enteric fever; but more frequently than in rheumatism without hyperpyrexia. From the beginning the patient’s rest is disturbed; and if the case be protracted, insomnia may become a grave and distressing incident. Some degree of nocturnal delirium probably accompanies almost every state of pyrexia, even that which attends a feverish cold; and it is sometimes severe in attacks of influenza otherwise mild. In this slight degree, shown by the persistence of the impression of dreams, and by confusion of time and place, delirium is probably present in every case of pneumonia. It seems never to take the terrible form occasionally seen in rheumatic fever, and associated with hyperpyrexia and pericarditis; but it is sometimes important from its preventing sleep and prompting attempts to get out of bed. When an intemperate person is seized with pneumonia, the febrile delirium, as in other cases of this nature, assumes the characters of delirium tremens; and the gravity of the case is at once apparent. But even in pneumonia of the most temperate persons diurnal delirium is a serious symptom.

Termination.—In a slight case of pneumonia, particularly when affecting a child, after a sharp onset and a day or two of fever the temperature falls rapidly, the skin begins to act, refreshing sleep is obtained, and the patient awakes on the third day to find convalescence begun. These
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Cases are, however, the exception. The symptoms much more often, even in favourable cases, last till the fifth, the sixth, or the seventh day; and in many persons who make a good recovery the illness is prolonged into the second week, so as to give a duration of eight, nine, or ten days. Beyond this the duration of the disease, unless due to some local cause, is certainly rare. The most frequent, extension of the inflammation is to the other lung; but pleuritic effusion, and particularly effusion of pus, pericarditis, or severe and persistent bronchial catarrh may protract the disease beyond its natural limit. (Appendix, p. 135.)

In unfavourable cases it is very rare for death to take place on the first or second day, as in malignant variola or scarlatina. Even with double pneumonia the patient seldom succumbs before the fourth day; the time of greatest anxiety is that of the latter days of the first and the early days of the second week. In such cases, while the temperature still ranges high, the insomnia and delirium become more severe; the tongue is dry and brown as in typhus, and the pulse more frequent, weaker, and perhaps irregular. The rapidity of breathing, the dyspnoea and the cyanosis depend upon the extent of lung involved. When the whole of one lung is hepatised, the other rarely escapes; and in such cases additional difficulties in aerating the blood are often caused by profuse bronchial secretion which chokes the air-passages, passes up and down with each weak breath, and is not expelled by a vigorous cough. In the other cases, even with moderate secretion and without signs of cyanosis, the heart begins to flag, the pulse grows small and weak, and the condition becomes like that of a typhoid patient at the end of the third week.

The favourable close of an attack of pneumonia, whether earlier or later, will almost always be marked by a critical fall of temperature; and even in cases which prove fatal by some intercurrent complication, this crisis may often be observed. Indeed a gradual fall of temperature, or a temperature which continues high after ten or twelve days, should lead to a revision of the diagnosis, and to a careful search for some disturbing condition apart from the primary disease.

Physical signs.—The auscultatory evidence of pneumonia, which was discovered by Auenbruger and Laennec, and has since been confirmed, corrected, and elaborated by a succession of physicians, is in the majority of cases striking and unequivocal. It suffices for the recognition of the presence, extent, and course of pneumonia without the aid of other symptoms; just as a sure diagnosis of the disease can be made from the aspect of the patient, the pain, the sputum, the urine and the fever, without the aid of percussion or auscultation. Together they make the recognition of primary pneumonia one of the easiest of the physician’s tasks.

It is seldom that we have the opportunity of examining the chest at the beginning of the disease. Within a few hours of the initial rigor and rise of temperature we often find the percussion note at the base of one lung less clear and flatter than at the other; the respiratory murmur has lost its normal character, and has assumed more or less of the bronchial,
tracheal, or tubular quality. Sometimes, however, mingled with this diminished dulness and with the "vesiculo-bronchial" breathing, or even preceding it by a few hours, may be heard an adventitious murmur which is very characteristic, and was thought by the earlier auscultators to be even more frequent and more decisive than later experience has confirmed. This is the rôle crépitant of Laennec, who briefly described it in the following words:—"Dans le premier degré de la périplineumonie, la respiration s'entend encore dans le point affecté, soit que la percussion donne en cet endroit un son mat, soit qu'elle n'indique aucune différence sensible, ce qui varie. Mais la respiration, quoique sensible dans le lieu affecté, est cependant beaucoup moins grande et moins sonore que dans les autres parties de la poitrine; elle est, en outre, accompagnée, dans l'inspiration surtout, d'une espèce de crépitation ou du rôle léger, dont le bruit peut être comparé à celui du sel que l'on fait décrépiter en le chauffant dans une bassine; ce rôle, que j'appelle rôle crépitant, est le signe pathognomonique du premier degré de la périplineumonie. Il serait difficile de le mieux décrire; mais il suffit de l'avoir entendu une seule fois pour ne plus le méconnaître" (17, § 209).

In other places the illustrious French physician admits that the same "rôle crépitant" may be heard in cases of oedema of the lung. He says: "Le cylindre, dans ce cas, offre deux moyens de reconnaître l'altération du poumon. La respiration s'entend beaucoup moins qu'on ne devrait s'y attendre, à raison des efforts avec lesquels elle se fait et de la grande dilatation du thorax dont elle est accompagnée. L'on entend en même temps, comme dans la périplineumonie au premier degré, une légère crépitation plus analogue au rôle qu'au bruit naturel de la respiration. Ce rôle crépitant est moins fort que dans la périplineumonie au premier degré: cependant on doit avouer qu'il est fort difficile de distinguer ces deux affections l'une de l'autre à l'aide des seuls signes donnés par le cylindre, et qu'il est nécessaire d'y joindre la comparaison des symptômes généraux." (17, § 500).

This remarkable sound is, as Laennec says, inspiratory; occasionally it may be heard with expiration also, but this is exceptional. It is a "moist" sound, or, to speak more strictly, it is an interrupted sound; the parts of which it is made up are very short, very numerous, and uniform in duration and quality. Though perfectly audible and distinct, it is not loud; lastly, its quality is thin, rather high pitched, and what is called "musical," "bright," or "clear"; that is to say, it has more tone and is farther removed from a mere noise than the respiratory murmur, a sonorous rhonchus, or than the bubbling râles in the trachea which form the death-rattle. On the other hand, it is more of a noise, and has less tone or musical quality than the metallic tinkling heard in a large cavity, or the clear percussion note of pneumothorax or of gastric distension. Unlike other râles, it is not influenced by coughing. It sometimes persists for a few hours only; sometimes it lasts until dulness and tubular breathing show that the lung is completely solid; sometimes it leaves the place where it was first heard, and ascends with the advancing
line of hepatisation; or it appears on the opposite side as the first sign of extension to the other lung. Lastly, under the title "crepitus redux," it may reappear, somewhat larger, louder and less musical, when the solidified lung is recovering and again admitting the air.

This pneumonic crepitation is so peculiar and remarkable a sound that after it has been heard two or three times it is easily recognised; but, beside the account of its acoustic properties attempted above, it may be compared to some other sounds. Laennec's own illustration, quoted above, is that of the crackling sound produced by heating salt over the fire; this resembles the rôle in being an interrupted sound, and in the clear sharpness of its quality; but the crackles are fewer, larger, and louder. The late Dr. C. J. B. Williams compared pneumonic crepitation to the sound heard when a lock of hair is gently rubbed between the fingers close to the ear. My own illustration for students was by squeezing a piece of the porous indiarubber, formerly much used instead of a toilet sponge, after it had been soaked in water. The late Dr. Sturges reproduced the sound by pressing dry tissue-paper into a ball, or by squeezing and relaxing a piece of sponge dipped in gum-water.

The "redux" crepitation is sometimes indistinguishable from that of pulmonary haemorrhage, or of broncho-pneumonia, or from that which is commonly regarded, since Laennec's time, as a sign of oedema of the lung.

The true pneumonic crepitation undoubtedly differs from these in being finer or smaller, that is to say, the crackles are shorter and more numerous; they have more tone and are less loud; they are more often confined to inspiration, and are uninfluenced by deep breathing or by cough.

Nevertheless the ear of most auscultators tells them that these sounds are all similar; thus from the more definite pneumonic crepitation we pass by small gradations through "redux" crepitation to that of broncho-pneumonia, and so on to the smaller consonating râles of phthisis. On the other hand, pneumonic crepitation is quite unlike any respiratory murmur, modified or unmodified; unlike sibilus or any other continuous sound, and unlike non-consonating râles of every degree. The only sound heard in the chest which may simulate it is a very soft pleuritic rub, not loud, but clear and audible at the end of inspiration.

The physical explanations of this remarkable auscultatory sign are of doubtful validity. Probably the most generally received opinion is that it is caused by the opening out of the small extra-lobular bronchioles, by the inspired air, while their walls are covered with viscous exudation. This explanation, however, appears improbable, as it can scarcely be applied to the explanation of expiratory crepitation; and we have no evidence that collapse of the channels of the air-passages occurs as a matter of fact. Moreover, true pneumonic crepitation is thus too widely separated from the allied sounds above enumerated. From these indeed it may be distinguished by the practised auscultator, who rightly teaches students to make the same distinction; nevertheless we must admit
with Laennec that the distinction is sometimes difficult. The air-vesicles cannot be the seat of this crepitant sound any more than they can be the seat of the normal respiratory murmur, or indeed of any auscultatory events; for the air in the vesicles is not changed save by diffusion: the strongest efforts of respiration produce a current in the air-passages only, which does not reach the lobules. Pneumonic crepitation is an interrupted and probably a "moist" sound, that is, a sound made by bubbles bursting, and is a consonating rôle, that is, a sound transmitted through a solid lung. Regarding it, then, as the finest or smallest of this group, we may ascribe its production to the same cause, namely, to air passing through fluid and making a series of crackles which are transmitted to the ear through a hepatised lung.

There is little reason to acquiesce in the three stages of pneumonia described by Laennec, either on anatomical or clinical grounds. The first stage, that of engorgement or active congestion, is in all probability the beginning of hepatisation, and with the afflux of blood comes the exudation; congestion and hepatisation begin and go on together, and the consonating role just discussed is the earliest sign, not of preliminary hyperemia, but of actually existing inflammation. Hence we find it mingled with the signs next to be described, signs which are admitted to denote complete solidification of the lung.

These are bronchial breathing, bronchophony, and increased vocal fremitus.

This is not the place to discuss the difficult questions involved in the physical theory of the respiratory murmur and its modifications in disease. It must suffice to assume, as most in accordance with physical facts and least contradicted by clinical experience, the hypothesis which refers the normal respiratory murmur to vibrations of air due to a fluid vein, which is produced in inspiration and expiration by the passage of the air through the narrow chink of the glottis into the wide channel of the trachea in the one case, and of the upper larynx in the other. The sounds thus produced are heard by a stethoscope placed upon the larynx or trachea; where they have the loud, somewhat harsh, continuous, blowing character, which is recognised as tracheal, or an extreme degree of bronchial or tubular breathing. On listening to the patient's voice with a stethoscope on the trachea we hear the loud vocal resonance which is known as bronchophony. The same inspiratory and expiratory murmur and vocal resonance are heard in most persons over the manubrium; and in some on applying the stethoscope between the shoulder-blades, or over the inner part of the first right intercostal space: but the sounds thus transmitted (not by the column of air, but by the walls of the trachea and right bronchus), while still harsh and blowing, are less loud than the tracheal murmur just described. This bronchial breathing is certainly no new sound; it is the tracheal murmur diminished in intensity, because less directly conveyed. As soon as we leave the neighbourhood of the trachea (that is to say, over nearly, if not quite, the whole of the chest) we find the resonance of the voice much diminished,
even when it is that of a strong man with thin covering of the thorax; while if the thorax be thickly covered by clothing, subcutaneous fat, or even thick muscles, or if the voice, as in women and children, be shrill and comparatively feeble, the vocal resonance is weak and not infrequently absent.

Since the vibrations of speech in the larynx are conducted by the walls of the air-passages, and when they reach the surface of a healthy lung are still the same sounds, though diminished in loudness or modified in character; and since again the sound of the voice is greatly altered when transmitted through a solid lung, or through pleural effusion, or through emphysematous lung, or through pneumothorax, yet, however altered or modified, is still the sound produced by the vibration of the vocal cords —so, according to the hypothesis now advocated, the breath-sounds heard over the chest are not produced in the pulmonary vesicles, the lobules or the bronchial tubes, but in the larynx.

The pulmonary murmur is heard indeed over the lungs, but does not arise there. It is the same sound which over the trachea we call tubular, with its loudness diminished and its quality altered by transmission through the spongy lung.

On this view we do not start with a so-called vesicular murmurs in the lung, which becomes bronchial in the bronchi and tracheal in the larynx; but in breathing and in voice we regard the audible vibrations as formed at the glottis and altered, whether in health or disease, by transmission through various media.

Now when, as in pneumonic hepatisation, the laryngeal breath-sounds are transmitted through a solid lung, they retain much of their loudness and quality. The expiratory murmur is not shortened and weakened, or even rendered inaudible, as in health. Inspiration and expiration are accompanied by a murmur nearly equal in length, loudness, pitch and quality.

The simplest way of describing the breath-sounds heard in pneumonia is to call them tubular, bronchial, or tracheal; and to define them by reference to the respiratory murmur as heard over the trachea or the manubrium in health. But, simple as it is, this comparison is not completely accurate; it may help the student in the rudiments of auscultation, but it will probably hinder him when he hears well-marked pneumonic breathing. It seems that in phthisis, lobular pneumonia of children, and other conditions of partial solidification, and again in some cases of narrowing of a large bronchial tube whether by stricture or compression from without, breath-sounds may be heard which are very different from the pulmonary murmur, and which closely approach that heard over the larynx. These may be fairly called bronchial or vesiculo-bronchial sounds. They differ from the tracheal murmur chiefly in loudness, so that expiration loses its later part and becomes shorter than when heard over the larynx, though longer than over a healthy lung.

The word "tubular" is often applied indiscriminately as a synonym of tracheal or bronchial breathing; but by many writers, and I think
with advantage, it is reserved for the special modification heard most perfectly in cases of pneumonic hepatisation.

This modification essentially resembles laryngeal, tracheal, and bronchial breathing, and differs essentially from the healthy pulmonary murmur. It has a blowing rather than a breezy quality; and expiration is often as loud and usually as long as inspiration; but it differs from the normal laryngeal murmur in the following points: it is not so loud, nay, it may be softer than the bronchial breathing above described; but, however subdued, it is remarkably distinct—audible, that is, by reason of its peculiar quality, when a much louder breath-sound might pass unnoticed. Again, it is higher pitched than the pulmonary murmur and than laryngeal or tracheal breath-sounds; thirdly, it has the quality of tone, that is to say, it is farther removed from a mere noise, and approaches though it never reaches the quality of a musical note. To these characteristics we may add that expiration does not follow inspiration as immediately as in health—possibly because the solidified lung does not contribute its resilient energy to that of the rest of the lungs, or it may be because of some disturbance of the nervous mechanism of respiration. In short, the laryngeal vibrations in breathing are transmitted to the ear better than in health, less changed and changed in another direction.

Along with the tubular breathing marked bronchophony is present, that is, when the patient either speaks or coughs, the laryngeal voice is heard more loudly and distinctly than with a healthy lung. The tactile fremitus is also more perceptible, that is, the laryngeal vibrations are more perfectly transmitted to the touch as well as to the ear.

A remarkable variety of vocal resonance usually heard over a pneumonic lung is that called by Laennec “pectoraliloquy”; in which case not only the laryngeal voice but also the articulated sounds in the mouth are transmitted, so that words as well as vocal sounds are heard. If the patient speak in a whisper, the latter of course are absent, and we hear the former alone, just as if a loud whisper were directed into the stethoscope. Thus pectoriloquy is best distinguished as whispered pectoriloquy, but vocal pectoriloquy is just as real a phenomenon, and was what Laennec first described.

There are some curious exceptions to the regularity of these physical signs. Occasionally the percussion note loses its resonance, but at the same time rises in pitch; this condition, sometimes found over a very tense coil of intestine, is known as tympanitic dulness. Still more rarely a solid and hepatised lung has been found by good observers to yield a resonant note, even in the presence of bronchial breathing and bronchophony. Occasionally, instead of the ordinary dull or flat toneless percussion note, a “cracked-pot” sound may be heard, particularly in children.

In some cases, where presumably there is a plug of fibrinous exudation, bronchial breathing and the associated signs are absent: such a case resembles a pleural effusion save that the heart is not displaced.
Since pneumonia is always accompanied by pleurisy, a pleuritic rub will almost always be heard early in the attack; most often to the outer side of the nipple or near the angle of the scapula.

The physical signs just described undergo little alteration as during the progress of the disease hepatisation spreads upward or attacks the other lung: pneumatic crepitation may be heard at the advancing border, and the area of dulness and tubular breathing extends. When the crisis arrives, the solidified lung clears up, but more slowly than the symptoms. The dulness gradually gives place to resonance; the "redus crepitation" is often heard, and instead of tubular breathing we hear râles, at first consonating and then losing this quality as they become larger and looser.

Sometimes the signs of consolidation continue for a week after the temperature has become normal, or even longer than this. Sometimes they are succeeded by the signs of pleural effusion.

Diagnosis. — It is customary, after describing the symptoms and signs by which a disease is recognised, to add special directions for distinguishing it from other diseases. But it is often impossible to predict what maladies may be confused in a given case; and when we draw up tables of distinguishing signs, although the exercise is useful for a student, we find, on the one hand, that each has to be so guarded and qualified that it ceases to bear the weight put upon it, or on the other that we are making our statements more absolute than facts will bear out: thus we may mislead rather than help the reader. There are no pathognomonic symptoms of any disease, nor is there any royal road to diagnosis.

In some cases, as in the discrimination of external tumours, in the distinction between measles and rubeola, rheumatism and gonorrhoeal synovitis, psoriasis and scaly syphilis, tuberculosis and enterica, it is practically useful to keep the contrasted features in our minds; though in each of these cases we may rely too much upon them, and may fail to observe other points of greater importance. But pneumonia is so well marked a malady in its symptoms and course, and auscultation gives us such clear and precise evidence of its presence, that, once suspected, it can always be discovered; and the disease is too common and striking not to be thought of whenever an acute febrile attack is before us.

In children, confluent pulmonary catarrh may simulate lobar pneumonia in its physical signs; but its onset and course are very different, so that we have only to bear in mind that true pneumonia may affect young children and even infants at the breast.

Apical pneumonia has been mistaken for phthisis; but if the sputa and the curve of temperature do not distinguish them, the onset and the crisis are decisive.

When pneumonia comes on in its secondary form as a complication of some chronic disease, it may be easily overlooked; but, if sought for, the characteristic physical signs prevent all doubt of its presence. A rise of temperature, with rapid breathing or quickly ingramescent muscular weakness, are the symptoms that should at once lead to auscultation of the lungs. When pneumonia complicates fevers we are already on the watch,
and an increased ratio of respiration to pulse and temperature at once excites suspicion and indicates the danger. In the course of delirium tremens we must always be on the look-out for the supervention of pneumonia; for the entire lower lobe of a lung may be hepatised before the appearance of characteristic symptoms.

In children, fever, dyspncea, and cough may coincide with dulness on percussion and characteristic tubular breathing at the base of one lung, and the cause may be not hepatisation, but pleural effusion, most often purulent. No sputum is obtainable, and the voice fails to give help. In such cases, and in such only, we are driven to leave the art of diagnosis by physical signs, and to solve the problem by puncture with a hypodermic syringe.

Other difficulties of diagnosis turn rather upon disputed pathological points than on technical skill in interpreting symptoms and signs.

The first depends upon the distinction of true pneumonia from what has been often described as "acute pulmonary congestion." This affection, described by practitioners in England, and admitted by some pathologists in France, may be defined as an acute disease with the symptoms but without the physical signs of pneumonia, running a short and for the most part a benign course, usually calling for and sometimes obtaining antiphlogistic treatment. That an attack of pneumonia occasionally aborts, that the physical signs of pneumonia are sometimes delayed for two or three days after the symptoms have appeared, and that they are sometimes strangely obscured or difficult to detect, are facts that probably explain most of these cases. The remainder may perhaps be regarded as examples of acute pulmonary oedema, like that which not infrequently attends the later stages of Bright's disease. In any case acute arterial congestion without exudation — hyperemia without inflammation — is a pathological event the existence of which has never been proved, and cannot be admitted until supported by the results of post-mortem examination.

Another question of diagnosis depends upon our view of the relation of lobar and lobular pneumonia in children. When patches of catarrhal pneumonia become confluent, the physical signs closely resemble those of fibrinous hepatisation; the symptoms are also more acute in these cases, and may be undistinguishable from those of the latter. Even anatomically it is so difficult to distinguish them that some good pathologists give up the attempt. Moreover, the pneumococcus may be present in lobular as well as in lobar pneumonia. The distinction rests on the more gradual onset, the relation to previous bronchitis, measles, and other primary disease, the more scattered localisation; anatomically, on the separate lobules being distinguishable, the surface less firm and granular; and microscopically, on the greater amount of leucocytes and epithelium, and the smaller amount of fibrin and blood-discs. Confessedly difficult as is the diagnosis between true lobar pneumonia and lobular catarrhal consolidation, there does not appear to be sufficient reason for giving up the attempt. The two diseases differ essentially in their natural history,
origin, incidence, course, and histology; and the difficulty of distinguishing them in what is after all a small proportion of cases is no more a reason for confounding them than in the similar occasional difficulty of distinguishing between rubella and morbilli, osteoarthritis and gout, carcinoma and alveolar sarcoma. If the whole duty of medicine were the practical one of healing the individual patient, we might be content with the knowledge that in the doubtful cases, which sometimes occur in children, the determination of the question is not of practical importance.

The third difficulty in diagnosis is between pneumonia with hepatisation of the lung and the cases of acute pleurisy and even of acute meningitis, cerebral or cerebro-spinal, of acute pericarditis, or ulcerative endocarditis, which, as it is asserted, have the same general symptoms and course, the same infective microbe, the same origin and pathological nature, and yet no affection of the lungs—pneumonic fever without pneumonia.

The diagnosis in these cases would rest upon the absence of the physical signs of a solidified lung and of rusty expectoration and other strictly pulmonary symptoms. The evidence for the theory in question will be noticed again in the section on pathology.

Complications and Sequels.—Pneumonia is more uniform in its course than most febrile disorders, and offers in this, as in other respects, a marked contrast to enteric fever. Its complications are few and rare.

Hyperpyrexia is comparatively rare. The temperature runs high, but seldom exceeds 106°; and deaths from this cause alone are uncommon. At the same time a temperature above 104° marks a severe attack, at any rate in an adult.

Pleurisy is not a complication, but part of the disease. In its usual fibrinous form it is insignificant except for the pain it causes. Serous effusion is seldom considerable, and rarely needs attention; but after the pneumonia has subsided, it sometimes happens that the dulness persists longer than usual, and the temperature rises again after the initial fall—the interval in Dr. White’s 26 cases was from one to four days. This almost always points to the presence of fluid in the pleural cavity, and as a rule this fluid is purulent. In a case lately under my own care, the pus, when drawn off, was found to be a pure cultivation of Fränkel’s pneumococcus; and this is frequently the case. But more often the organisms found in the effusion are the strepto- or staphylo-coci of non-specific suppuration. It is certainly remarkable that the diplococcus, which usually produces non-suppurative inflammation of the lungs and pleura, should occasionally cause the purulent infiltration which marks the last stage of hepatisation, and the empyema which we are considering: the two conditions, though so far comparable, seldom or never exist together; a mixed infection might rather have been anticipated, pyogenetic

1 In a paper in the Guy’s Hospital Reports for the past year (vol. II.) by Drs. Hale White and A. C. Pearce, 26 cases of empyema are recorded following 890 cases of lobar pneumonia. The percentage on the total is three, but in different years it varied widely from none up to more than five per cent. Of 705 cases of pneumonia recorded in the St. Thomas’s Hospital Reports (vol. xi.), only 6 were followed by empyema.
microbes being added, as in the latter stages of enteric fever, to those which are specific. On this point further evidence is needed.

From the clinical point of view it seems very doubtful whether time will justify the belief entertained by some French pathologists, that an empyema of diplococceous origin is benign, and may be absorbed without surgical interference, while the contrary is true of one in which streptococci are found. In the case above mentioned a second paracentesis was necessary, and ultimate incision and drainage before recovery ensued.

A common and more dangerous complication is pericarditis, which, when it complicates double pneumonia, is most often fatal. It occurs during the height of the fever, and is recognised by the usual friction sound, which must be distinguished from a pleuritic rub produced by the impulse of the heart. The effusion is usually plastic and serous, but occasionally it is purulent. There is no added pain, but dyspnœa and orthopœneœ are increased, and the pulse becomes smaller and irregular.

Another complication or sequel is ulcerative endocarditis. Dr. Osler, who in 1885 drew attention to this connection of the two diseases, met with it sixteen times in 100 cases. This was probably an accidentally high number. In the 425 cases tabulated by myself it occurred seven times. It often accompanies empyema.

A rarer complication is acute meningitis. Of this I have had but small experience. In the few cases I have seen, the pneumonic diplococci were found in the lymph at the base of the brain; and this appears to be the rule. Meningitis is often associated with acute ulcerative endocarditis.

Dr. Bristowe and other writers have described a membranous catarrhal or ulcerative colitis as complicating acute pneumonia. There have been several cases at Guy's Hospital during the experience of many years, but I have myself only once found this condition of ulceration of the colon. It is certainly far more common in cases of Bright's disease.

Severe epistaxis occasionally marks the onset of pneumonia, and may recur during its course. Except in elderly patients, this is not a serious symptom.

A curious occasional complication of pneumonia is jaundice; and this is more often observed when the right lung is affected. None of the attempted explanations of its occurrence is satisfactory. There is no evidence of catarrh of the bile-duct (if catarrh is ever the cause of icterus), there is no reason to suppose the blood differently constituted in these particular cases (if there be such a thing as hematogenous jaundice), the hepatic circulation is not more obstructed in these cases than in others without the symptom in question, and deficient movement of the diaphragm, if a cause, would be more likely to induce jaundice in cases of right hydrothorax or phrenic pleurisy. It does not appear to affect the course of the attack or its prognosis.

Relapse after recovery from pneumonia has been often reported; and cautions are given to guard against it. But I have never met with a case, and believe that a relapse occurs very rarely, if ever. The spread of
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Pneumonia to the other lung when it had subsided in the first, or the supervision of empyema, may perhaps have been thus misinterpreted.

Recurrence of pneumonia after several months or a year's interval is, however, often observed. In the 425 cases tabulated, such recurrence is noted in nineteen. This shows that pneumonia does not protect against a future attack like small-pox or measles, not perhaps that it disposes to a repetition like erysipelas; occasionally the same patient may suffer five or six times from pneumonia in his lifetime.

SECONDARY PNEUMONIA.—The description of the symptoms and course of pneumonia above given refers with comparatively small variation to cases of primary, or, as we say, idiopathic pneumonia; to cases, that is, in which the disease, whatever its cause, is not a complication of any previous malady, but attacks a person in apparent health. There are, however, numberless cases in which after death we find lobar hepatisation as the closing event of preceding disease, acute or chronic. The anatomy to the naked eye is the same; and neither histological nor bacterial investigation enables us to distinguish the forms of primary from those of secondary pneumonia; or to distinguish one from another those which occur in the course of typhus, rheumatic fever, diabetes, or Bright's disease.

It would be an arbitrary proceeding to separate the one group of cases from the other: for, in the first place, the anatomical changes of disease are by far the safest guide in their discrimination and recognition; and, secondly, although the symptoms of these secondary forms of pneumonia are less clear and distinct than in the primary cases, yet they are present, curtailed or obscured it may be, but capable of detection and accompanied by the same physical signs which denote the presence of primary pneumonia.

Moreover, we find a connecting link between primary and secondary pneumonia in cases following influenza, which in other respects have the character of the idiopathic disease rather than that of a complication or sequel.

When one disease supervenes upon another, the former is not likely to preserve the distinctive features which mark its invasion of a normal organism. The contrast between health and disease is more obvious than between one kind of sickness and another. The antecedent disorder of the pulse, temperature and respiration, of the appetite, of the blood, of the tongue, and of the skin, blurs the outline of those striking changes which in a healthy subject denote the advent of pneumonia. In like manner after death we may discover the ulceration of enteric fever which has supervened in a course of protracted rheumatism with relapses; or cancer in the body of a patient who has died from alcoholic intemperance; or Bright's disease in a case of emphysema and bronchitis; or phthisis latent in a diabetic or insane patient.

The diseases in which secondary pneumonia frequently occurs as a complication are the following: typhus, enteric and relapsing fevers, small-pox, erysipelas, puerperal septicæmia, and occasionally diphtheria.
Lobar fibrinous pneumonia, of which alone we are now speaking, is rarely seen as a complication of measles, but it is less rare in scarlatina. It seldom appears in the course of phthisis, or again of quinsy, bronchitis, pleurisy, or asthma. It may be seen as a complication of cardiac disease, but is much more frequent in both the acute and chronic form of Bright’s disease. It is an occasional and often fatal complication of rheumatic fever; and is almost invariably fatal when associated with pericarditis. Acute lobar pneumonia of the base is often found as the cause of death in diabetes, and in chronic alcoholic poisoning; and it may occur as a fatal complication in the course of many chronic diseases, such as paraplegia, tabes, or cancer. Pneumonia, probably of septic origin but neither lobular nor suppurative, is also not an infrequent cause of death after surgical operations. It was remarked by the great Dr. Arbuthnot, in the third chapter of his book on Diet, that a peripneumony is the last phase of every disease; for nobody dies without a stagnation of the blood in the lungs. If we include broncho-pneumonia and hypostatic congestion under the name of pneumonia, as Arbuthnot, no doubt, would have done, the proposition is not very far from the truth.

In what points do these secondary forms of pneumonia differ from the primary? In the first place, they are far more dangerous. Secondly, although their anatomy and their physical signs are the same, they lack some of the symptoms of the idiopathic disease: more particularly it may be observed that the initial rigor is frequently, perhaps usually, absent; that the temperature does not rise so abruptly or so high; that although pleurisy is present, and a rub may be heard if sought for, yet pain in the side, instead of being a constant symptom, is often absent; that a cough may also be wanting; that the burning heat of the skin and the characteristic conditions of the urine may be absent or slightly marked. Herpes is usually absent. The sputum is rusty, and contains diplococci; but often in these cases, either from absence of bronchial irritation or from the weakness of the patient, no sputum is expectorated. If all these symptoms are wanting, and fever already present, a secondary pneumonia may be called latent; and we have then to depend entirely upon physical examination of the chest for its recognition. Apart from these signs, probably the most important symptom is increased frequency of respiration.

Other clinical varieties.—Beside the distinction between primary and secondary pneumonia, certain other divisions of the disease have been made, by some writers, into varieties which scarcely deserve separate nomination.

 Abortive or ephemeral cases, which last only a day or two; wandering pneumonia (P. migrans), in which form the disease attacks first the right base, then the left, and then perhaps returns to the right apex; malarial pneumonia, in which under the influence of this poison the fever assumes an intermittent character, are among the varieties which might be multiplied without advantage.

In children, pneumonia is comparatively rare, its course usually short,
and its prognosis very good. The pneumonia of old people is often attended by only moderate fever; bronchitis is common and exhaustion speedy. The drunkard's pneumonia is complicated with early delirium.

Epidemic pneumonia, apart from that consecutive to influenza, has been frequently observed in former and recent times. An interesting historical account of it is given by the late Dr. Wilson Fox (12) (13), and also in Sturges' and Coupland's valuable monograph. Recent epidemics have been described by Dr. Whitelegg in the first volume of the present work (p. 655).

Morbid anatomy.—The anatomy of pneumonia is no less striking and characteristic than its symptoms or its physical signs. The hepatised lung does not collapse on exposure to atmospheric pressure. It feels heavy when lifted out of the chest, and readily sinks in water. Its surface is covered by a more or less extensive layer of fibrin. This false membrane, or coagulated lymph as it used to be called, is sometimes a thin, adherent gray film, contrasting with the smooth and shining portions of the membrane which are unaffected. Sometimes it is in thick and comparatively tough layers, which can be peeled off; sometimes in soft shaggy masses of moist fibrin; and sometimes, together with the solid exudation, there is more or less of serum or pus.

On cutting into the lung the section is seen to be dark red, the colour of liver; or at a later stage a pale and yellowish gray. The advancing border is red and edematous, but not yet solid. The surface is granular, uniform, and dry compared with most other morbid states of the lung. As in other acute inflammations, the tissue is so softened that it readily breaks down under pressure. A scanty, blood-stained liquid, characteristically free from froth, issues from the squeezed tissue; and this becomes more abundant, paler, and thicker in the parts longest hepatised —that is to say, in the gray as compared with the red hepatisation.

The traditional stages of pneumatic hepatisation are—(1) Engorgement (Bayle), that is, acute congestion with edema, but without solidity; (2) Red hepatisation, that is, solid exudation with hyperemia; (3) Gray hepatisation or solid exudation with anemia from pressure on the blood-vessels; (4) Purulent infiltration, the gray tissue yielding thick purulent exudation.

Histology.—On scraping the cut surface we obtain a turbid liquid, of which a drop under the microscope shows abundant red blood-discs, with a few leucocytes; or, in the latter stages, abundant leucocytes, and a few blood-discs, together with minute fibrinous casts of the vesicles. Larger branching casts, plainly visible to the naked eye, can often be pulled out of the bronchioles with forceps. Diplococci may be detected after staining, and sometimes streptococci in addition.

A thin section of the hepatised lung shows the vesicles and intralobular air-passages, as well as the smallest bronchial tubes, to be completely filled with exudation: blood-discs, threads of fibrin, and leucocytes, the latter at first few in number, but becoming more numerous as the red turns to the gray stage.
The absence of epithelial cells is important. The walls of the air-vesicles are usually thickened, but no other change of the texture of the lung is apparent, no alteration of the elastic fibre, and no increase of the exceedingly scanty interstitial connective tissue of the healthy lung. The capillary blood-vessels in the early stages of hepatisation are dilated; but the blood is gradually squeezed out of them as consolidation goes on until red gives place to gray hepatisation, when the lung becomes very anaemic. The remarkable friability of a pneumonic lung is caused partly by the swelling and softening of the tissue, but chiefly by its being solid instead of spongy, so that instead of yielding to pressure, it resists and breaks down under it. In the later stages the blood-discs are no longer seen, or only few of them; the leucocytes, on the other hand, have greatly increased in number, and have assumed the granular character of pus-cells. The air-vesicles, being thus stuffed and swollen with inflammatory exudation, expel the blood from the pulmonary capillaries and cause the exsanguine pale aspect of gray hepatisation. The gray colour is due to the accidental pigmentation with granules of carbon due to inspiring smoky air, and is absent when the hepatisation of the lungs is observed in children or in cattle.

Anatomical distribution.—The site of hepatisation is important. In the majority of cases (288:92) it begins at the base of the lung and travels slowly upwards—in the reverse direction to that characteristic of phthisis. The epithet lobar is to some extent unfortunate; for the process is seldom exactly bounded by the fissures of the lung. The lower lobe may be hepatised only in its posterior-inferior part, or in all but its apex; or the back of the upper lobe and the whole of the middle and lower lobes of the right lung may be solid and the apex alone escape. Sometimes every part of one lung is found solid after death; the base gray and the upper parts red, with hepatisation of the base of the other lung.

Not infrequently, however, pneumonia attacks the apex and travels downwards. This local variety is more common in children than in adults; it is often latent, and is said to be more often associated with delirium. The prognosis of apical pneumonia, when it occurs in an adult, does not seem to be better or worse than that of the ordinary basal form.

Apical pneumonia of one lung is often accompanied by basal pneumonia of the other. Double apical pneumonia is very rare.

A third site, still more seldom selected by the pneumonic process, is the deep part of the lung near its root. This “central pneumonia” is naturally difficult of detection before it has advanced towards the surface.

Occasionally the anterior tongue-like process of the left lung is alone affected on that side.

Right pneumonia is rather more frequent than left, whether it affect base or apex, and unilateral is happily more common than double pneumonia. (See Appendix, § 4, p. 132.)

1 It happens that in the cases collected by the writer there were rather more of the left than of the right base (151:187), but in larger numbers the proportion is reversed.
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Anatomical Events.—In the majority of the cases pneumatic inflammation ends by resolution. The exuded blood-discs, and leucocytes, and fibrin are no longer expectorated as such, but degenerate and are broken up into granules, chiefly fatty in nature. These are partly mingled with the bronchial secretion and coughed up, but probably a larger proportion are absorbed by the lymphatics, conveyed to the veins, and finally excreted. The abundant salts (chiefly sodium-chloride) deposited in the inflamed lung are rapidly carried off by the veins, and discharged in the urine. The air re-enters the minute bronchi and air-vesicles as they re-expand in inspiration. The lung more or less rapidly regains its spongy character, and the pleural exudation is gradually absorbed. Once only have I had the opportunity of seeing a hepatised lung in process of recovery about ten days after the crisis; it was a case in which death occurred from another cause. The tissue contained air, exuded frothy serum, and floated in water; it was anaemic rather than congested, did not break down under pressure, and was very oedematous. As above stated, we learn from physical examination of the chest that the hepatised lung sometimes continues solid for several days after the raised temperature and other febrile symptoms have disappeared.

In fatal cases we may find any stage of hepatisation, as above described, and any combination of these stages. There is no reason to conclude that gray consolidation, or even purulent infiltration, is incapable of resolution; but the contrary opinion is obviously almost as difficult to prove as to disprove.

When the lung has passed into gangrene the surface is obscured by a cloudy film; the tissue breaks on the slightest handling, is of a very dark colour, and emits a characteristic foul and pungent odour. Under the microscope no air-vesicles or other structural forms are distinguishable; the organ and the exudation are alike dead and disintegrated, and blood-vessels and elastic tissue alone remain. Gangrene is always limited in extent, but perhaps never by a separating capsule. In many cases, probably in most, the cause of the gangrene can be ascribed to the presence of particles of decomposing food which have gained an entrance to the air-passages in the last day or two of the patient’s life.

It has been stated by Addison, and by other pathologists, that occasionally a pneumatic lung never recovers its permeability to air, and may gradually pass into an unnaturally firm, pale, solid state, containing an excess of fibrous tissue, a state which has been included by some writers under the head of cirrhosis. Addison described it as “marbled induration.” This condition must be a very rare one, and it does not appear to lead to the contraction of tissue or the dilatation of bronchi which mark Corrigan’s cirrhosis.

Equally supported by credible authorities, both past and present, is the statement that pneumatic hepatisation may end in abscess of the lung. Of this, as of the previously mentioned condition, I have no experience, and without denying the occurrence of either,

1 Among the former may be mentioned Stokes, among the latter Osler.
would regard this also as an extremely rare event. The "abscesses of the lungs" of the physicians of the 17th and 18th centuries were tuberculous vomicae. Multiple pyemic abscesses, now happily seldom seen, are well-known morbid conditions, but they do not follow true pneumonias. A small circumscribed empyema following pneumonia may burst and simulate an abscess during life, and may even be misinterpreted after death. And when the stage of purulent infiltration has been reached, it is not difficult by pressure or accidental laceration of the rotten tissue to produce a cavity filled with purulent fluid not at all unlike an abscess. These facts are, however, familiar to the able pathologists who describe abscess as a not infrequent event of (acute, lobar, fibrinous, or "croupous") pneumonia, and therefore its occurrence cannot be denied; but subsequent experience has certainly confirmed the observation of Laennec—"Il n'y a pas de lésion organique plus rare qu'une véritable collection de pus dans le tissu pulmonaire" (22, § 192).

Other organs.—In an autopsy on a case of primary pneumonias we expect to find the lungs only diseased, and an exception is rare. There will always be lymph on the visceral pleura, and sometimes pleuritic effusion, serous or purulent. The bronchial lymph-glands are soft and swollen. Occasionally the pericardium or endocardium, the meninges, larynx, or large intestine may show the lesions above described.

The blood in pneumonia when drawn coagulates slowly, and forms an abundant and firm clot with the "buffy coat," due to the red discs having subsided before becoming entangled in the meshes of the fibrin as it forms. This condition (so-called hyperosmosis), like the pneumonic pulse and fever, is present in many other inflammatory diseases. The leucocytes of the blood are also considerably increased in number, a fact first observed by Piorry, confirmed by Virchow, and more recently by Billings.

Bacteriology.—Klebs, in 1877, was the first to describe a microbe characteristic of pneumonia; but it was probably not the same as that afterwards discovered by Friedländer, and certainly not the same as Fränkel's diplococcus. The former was described by the late Dr. Friedländer in 1882 as an oval body 1 μ in length, occurring in pairs (diplococcus) or in chains (streptococcus); and, as was soon after ascertained, enclosed in a thick transparent envelope. On cultivation in gelatine it forms a colony of a characteristic nail-shape. When inoculated it produced pleuro-pneumonia in mice, and often in guinea-pigs, but not in rabbits. Accordingly it was named Pneumococcus (15).

It was soon ascertained, however, that this organism is not a coccus, but a bacterium or bacillus; that it is not invariably present in pneumatic lungs or sputa; and that it is often to be found in the mucus of the nose and bronchi, in the absence of pneumonia.

In 1884 Dr. A. Fränkel brought forward another claimant to the distinction—a microphyte, also oval or lancet-shaped, also occurring in pairs, also encapsuled, but differing in the conditions and results of successful cultivation. It also produced death in rabbits (but not pneu-
monia), and pneumonia in mice and guinea-pigs (14). The same microbe, in all likelihood, had been independently detected in hepatised lung by Talamon (30); and this again was the streptococcus observed by Dr. Sternberg of the U.S. Army in 1885, named by him "Microcococcus of Pasteur," and identified by many observers with that of sputum-septae, and with a similar organism occurring in healthy human saliva—"Bacillus sputigenus," "Bacillus salivarius" (26).

The extended researches of Weitshelbaum in Vienna on 129 cases of pneumonia (in the wide sense of the term) resulted in the discovery of Fränkel's diplococcus in 94, of Friedländer's "pneumococcus" or "bacillus-pneumonia" in only 9, streptococcus pyogenes in 21, and staphylococcus pyogenes aureus in 5 (33). This appears to show that while one microbe is most frequently present, others are competent to produce "pneumonia"; but, unfortunately, it is not clear what precise kind of pulmonary disease was present in the several instances.

The experiments of Gamaleia in Pasteur's laboratory led him to believe that the pathogenetic organism is to be found in the diplococcus described by Fränkel (now often called pneumococcus, in succession to the title enjoyed for a short period by Friedländer's bacillus), which, however, he identified with that of Talamon and Sternberg, and calls "Streptococcus lanceolatus Pasteuri" (16).

Lastly, we must remember the mobile rod-shaped microbe which was found by Dr. Klein (20) in the epidemic pneumonia of Middlesex.

While recognising the interest and importance of these laborious researches, we must observe that even the diplococcus of Talamon and Fränkel does not fulfil Koch's three tests of a pathogenetic organism; as they are fulfilled, for example, in anthrax, relapsing fever, and tuberculosis. It is not invariably present in the tissue of pneumatic lung; it does occur in other situations in health; when injected as a pure cultivation it does not always reproduce itself and cause a fresh case of hepatisation of the lungs. Nevertheless the frequency of its occurrence, and the fact that it often reproduces the disease by inoculation, make it probable that it plays an important part in the natural history of pneumonia.

There are, indeed, two other and preliminary postulates which are no less important than the three of Koch, but which have not always been kept in view in these or in similar investigations.

The first is that the microbe of which the action is under investigation shall be "specific"—that is, it shall be possessed of definite and constant characters; it must be a good botanical species which can be identified beyond dispute. The mere shape is admitted to be illusory. The same micrphyte varies in different stages of its growth and under different conditions, and may almost arbitrarily be described as a micrococcus, a diplococcus, or a streptococcus; as an oval or lancet-shaped bacterium, or as a bacillus. The presence of a capsule is not a constant distinction; and even the reaction to staining agents and the
shapes assumed under various methods of cultivation, are not always
decisive.

But if a certainty that different observers are discussing the same
microbe is essential before their results are compared; it is no less essen-
tial that they should be agreed as to the disease which is under inves-
tigation. Septic pneumonia, lobular broncho-pneumonia, and lobar fibrin-
osus ("croupous") or vesicular pneumonia are different pathological
conditions; and unless they are carefully discriminated, statistics of
"inflammation of the lungs" are as useless as would be similar facts with
regard to "inflammation of the joints" or "inflammation of the skin."

Pathology.—Looking back at the natural history of the disease now
described, to what conclusion concerning the nature and essential characters
of pneumonia and its relation to other disorders are we led? Are we,
like Laennec and his successors, to regard it as an acute inflammation of
the lung, of which the pyrexia and other symptoms are only the effects?
Or are we, with most modern writers, to look on the local lesion as but
one element in a specific infective fever? Or is any alternative opinion
open to us?

In the first place, the attempt, which has more than once been made,
to disprove the inflammatory nature of pneumonic hepatisation has
certainly failed. We have the hyperaemia and softening of the tissue
which are the characteristic signs of acute inflammation everywhere; we
have the pyrexia and other febrile symptoms which also attend acute
inflammation; we have the exudation of liquor-sanguinis, with leucocytes
and fibrin; the last stage of the process is purulent infiltration of the
affected lung, and, lastly, in every case of pneumonic hepatisation we
find the obviously related pleurisy, which no one can deny to be
inflammation.

It is true that pneumonia cannot be produced by injury, or by
ordinary irritants, as is proved by experience in men and experiment
on animals. The few supposed cases of pneumonia of traumatic or
irritative origin are mostly accidental coincidences. It does not arise by
extension from capillary bronchitis, nor by the irritation of dust, nor by
the more specific stimulus of the tuberculous microbe; but this only
shows that the process is a special, peculiar, or, as we say, specific kind
of inflammation. Much the same is true of the tubal nephritis of acute
Bright's disease, which is no doubt inflammation, but inflammation of a
peculiar kind; not traumatic, or irritative, or septic.

Admitting, however, that pneumonia is truly inflammation of the
lungs, are all its symptoms due to this local inflammation? This cannot
be maintained, for occasionally we find the symptoms precede the signs
of hepatisation by several days; the temperature and even the number
of respirations fall when the crisis comes, long before the solidified lung
has cleared up; the violence of the fever is not in proportion to the
extent of the local lesions, although the amount of lung involved does
produce its direct effects on the heart.

On the other hand, the differences between pneumonia and the
group of diseases to which typhus, small-pox, and measles belong must not be overlooked. It resembles them in its sudden onset, in its pyrexia, its definite course, and its limited duration; but in the vast majority of cases it is sporadic and idiopathic. It does not in common experience arise from previous cases, nor produce fresh ones; it does not protect from future attacks, but rather disposes to them. Although epidemics of pneumonia undoubtedly occur, they are local, and the most striking cases are those which follow influenza, a true specific fever, just as nephritis follows scarlatina. Moreover, unlike the local lesions of enteric fever or of mumps, hepatisation of the lungs is found in the course of other fevers, and of chronic affections such as Bright's disease. Against these broad differences the fact of a distinctive microbe being present is inconclusive; for, not to insist on the absence of any such microbe in some of the most definite specific fevers, its presence is not constant in pneumonia. It is associated with other microbes, strepto- and staphylococci, and is sometimes replaced by a bacillus which Dr. Klein found constant in an epidemic of pneumonia at Middlesborough in 1889 (vol. i. p. 658). The diplococcus, which is almost constantly present, is sometimes found in lobular pneumonia, sometimes in pleurisy without pneumonia, and sometimes in healthy saliva.

The fact seems to be that in this case, as in so many others, the phenomena of disease cannot be fitted into current classifications.

Inflammation itself is a much more variable condition than it was once thought to be. It differs as the cause which produces it, and as the organ or tissue which reacts to the irritant. Bacterial diseases differ also among themselves. The constancy of the presence of a peculiar bacillus in phthisis is most important, but phthisis remains a very different disease from tuberculous meningitis, or pulpy disease of the knee; while diphtheria, erysipelas, gonorrhoea, and relapsing fever differ in almost every particular except in respect of a specific microbe. At present our knowledge is too imperfect to decide the true nature, origin, and pathology of pneumonia; but we can affirm that, on the one hand, it is not an ordinary inflammation produced by injury, or mechanical irritants, or cold, with symptoms directly proportionate to the extent of the tissue inflamed; and that, on the other hand, it differs from such specific fevers as measles, enterica, and small-pox in that, though self-limited and definite in its course, it is not strictly "specific," not contagious, and often not idiopathic but secondary.

We may admit that the presence of a special microphyle is nearly constant in the disease; so that its presence in the sputum, like that of Koch's vibrio in cholera-stools, is a useful means of diagnosis: nevertheless, the disease is a local inflammation; not a common inflammation produced by common irritants but an inflammation peculiar to the lung, and incapable of artificial reproduction. Of many, perhaps of most organs of the body we may say that they are capable of three kinds of inflammation at least: that which is acute and supplicative, marked by abundant exudation of leucocytes, by the presence of some
forms of staphylococcus or streptococcus, and accompanied by what used to be called constitutional disturbance; secondly, a chronic interstitial inflammation with hardening of the tissue, shrinking and destruction of its peculiar elements by the new inflammatory growth; and, thirdly, an acute form of inflammation, not suppurative and peculiar to each organ. Pneumonia belongs to the last of these classes, and may be compared with acute Bright's disease, acute atrophy of the liver, and acute myelitis.

Of late years attention has been directed by Dr. Washbourn and others to the possibility of cases of acute pleurisy depending on the presence of the specific diplococcus of pneumonia, and running a short febrile course with the clinical features of pneumonia, but without the physical signs of hepatisation of the lung. If in such cases the lung really remains unaffected, and is found after death anatomically sound, while the pleura is the seat of a specific diplococcus inflammation, our notion of the disease pneumonia will be much modified; we shall have to admit it without reserve among the specific fevers, and to regard the inflammation of the lung as a very frequent but not a constant lesion.

Causation.—Of the causes of pneumonia we are still ignorant. Unless we disbelieve careful observations, because they do not fit a hypothesis, pneumonia can exist without the pneumococcus; and the same microbe may be found, not only in disease without pneumonia, but under healthy conditions also. Whether under certain circumstances this usually innocent microphyle acquires virulence, or whether a noxious and a harmless parasite resemble each other too closely to be distinguished, we cannot yet say; nor can we define the exact conditions which favour the occurrence of the disease. It is certain, however, that pneumonia is connected with cold, with north winds, with high ground, and with sudden fall of temperature.

Excluding bronchitis and ophthalmia, in which cold air seems to act as a direct common irritant, we may say that pneumonia affords the best evidence that there is truth in the common belief that a chill may "strike inward," and lends probability to the view that other acute inflammations—as pleurisy, colitis, acute Bright's disease, and even myelitis—may sometimes be due to a similar proximate cause.

We must also recognise as occasions of pneumonia certain previous morbid states, of which the most striking and important is influenza. Pneumonia following this disorder forms an important link between primary idiopathic pneumonia and the secondary pneumonia of fevers and septicemia. Of the latter group of diseases, pneumonia appears to occur most frequently in typhus, less frequently in enteric fever, and in certain epidemics only of relapsing fever. It is not common in fatal cases of scarlatina or variola, and seldom takes the place of so-called lobular pneumonia in measles, whooping-cough, or diphtheria. In rheumatic fever its occurrence is happily infrequent. Among chronic diseases it is perhaps most frequent towards the termination of Bright's disease; but certainly it is not nearly so common as acute oedema of the
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lung, or pleurisy, or pericarditis. Pneumonia is probably more common, and is certainly not less fatal in the latter stages of diabetes. Beyond these there is perhaps no chronic disease in which its occurrence is sufficiently frequent to be of etiological or practical importance; but pneumonia is one of the intercurrent maladies which bring invalids to their end.

It is important to notice that lobar pneumonia is very rare as the consequence of phthisis, or of bronchitis, whether acute or chronic.

Natural history and incidence, distribution and local prevalence.— Pneumonia, as we see it in this country, is a sporadic and endemic disease. In its primary form it appears to be more common in winter and spring.

It is common all over temperate Europe, in the United States, and in the inhabited parts of the South Temperate Zone—in Australia and New Zealand, at Buenos Ayres, and in South Africa. It is less common in the Tropics; but on the hill stations of India it is far from infrequent during cold weather. It is also common in the highlands of Central Asia. In Cabul and Beloochistan it is ascribed, as in Italy, to the sudden change from the scorching heat of the day to the severe cold after sundown.

Pneumonia sometimes occurs in an epidemic form. From the Middle-Ages downwards we have accounts of acute epidemic disorders, which seem more like pneumonia than anything else; and from time to time circumscribed epidemics are still reported in this country and in other parts of Europe. There is, however, no instance of prevalence of the disease so widespread as that of plague or cholera. All that happens is that a disease never uncommon becomes more common at certain times or in certain localities. This prevalence can sometimes be referred to a coincident prevalence of cold, dry winds, and sometimes is confined to a particular locality; but on the whole the phenomena do not seem to be of a different order from those which at certain seasons determine a greater prevalence of rheumatic fever, or quinsy, or bronchitis, or diarrhoea. What is more important is that we sometimes find a group of cases of pneumonia occurring together in the same village or in the same house. When influenza is epidemic, pneumonia follows it so often as to simulate an epidemic.

Sex and Age.—Pneumonia affects men more commonly than women—a preference usually explained by the greater exposure of men to changes of weather (Appendix, § 1).

No period of life is exempt from lobar pneumonia. It is, however, rare in infancy; and it is less common in children than in adults (Appendix, § 2).

It must be remembered that these facts concerning the incidence of pneumonia refer to the primary disease. Very little is known of the conditions under which pneumonia supervenes as a fatal complication of other diseases.

Prognosis.—There are few diseases of which the forecast varies so much with circumstances as it does in lobar pneumonia. Speaking broadly, primary pneumonia is much less dangerous than secondary; and in cases
of primary pneumonia the danger increases, first, with the extent of the lung involved; secondly, with the age of the patient.

Age.—The latter point is one in which pneumonia agrees with most acute specific fevers—particularly with typhus, enterica, and variola. In this point of increasing danger with increasing age pneumonia is in striking and instructive contrast, not only with scarlet fever, but also with rheumatism and with diabetes.

Lobar pneumonia is rare in infancy and difficult to distinguish from extensive lobular catarrh; not only during life, but sometimes even after death. When present, however, as it often is in children between two or three and thirteen or fourteen years of age, lobar pneumonia is a most satisfactory disease to treat. Its symptoms and physical signs are well marked; the fever is high, and the condition of the patient threatening, but, with few exceptions, the temperature will fall on the fifth or sixth day, if not earlier, the symptoms will rapidly subside, and the recovery of the patient will be as safe and permanent as it is rapid.

With girls and boys between fifteen and twenty the extent of pneumonia is commonly greater than at a younger age; and the disease is more often severe and prolonged; but the prognosis is nearly or quite as good. Between twenty and five-and-thirty or forty the pneumonia of young adults is still of good augury on the whole; but at this time of life it is more frequent for both lungs to be affected. Intemperate habits begin to weigh in the scale, and cases of pneumonia, secondary to rheumatism, to influenza, to disease of the cardiac valves, to Bright's disease and to diabetes, begin to bring down the proportion of recoveries; although it is still high for idiopathic cases. After the age of forty acute pneumonia is always a serious disease. The prognosis in primary cases depends upon the temperate habits of the patient, on his being neither over-fed and obese on the one hand, nor, on the other, under-fed and enfeebled by want and misery; on his being early put to bed under judicious treatment; on the extent of pulmonary tissue invaded, the height of the fever, the presence of cyanosis, and the effect of the pulmonary obstruction upon the heart. Secondary pneumonia at this age is a more dangerous disease than at the earlier period of adult life; but we see recovery from it in cases of enterica, of rheumatism, and even of Bright's disease and diabetes.

In old age pneumonia is a very fatal malady. As a secondary complication it frequently decides the termination of fevers, of chronic disease of the kidneys, of internal carcinoma, of lingering cases of hemiplegia, and other chronic afflictions of the brain and spinal cord. Even primary pneumonia, limited to a single lung, is dangerous in an aged patient, and we see recovery after the age of 70 with surprise. It does however occur, and sometimes even at so advanced an age as 80, but the cases are very rare.

Extent.—With respect to the area of pulmonary tissue affected, it is well known that double pneumonia is of graver prognosis than single (Appendix, pp. 133, 136). Pneumonia affecting the whole of one lung, from
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base to apex, though serious enough, is probably less so than that which affects a part of both bases. Apical pneumonia is more common in children than in adults, and probably for that reason is believed to be less dangerous. If, after one lung has partly or entirely cleared, pneumonia attacks the opposite one, the prospect of recovery is better than when both are hepatised at the same time.

Prognostic complications.—Of the conditions which affect the prognosis of pneumonia, probably the most important, apart from the age of the patient and the local extent of the disease, is intemperance. Such patients almost invariably become delirious, and the combination of delirium tremens and pneumonia is almost always fatal. Scarcely less serious is the presence of chronic renal disease; and next in gravity is that of valvular cardiac lesions. These two conditions, however, make the disease no longer primary but secondary.

Of the complications of idiopathic pneumonia, much bronchitis, and particularly bronchitis with bronchorrhea of the unaffected lung, is perhaps the most serious. Pleurisy, or even purulent effusion, is much less important; but pericarditis is a not infrequent and an extremely dangerous complication. Double pneumonia with pericarditis is almost invariably fatal (Appendix, p. 189).

A weak sound of the heart and a very rapid pulse are well-known indications of danger. Cyanosis, with very frequent breathing and action of the alae nasi, is equally grave; and the two conditions often go together. They are partly due to the mechanical obstruction to the lesser circulation, and partly to the direct effort of the high temperature on the cardiac muscle; but in many cases both are aggravated by more or less pronounced aspremia (24). Tympanites is a very unfavourable symptom in pneumonia, as in most other illnesses; so also are hicouche and subnatus tendinitum. When delirium persists during the day, and above all when it prevents sleep, the symptom is a grave one, and calls for decided treatment. Very high temperature indicates a severe case, and often goes with failure of the heart; but of itself hyperpyrexia is seldom fatal (Appendix, p. 140). Febrile albuminuria, even when abundant, is not of bad omen, and seldom or never persists after recovery. Frey rusty expectoration or even hæmoptysis need give no anxiety. Diarrhea is neither common nor dangerous, and if necessary it can be controlled without difficulty. Sweating during the height of the disease is not common, and when present is not of bad import. A profuse sweat, an abundant discharge of urine, or a sharp attack of diarrhea, sometimes accompanies the critical fall of temperature.

Treatment.—Historical sketch.—Acute pneumonia is so striking and so severe a disease that as soon as it was definitely recognised it was attacked by all the resources of medicine. During the whole of the present century the treatment of pneumonia has reflected the various theories of disease and the changing practice of therapeutics.

The conception of pneumonia current at the end of the last century was that of an acute inflammation, directly produced by cold, and
attacking a healthy subject. The business of the physician was to combat the enemy by the potent weapons of bleeding, blistering and starvation, aided by purgative and alterative drugs. The high fever, the flushed face, the acute pain and the burning skin were evidence of a “stenic” inflammation. The physician felt confident that by antiphlogistic remedies he could subdue the disease; and his only fear was lest the patient's strength should fail under the necessary treatment, that he might die, not of the disease, but of the weakness attending its cure—mort guéri. The “corroborant” practice of the Brunonian school of medicine never obtained such vogue in England as on the Continent. It was as baseless as the iatro-chemical or the iatro-mechanical systems which prevailed earlier in the 18th century, and had deservedly fallen into disrepute. During the first half of the present century the antiphlogistic treatment of pneumonia and of other acute inflammations continued to be the only one followed in civilised countries—in Dublin as well as in Edinburgh, in Vienna as well as in Madrid; and precisely the same treatment was adopted by surgeons for compound fractures, inflammations of the eye, and for what we now call pyemia. To realise the confidence and energy with which this absurdly called “heroic” treatment was carried out, one must have seen, as I saw so late as 1863-64, the treatment not only of pneumonia and pericarditis, but of rheumatic and typhoid fever, by Bouilland at the Charité; or one must read the lectures of the late Dr. Peter Latham (1845), in which with admirable rhetorical skill he enforces the dogmas of the day.

The only important modification of the antiphlogistic treatment of pneumonia introduced during the period between 1790 and 1850 was the introduction of large and repeated doses of antimony by Rasori (1808), a practice much followed for a time both in Italy and France. The undoubted effects of this drug, in producing nausea and disinclination to food, lowering the blood-pressure, and causing diaphoresis, were quite in harmony with the effects of bleeding, purging and salivation.

It was and still is true, when a patient is suffering from acute pain in the side with fever and a frequent, strong, and hard pulse, that venesection and free purging will relieve the pain, reduce the arterial tension, and give him grateful relief from his sense of fulness and oppression. It was no doubt from observation of these effects, which were well known to Sydenham, Mead, and Boerhaave, that the antiphlogistic practice began; and when the discoveries of Laennec made it possible to recognise pneumonia from the first, and to trace its daily progress, it seemed right to continue and to reinforce the treatment apparently so appropriate.

The mistake lay in having no control-observations. Physicians saw patients in an illness apparently desperate, and under treatment by bleeding and antimony they saw most of the symptoms relieved; frequently, after a battle of several days, the disease was subdued and the patient convalescent; but they did not know, because they never ventured to try what would happen if these remedies had been omitted. It is a humiliating but instructive fact that the possibility of recovery from acute
disease without active treatment was established by the assumed success of a demonstrably futile system of therapeutics, the last, we may hope, of attempts to answer the absurd question, "On what universal principle should disease be treated?" When it could not be denied that persons suffering from pneumonia and other acute disorders did recover when treated with infinitesimal doses of useless drugs, it could not be long doubted that some acute diseases might get well of themselves.

The report of some cases of pneumonia which recovered in the Homoeopathic Hospital at Vienna awakened thought on this subject, and an article by the late Sir John Forbes, which appeared in the British and Foreign Medical Chirurgical Review (1846), pressed the lesson home. Skoda had given fair trial to other methods of treatment, and found that his mortality from acute pneumonia was much less than when treated by bleeding, blisters, and antimony. These facts were made known in England by Dr. Geo. Balfour, who had followed Skoda's practice in Vienna (6). Dr. Hughes Bennett of Edinburgh also published a series of cases of pneumonia treated without bleeding, antimony, or mercury with unusually small mortality (1848); and he gave an interesting account of the arguments of Alison, Watson, Christison and Markham (8). Discussion followed, but it was less prolonged than might have been supposed; as so often happens, general opinion had been gradually altering, and was ready to turn at the first summons. Moreover, the advocates of antiphlogistic treatment threw away their case by the assertion that they were right in bleeding before, and right in doing nothing afterwards—not because their opinions but the nature of the disease had changed; and a presumed "sthenic type" of fevers and inflammations, with a successful heroic treatment corresponding thereto, was dwelt upon with the same satisfaction that an old man contrasts the hard frosts and heroic exploits of his youth with the mild winters and feeble powers of his contemporaries. For a long time the antiphlogistic treatment held its ground in books and lectures; but those who taught it always found in practice an excuse for disobeying their own precepts. By 1860, however, the change in treatment was nearly universal; and during the latter half of the 19th century, English physicians, under the guidance of Jenner and of Gull, have given up the "heroic" treatment of pneumonia.

In too many cases the treatment which supplanted it was of a purely negative kind, disguised under such platitudes as the prescription of rest in bed for a patient who could neither sit up nor rest; of light and nourishing food, as if the opposite was ever ordered for a fevered man; and of avoiding cold for a patient with a temperature of 104°.

At present we may hope that a more rational system is established. We know that under favourable circumstances pneumonia needs no treatment beyond the following of the indications of the patient's own feelings, and awaiting the favourable result which will follow in the course of a week.

Abortive treatment.—It is clear that no means known at present can cut short pneumonia. There is nothing absurd, however, in supposing
that this may one day be done. We do cut short the manifestations of syphilis and ofague, of hydrophobia, and of diphtheria.

Since the presence of microbes has been ascertained in pneumonia, and the pathogenetic significance of the diplococcus of Talamon and Fränkel has been admitted by physicians with more or less confidence, it is not surprising that attempts have been made to apply the theory of immunity in treatment. The method adopted has been to render animals immune from the disease by introduction of the supposed pathogenetic microbe in doses of increasing strength until this immunity is attained, and then to inject the serum of such an animal into the veins or tissues of patients suffering from pneumonia. This practice was introduced by F. and G. Klemperer in 1891, and has been followed by some apparent success (21).

Whether pneumonia can be cut short or not, it may be successfully guided. No reasonable observer would deny that, although we are rarely able to say that a patient's life was saved by such and such a timely measure, yet in the long run the expectant method of treatment, which interferes only as occasion requires, is followed by a far lower mortality than misplaced attempts to "jugulate" the disease, or than a completely negative treatment.

General treatment of uncomplicated cases.—In a case of primary pneumonia in a young subject our first care should be to keep him cool by light covering, cradles under the bedclothes, and frequent sponging with cold or tepid water. He has no appetite, and there is no necessity to force food upon an unwilling and often flatulent stomach. If the patient will drink two pints of milk, or one pint of milk and one of broth, in the twenty-four hours, he will not starve. Stimulants should not be given until required by some special indication.

The thirst, the parched tongue, the fever, the scanty and concentrated urine, and the hot, dry skin all call for drink; and the patient should be allowed to take as much cold water as he pleases. It relieves his thirst, it moderates the sensation of heat, it flushes the kidneys, by inducing perspiration it relieves discomfort, and by evaporation it helps to lower the temperature. If patients prefer effervescing drinks they may have them, and milk, with soda water is often the pleasantest mode of supplying nourishment; but it must be remembered that milk is food, and to keep a pneumonic patient on milk and beef-tea without water is a practice as unphysiological as it is disagreeable. The exception is when a child refuses nourishment and will only drink milk when compelled by thirst. Toast and water, barmy-water with or without a slice of lemon, tamarind or red-currant water may be given according to the patient's preference; and, although the cold and unadulterated element is as a rule most grateful, some patients, particularly if troubled by gastric disturbance, much prefer to drink hot water. There is no reason for withholding tea as a beverage; but this and other indulgences the patient will probably enjoy the more if not given until asked for. Oranges or grapes are more pleasant in convalescence than
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during the height of the disease; but there is no objection to their use at any time.

With regard to drugs, though an uncomplicated case of pneumonia will do well without them, yet long and wide experience shows that solutions of neutral salts are of service in diminishing the sense of heat and tension, and in promoting secretion. Nitre is perhaps the best of these; but citrate or acetate of potash or acetate of ammonia act in a similar way; the potash salts are supposed to act most on the kidneys, and those of ammonia on the skin. They may be given with chloroform or peppermint, or in any bitter infusion such as serpentine, orange, or quassia. They are not necessary, but beside their undoubted, though slight, physiological effects, an occasional draught of medicine is liked by most patients, and it helps to keep up the attention of the nurse.

It is an old custom, and perhaps a wise one, to administer a purge at the beginning of any acute disease. The furred tongue, the headache, and the customary constipation seem to call for it; and it helps to prevent flatulence and so to favour respiration in the course of pneumonia. A blue pill, followed by a black draught—or, what is much pleasanter and nearly as efficient, by sulphate of magnesia with bicarbonate of soda in a carminative vehicle, or some other natural or artificial solution of purgative salts, are the best ways of meeting this indication. An efficient action of the bowels on the first day of the disease has the important advantage of setting the mind of the patient and of his nurses at rest, and of preventing unseasonable purging afterwards.

From the first day constant attention should be directed to the chart of temperature, to the pulse and respirations, and to the auscultatory signs. The chest should be thoroughly examined on the first or at least on the second day; and, when the diagnosis and seat of the disease are thus made clear, the frequency and minuteness of subsequent examinations may depend upon the patient's condition. It is often wise to refrain from rousing a patient from sleep for this purpose, or from exciting the resistance of a child. In such cases, or when delirium or extreme weakness forbids a thorough physical examination, we may generally judge of the condition of the lungs by counting the number of respirations, by noticing the colour of the face and lips, and by observing the action of the nostrils and muscles of forced respiration. By gently rolling the patient on his side, first one and then the other, we can obtain sufficient evidence of the state of the lung, by means of the flexible stethoscope, without raising him in bed.

Treatment of special symptoms.—We now come to the treatment of symptoms, ordinary or extraordinary.

The temperature is always or almost always high, and hyperpyrexia is frequently met with; but it is less common than in rheumatism, typhus, or scarlatina, and its danger is certainly less than in rheumatism. A temperature of 104° demands attention, but in children and young adults does not in itself require interference. When it rises above this point
a tepid bath for a child, and sponging the body with cold water in an adult are indicated. In some cases it is more convenient to put bladders or India-rubber bags filled with ice in a man’s armpits and between his thighs. If the temperature rise above 105°, frequent sponging with ice-cold water, rubbing with ice, or immersion in a bath at a temperature of 80° is called for.

In some cases wrapping the patient in a sheet wrung out of cold water is more convenient or less unpleasant, and it is an efficient means of relieving moderate pyrexia; or Leiter’s tubes may be carried in coils round the head and placed over the great blood-vessels, above the collarbones, in the armpits, and in Scarpa’s triangle.

In children a high temperature is more frequent and less injurious than in adults; whereas in elderly patients a temperature above 103° is a serious matter, and cooling measures must be sedulously and yet cautiously employed.

Whatever the temperature which appears to call for interference (and in this we must be guided not only by the thermometer, but also by the mental condition and the state of the pulse), direct application of cold should be the treatment adopted. Antipyretic drugs are either inefficient or their action is transient; and they often cause dangerous depression of the heart’s action or complete collapse. Salicyl-compounds are only indicated when pneumonia occurs as a complication of rheumatism; and full doses of quinine only in the case of persons who have suffered from malaria. Antipyrin or phenacetin is sometimes useful for relieving severe headache; but even then they must be used cautiously. When headache is troublesome, the application of ice to the head often gives the greatest relief. A dose of bromide is sometimes efficient, or the aromatic spirits of ammonia, or a strong cup of coffee.

The pain of the pleurisy which always accompanies pneumonia varies greatly in its severity and duration. Often it is so slight that a warm poultice, or an ice-bag, or rubbing with menthol is enough to allay it. In severer cases two or three leeches are sometimes of striking benefit; or, if there be no counter-indications, the sixth or the fourth of a grain of morphia may be injected under the skin. When the pain is persistent, a blister relieves it more certainly than any other remedy.

The effusion of pneumatic pleurisy is seldom large enough to demand special treatment. If it should be considerable, it is best to aspirate at once; if it be small, it will often disappear of itself after the crisis, or may be dealt with during convalescence by blisters and diuretics or, if these fail, by paracentesis.

Empyema is the most important sequel of pneumonia. When its presence is discovered, it should be tapped at once, and afterwards incised and drained. It is said that if the diplococcus of pneumonia be present alone in the exudation, the pleura will recover without fresh secretion of pus; whereas if colonies of staphylococcus or streptococcus are found, it is better not to aspirate but to incise at once, or the
pus is sure to recur again: that the latter rule is true, at least for adults, is supported by general experience; the former statement is more disputable.

One of the most formidable complications of pneumonia is pericarditis. It is most common in cases of double pneumonia, with well-marked pleurisy, and may be chiefly fibrinous, or accompanied with large effusion of serum, or occasionally purulent. Its recognition is often far from easy. Marked orthopnoea, an irregular or intermittent pulse, and precordial distress are each of them valuable signs of pericarditis, but they are neither constant nor invariable. In every case of pneumonia the apex and base of the heart should be examined each day: from the former to learn the strength and character of the first sound of the heart, from the latter to detect the earliest sign of pericardial friction. This is difficult to make out when noisy and frequent breathing obscures the cardiac sounds; and the difficulty is often increased by the restlessness and distress of the patient. When it is impossible to obtain even a momentary pause in breathing, close attention to the pulse will impress the cardiac rhythm on the ear; and when this is once done, the practised auscultator can neglect the bronchial rales almost as much as the noises going on in a room. At least we may say whether the sounds are normal or accompanied by a murmur; and in the latter case, if we know that they were normal on the first day of the illness, the murmur is most likely a pericardial rub.

It is sometimes impossible to detect a large pericardial effusion by percussion. We must depend upon the situation and force of the cardiac impulse, on the faintness of the cardiac sounds, and on the signs of downward pressure of the left lobe of the liver. If called for the first time to a case of acute pneumonia with pericarditis we may find the diagnosis extremely difficult.

Our treatment of pericarditis, when recognised, is unfortunately not yet very efficient. There is no reason to believe that bleeding, leeching, or mercury has any effect on the inflammation. A blister, however, over the cardiac region will often relieve the precordial oppression; and twice I have seen it signally and demonstrably successful in removing the signs of friction and of effusion. In a severe case of pneumonia, however, blisters are undesirable; and if we believe that pericardial effusion is extensive it is probably better practice to introduce a hypodermic syringe at once and draw off a few drops of the fluid: if serous, the pericardium may then be aspirated; if purulent it should be incised and drained like an empyema. I have repeatedly tapped the pericardium, and have never seen harm to follow the practice; on the other hand, I have been unfortunate in not witnessing the marked relief which many physicians have recorded. Several most successful cases of draining the pyo-pericardium have been published.

The most imminent danger in pneumonia is dyspnoea from extensive consolidation of the lung, overloading of the right side of the heart, and arterial anæmia. We are at present powerless, or nearly so, to limit the
spread of hepatisation; all we can do is to help the patient to outlive the stress of the disease.

For this purpose frequent feeding with small quantities of nutriment is necessary. The most useful forms of nourishment are milk, raw eggs, beef-tea, and meat jelly, or one of the various meat extracts in the market. Food should not be given oftener than every two hours. When there is vomiting, or when the patient refuses food—as sometimes occurs in a child or in an adult who is delirious—it is best to abstain altogether from feeding by the mouth, and to use instead a nutriment enema or nutrient suppositories, after the rectum has been washed out.

In cases of secondary pneumonia, and in primary cases occurring in later life with few exceptions, alcohol is indicated, and in all cases when the pulse is irregular or very rapid, and the first sound of the heart weak. It may be given in the egg-and-brandy mixture of the Pharmacopoeia, or diluted with water as a beverage. Its effect should be watched, and the amount and frequency of its administration regulated by its effects. It often improves the pulse and soothes the brain; when these effects are apparent, it should be pushed freely. Half an ounce given every four hours is suitable for an uncomplicated case of pneumonia in a patient over fifty years of age. Six or eight ounces in the twenty-four hours are needed in severe cases with feeble circulation; and as much as twelve ounces when by the patient's symptoms he seems to demand it, and his state to improve under the remedy. In some cases champagne is better tolerated than brandy, whisky, or rum, and has as good or perhaps a better effect. Sometimes, however, we find that any form of alcohol causes excitement and discomfort without strengthening the pulse; in such cases it is best to omit it for a time, and to rely upon strong beef-tea and strychnine.

As the pulse affords the chief indication for the administration of brandy, so the state of the patient's breathing guides our use of ammonia. This admirable drug acts not only on the heart, but also on the respiratory centre, stimulating the reflex action of coughing, and so clearing the air-passage. Like other alkalies, its action on the bronchial secretion is to make it thinner and more easily got rid of. In all cases of pneumonia, except those affecting a single lung in children or healthy young adults, carbonate of ammonia should be given in four or five-grain doses combined with syrup of tolu, liquorice, or treacle, to soften its pungency. Compound tincture of cardamoms or lavender may be added with advantage, or the drug may be given dissolved in peppermint or chloroform water. It should be repeated every four hours or more frequently, up to five grains every hour, if dyspnoea and cyanosis threaten imminent danger.

There is always some bronchitis with pneumonia, and this may add considerably to the dyspnoea; but bronchitis is most serious when in a case of unilateral or extensive pneumonia it affects the healthy lung or the healthy part of one. It is in such cases, perhaps, that carbonate of ammonia is most signally useful.

In addition to brandy and ammonia, or in cases where one or the other
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seems to fail, we may use strychnine as an efficient stimulus of the centres in the bulb. It is most valuable in cases of failure of the heart’s action, when alcohol seems useless or even mischievous to the patient; five drops of liquor-strychninæ may then be given with excellent effect, and repeated should occasion arise. Strychnine is much more useful given in one or two full doses than in smaller ones frequently administered. Of course it must never be given with ammonia. When a pneumonic patient is at the point of death, three or four drops of solution of strychnine injected under the skin of the arm are more effectual, and less liable to lead to local abscess than brandy, ammonia, or ether administered in the same way; but each of these drugs thus exhibited has undoubtedly saved life.

In severe cases of pneumonia, the rapidity of the pulse, its frequent irregularity, and the low blood-pressure naturally suggest the use of digitalis. It is generally prescribed along with other remedies, and it is therefore difficult to estimate its individual effect; but my own experience has been disappointing, and my disappointment is shared by many physicians who have used it since Traube recommended it fifty years ago.

In pneumonia its effect is incomparably inferior to that which we see every day in mitral regurgitation, with dropsy and rapid, weak, irregular pulse. The experiments of Drs. Brunton and Cash (8) indicate that the effect of digitalis on the heart is greatly weakened by pyrexia. It has been conjectured that the right ventricle, the cavity most burdened in pulmonary obstruction, has not muscle enough to utilise the drug; but, if the objection were theoretically admissible, it is contradicted by the fact that it is this right ventricle which we successfully stimulate and control in cases of mitral regurgitation. Whatever the explanation, digitalis would probably be seldom employed if our only experience of it were in pneumonia. At the same time, it is right to add that some good observers, both at home and abroad, have a much more favourable experience of this powerful drug in the disease. Dr. Petrescu of Bucharest reports a remarkable low percentage of death in pneumonia treated with large doses of the powdered leaves or of the infusion of digitalis.

An ancient stimulant, which had long fallen into disuse, has been lately revived in cases of pneumonia, enteric fever, and other exhausting diseases; namely, muss, given in ten-grain doses, and repeated in three or four hours. It is very expensive, and often it fails entirely; but I have seen it produce striking improvement for a time in severe cases of bronchitis, pneumonia, and fever; and in four or five of these instances it probably saved the patient’s life.

When dyspnoæa is urgent, and the patient apparently dying of cyanosis, the inhalation of oxygen is a rational mode of treatment, and has been advocated for many years past. It is now possible to obtain the gas ready made in large iron cylinders much more conveniently and cheaply than before; and it sometimes proves remarkably useful. It seems never to do harm, and it is a matter of surprise that its effects are not more uniformly and obviously beneficial. It is possible
that we have yet to learn how to use it most efficiently. At present it takes its place among the adjuvants of successful treatment.

Delirium is an important complication of acute pneumonia; this is often best treated by an extra dose of brandy in the evening. In the case of an intemperate patient digitalis is here a valuable drug. Bromidé of potassium, or full doses of henbane or chloralamide, may be used with good effect. When coincident with high temperature, a wet pack or cold sponging is often the best cure for delirium.

Sleeplessness is a frequent and trying symptom. In some degree it is inevitable, and, when there is much bronchitis, to prolong sleep might be dangerous to the patient; but in other cases the insomnia is purely injurious, and must be met by every means in our power. In ordinary cases a cup of beef-tea with a spoonful of brandy is an excellent sedative; and if, before this is taken, the patient has been well sponged, furnished with a clean night-dress and a fresh pillow, if the head is kept high and as cool as possible, and the room dark and quiet, natural's sleep will often follow. Nothing is worse at such times than meddlesome nursing, moving about on tip-toe, conversing in whispers, and smoothing the patient's pillow. The administration of food or medicine may well be omitted for three hours, or even longer. In cases where this would be dangerous the patient is not likely to sleep too long.

The refreshment of sleep at some time of the day or night is of primary importance in pneumonia as in other fevers; and when the means above mentioned fail, and the patient has been sleepless for twenty-four hours, the question arises of exhibiting our most powerful remedy, opium. It has been forbidden lest its use should increase cyanosis, diminish respiratory efforts, and lead to fatal coma. These fears are far from groundless. When there is extensive consolidation or much bronchitis, when the patient is becoming livid, and the expectoration scanty, it would be bad practice to give this drug. Mustard plasters, strychnine subcutaneously, and ammonia by the mouth are the remedies indicated. Often during a whole night the patient must be restless, must continue to cough and expectorate, and must use every muscle available to keep himself alive by forced breathing. The orthopnoea, the coughing, the sleeplessness, the distress are all evidence of the struggle for life; and the worst sign is when a cyanotic patient lies low in bed, drowsy, with weak and shallow breathing, the respiratory centre in his bulb poisoned by carbonic acid. After such a restless night as we have just described the patient will often find the breathing relieved when morning comes; and, after taking a cup of coffee or a glass of wine, he may sink into natural slumber, and awake refreshed and ready to renew the struggle.

But in many cases of pneumonia the danger is not directly from suffocation; it is rather from the effects of a continued high temperature upon the heart, the impending weakness of the respiratory muscles, and exhaustion of the reflex activity of the nervous centres. In such cases 10 grains of Dover's powder, 5 grains of a compound soap pill, 15 or 20 drops of laudanum are often invaluable, and succeed when all other
hypothenics are powerless. An additional warrant for the use of opium is dilatation of the pupils. The presence of albumin, if only of pyrexial origin, is no counter-indication; but if the patient be a subject of chronic Bright's disease we must forgo the use of this valuable drug, or use it at his peril, to escape a still more imminent danger.

There are two remedies which have fallen into general disuse, but each of them worthy of being employed on occasion.

One is the use of emetics—antimonial wine, or ipecacuan in full doses, or sulphate of zinc, or subcutaneous injection of apomorphia, or a draught of mustard and water and tickling the fauces with a feather: such drugs, in emptying the stomach, also get rid of accumulated bronchial secretion, and produce deep and efficient respiratory effort. This method of treatment is still much used with children suffering from bronchopneumonia, for in them vomiting is easy and expectoration difficult. In adults a vomit and a purge no longer form a routine prescription; and, although no doubt an emetic is sometimes useful, it is an unpopular remedy, and its effects are often disappointing. Not infrequently even large doses of the emetic fail to excite vomiting, and the patient's condition is then uncomfortable to himself and embarrassing to the physician. A full dose should always be given, and in pneumonia and bronchitis stimulant emetics like mustard and sulphate of zinc are more suitable than anodyne. Though often disappointed in this plan of treatment, I have seen cases in which it was of undoubted benefit.

The other ancient remedy is that of bleeding. We saw that it was once employed to subdue fever, and to cut short inflammation, and that its use for these purposes is deservedly discredited. Venesection is less potent for good and also for evil than used to be thought, but it is not to be forgotten or neglected. Its effect in relieving the pain of aneurysm was insisted on by the late Dr. Hughes Bennett in the midst of his attack upon its use in pneumonia. The same iconoclastic reformer also recognised its value when used in the very first stage of pneumonia before dulness had appeared. It is not often that a patient is seen in this stage, which is usually very short; but when pneumonia occurs as a primary attack in a young and robust subject, with severe pleuritic pain, I would advise bleeding, not as a cure, but as a means of relief. If the pulse be full, strong and hard, and a great sense of precordial oppression be present, the withdrawal of 6 or 8 ounces of blood from the arm, by temporary lowering of the arterial pressure, will sometimes remove distress better than any other remedy, and will leave, if not a beneficial, at least no deleterious effect on the subsequent course of the disease.

In cases of cyanosis, with a small and feeble pulse, congestion of the surface, and distension of the right ventricle, as shown by epigastric pulsation and pulsation of the great veins, our object is not to lower the arterial blood-pressure, but to relieve the over-pressure in the right side of the heart and the systemic veins. The withdrawal of 10 or 12 ounces of blood under such circumstances is a rational procedure, and in practice is often successful in tiding over a dangerous
period of the disease. In my experience, however, venesection in the cyanosis of pneumonia is less strikingly useful than in the corresponding phase of bronchitis; although it is more so than in the cyanosis of valvular disease of the heart.  

In the treatment of convalescence from pneumonia we have fortunately little to discuss. Like typhus, and in striking contrast to enteric fever and scarlatina, acute lobar pneumonia is a disease which either kills the patient or leaves him much as he was before. When the crisis is past the inflamed lung very seldom fails to clear up rapidly and completely.

As soon as the temperature falls, brandy and medicine should be omitted or greatly reduced in amount and in frequency of administration. Sleep should be encouraged, and food given in accordance with the patient’s returning appetite. Wine is often useful during the first days of convalescence; or, if the patient prefer it, malt liquor may be taken with at least equal advantage. There is no danger of catching cold, and the patient may go out of doors in favourable weather as soon as he desires to do so.

APPENDIX OF CASES

The statistics subjoined refer to 434 cases of (acute fibrinous) pneumonia collected from the following sources: 329 schedules were filled up from the records of Guy’s Hospital during years 1891-94 by the medical registrar, Dr. J. H. Bryant, assisted by Mr. F. J. Steward, and to both these gentlemen I am greatly indebted for this valuable help. I have added 32 hospital cases of my own, admitted in 1895 and 1896; the remaining 73 were private cases seen in consultation. The number of cases is not large enough for all purposes, but it is, I hope, large enough to be of service for others.

The facts tabulated in the schedules were:—(i.) the sex, and (ii.) the age of the patient; (iii.) the assigned cause, the antecedents, and initial symptoms of the attack; (iv.) the part of the lungs affected; (v.) some of the chief symptoms, particularly the highest temperature attained, the presence and characters of the sputa, the presence of herpes, and (vi.) any important complications or sequels; (vii.) the duration of the disease, reckoned from the rise of temperature to the end of pyrexia, and, lastly, (viii.) the result in recovery or death, with details of the fatal cases.

1. Sex.—Of the 434 cases, 320 occurred in male, and 114 in female patients, a somewhat larger disproportion than usual. The difference is most marked in early adult life, least in children, and disappears in the statistics of prisons, where both sexes are under similar external conditions, and where epidemic pneumonia would affect both alike.

2. Age.—The following is the incidence of the 434 cases at the several periods of life.

(a) Under five years.—Total, 29 patients. Of these, 6 were above four years old; 9 were between three and four; 10 were between two and

1 See forty-nine cases of venesection in the Medico-Chirurgical Transactions for 1891.
three; 1 was eighteen months old; and 2 were under a year—one four and the other seven months old.

Between five and ten years there were 66 cases, making a total of 95 patients under ten years old.

Between ten and fifteen there were 45 patients, and between fifteen and twenty 48, making a total of 93 between ten and twenty.

(b) Arranging the figures in perhaps a more instructive way, we have 3 cases in infancy, 26 in early childhood (two to five), and 159 between five years old and adult age.

Between twenty and forty there were 149 cases.

Between forty and sixty there were 74 cases.

Between sixty and seventy there were 18 cases; showing a greater frequency than between fifty and sixty, if we allow for the fewer possible patients at the more advanced age.

Above the age of seventy there were 5 patients—2 aged seventy-two, 1 seventy-three, and 2 seventy-five.

These numbers confirm what is a matter of general observation, that lobar pneumonia is rare in infancy, very common between two years old and twenty, gradually less common in adult life, more rare after fifty. The cases in later life are mostly secondary.

(c) Arranged in decades the numbers are—

<table>
<thead>
<tr>
<th>Age (in years)</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-10</td>
<td>95</td>
</tr>
<tr>
<td>10-20</td>
<td>93</td>
</tr>
<tr>
<td>20-30</td>
<td>87</td>
</tr>
<tr>
<td>30-40</td>
<td>62</td>
</tr>
<tr>
<td>40-50</td>
<td>51</td>
</tr>
</tbody>
</table>

3. Origin and onset.—In 38 cases only was the attack of pneumonia explicitly attributed to a shortly preceding exposure to cold; a "chill" received more than a week before the illness began was not counted. In 14 cases the origin of the disease was imputed to an injury or "accident"; but I have never seen a case in which this supposition was borne out.

In 3 cases only was there a probability of infection from another case in the same house. Two of these patients were children; the third was a lady who was attacked with pneumonia while nursing her child with the same disease and while her husband was convalescent from it. Another patient, whom I saw with Dr. Charles Addison at Colchester, was one of no less than four cases of pneumonia in the same house; but in each of them the pneumonia had been preceded by influenza, so that the cases should come under another heading.

In the hospital cases it is likely that many more had their origin in influenza; but in only three was the sequence definitely recorded. Of 73 private cases, influenza had preceded the pneumonia in 8.

The onset of the disease was gradual in not less than 93 cases, an unexpectedly large number—more than a fourth of the whole. In the remaining 234, in which the early symptoms were clearly ascertained, the onset was sudden; it began with well-marked shivering in 95 cases, with vomiting in 50 (chiefly children), with convulsions in only one case (a
child), in the remainder with sharp pain in the side, or once or twice with syncope.

A previous attack had occurred in 18 cases—in 3 within a year, in 3 within two years, in 1 three years ago, and in 3 so long as fourteen, seventeen, and eighteen years before the second attack. One patient had suffered five or six times from the disease, two had a fourth, and one a third attack.

4. Locality.—The right lung was, as generally observed, more often affected than the left; but the difference was due to the large excess of right-sided over left-sided apical pneumonia. When the base only was affected, there were in my tables rather more cases on the left than on the right side.

Cases beginning in the right base

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>left</td>
<td>140</td>
<td>151</td>
</tr>
<tr>
<td>right apex</td>
<td>60</td>
<td>55</td>
</tr>
<tr>
<td>left</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>affecting the middle part of the lungs—right, 2 left</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>both lungs</td>
<td>55</td>
<td></td>
</tr>
</tbody>
</table>

Total number of cases affecting the right lung only

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>left</td>
<td>206</td>
</tr>
<tr>
<td>both lungs</td>
<td>55</td>
</tr>
</tbody>
</table>

Double Pneumonia.—The 55 cases in which both lungs were affected seem to deserve separate attention, particularly in their relation to age and to prognosis. The table explains itself.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Locality</th>
<th>Complicated by</th>
<th>Complicating</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.</td>
<td>31</td>
<td>Both bases</td>
<td>Tympanites</td>
<td></td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>43</td>
<td>R. base, L. apex</td>
<td>...</td>
<td>Chronic phthisis</td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>49</td>
<td>Both bases</td>
<td>...</td>
<td>Chronic phthisis</td>
<td>Died</td>
</tr>
<tr>
<td>F.</td>
<td>12</td>
<td>Both bases</td>
<td>Gangrene</td>
<td>Spinal caries</td>
<td>Died</td>
</tr>
<tr>
<td>F.</td>
<td>4</td>
<td>Both bases</td>
<td>...</td>
<td>Chronic otorrhcea</td>
<td>Recovered</td>
</tr>
<tr>
<td>M.</td>
<td>36</td>
<td>Both bases</td>
<td>...</td>
<td>...</td>
<td>Recovered</td>
</tr>
<tr>
<td>M.</td>
<td>49</td>
<td>Both lungs</td>
<td>...</td>
<td>Morbus Brightii</td>
<td>Died</td>
</tr>
<tr>
<td>F.</td>
<td>46</td>
<td>Both bases</td>
<td>Gangrene, Endocarditis</td>
<td>...</td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>6</td>
<td>Both bases</td>
<td>Empyema</td>
<td>...</td>
<td>Recovered</td>
</tr>
<tr>
<td>M.</td>
<td>36</td>
<td>Both lungs</td>
<td>Gangrene</td>
<td>Intemperance</td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>21</td>
<td>R. base, L. apex</td>
<td>Diarrhoea</td>
<td>...</td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>15</td>
<td>Both lungs</td>
<td>...</td>
<td>...</td>
<td>Recovered</td>
</tr>
<tr>
<td>M.</td>
<td>39</td>
<td>Both bases</td>
<td>...</td>
<td>...</td>
<td>Recovered</td>
</tr>
<tr>
<td>F.</td>
<td>18</td>
<td>Both lungs</td>
<td>Laryngitis</td>
<td>...</td>
<td>Recovered</td>
</tr>
<tr>
<td>M.</td>
<td>14</td>
<td>Both lungs</td>
<td>...</td>
<td>...</td>
<td>Recovered</td>
</tr>
<tr>
<td>M.</td>
<td>22</td>
<td>Both lungs</td>
<td>Empyema</td>
<td>...</td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>39</td>
<td>Both lungs</td>
<td>...</td>
<td>Admitted moribund</td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>8</td>
<td>Both lungs</td>
<td>Empyema, Pericarditis</td>
<td>...</td>
<td>Died</td>
</tr>
<tr>
<td>M.</td>
<td>17</td>
<td>Both lungs</td>
<td>...</td>
<td>Admitted comatose</td>
<td>Died</td>
</tr>
</tbody>
</table>
Summary.—Deaths, 30; recoveries, 25. Only one recovery took place among patients who were over forty years of age when attacked. This was in the case of a man aged forty-eight with pneumonia of both bases.

5. Symptoms.—Those which I particularly recorded were the temperature, the character of the sputa, and the presence of herpes labialis.

Albuminuria was frequently reported, and in cases when stated to be absent an earlier or more frequent examination might often have found it. Its presence from more or less latent Bright’s disease, from acute nephritis or renal embolism, and the occurrence of traces of albumin from leucorrhœa, cystitis, spermatorrhœa, or gonorrhœa virulenta would also disturb the results.
Temperature.—In two cases this is recorded as subnormal; both these patients were admitted into hospital in a collapsed and moribund condition, and died soon after being got to bed. In the rest the highest point observed was as follows:

<table>
<thead>
<tr>
<th>Degrees Fahr.</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>100–100·4</td>
<td>5</td>
</tr>
<tr>
<td>101–101·8</td>
<td>22</td>
</tr>
<tr>
<td>102–102·8</td>
<td>55</td>
</tr>
<tr>
<td>103–103·8</td>
<td>114</td>
</tr>
<tr>
<td>104–104·8</td>
<td>164</td>
</tr>
<tr>
<td>105–105·8</td>
<td>42¹</td>
</tr>
<tr>
<td>106 (bis), 106·4, 106·6</td>
<td>4²</td>
</tr>
<tr>
<td>107·8</td>
<td>1³</td>
</tr>
<tr>
<td>109</td>
<td>1³</td>
</tr>
<tr>
<td></td>
<td>408</td>
</tr>
</tbody>
</table>

Herpes.—A herpetic eruption was noted in only 53 patients. Of these, 46 recovered and 7 died.

Sputum.—Notice of this is not always explicitly made, and patients may have died too soon for it to be seen. Of the 290 cases in which definite statements were made, there were—

116 in which the sputum was "rusty."
7 " " " greenish."
24 " " " mucus or muco-purulent.
13 " there was free haemoptysis.
130 " no sputum was expectorated.

In 113 cases of children under fifteen expectoration was absent. The only children who succeeded in coughing up their spueta were 4, all between eleven and fifteen years of age, who brought up rusty sputa; and 3 between six and eight years old, who coughed up blood-stained mucus.

Of adults who did not expectorate at all during the whole attack of pneumonia there were as many as 17. Three of these were between fifteen and forty (out of 194); 5 were between forty and fifty (out of 52); 3 between fifty and sixty (out of 16); 3 between sixty and seventy (out of 20); and 3 between seventy-two and seventy-five (out of 5).

6. Complications and sequels.—In five patients the pneumonia ended in gangrene of the lung; one a case of chronic caries of the spine, one complicated with pericarditis, one with ulcerative endocarditis, and one with delirium tremens. All of these five patients died.

Ulcerative, septic endocarditis occurred six times, and in three of these meningitis was also present; while in two other cases meningitis

¹ Of these forty-two, only four reached 105·8°, and nearly half did not exceed (so far as was noted) 105°.
² Two of these patients recovered after temperatures of 106° and 106·6°.
³ Both of these patients died.
was found after death. *Pericarditis* was a complication in fourteen cases, and in one of these there was also endocarditis (in addition to the six above mentioned).

*Icterus* occurred in four cases; in three of these the right base, and in one the right apex, was affected.

*Delirium tremens* complicated pneumonia in seven cases, and five of these were fatal.

*Otorrhoea* from tympanitis occurred three times, and pulmonary embolism once, with recovery.

Many other complications recorded, as tonsillitis, gout, asthma, cardiac and renal disease, and laryngitis, were no doubt accidental coincidences. None of these occurred more than four times among the 434 cases.

In probably every case there was pleurisy; but serous effusion was only abundant enough to be noticed in 17 cases.

The most frequent sequel was *empyema*, which followed pneumonia in 24 cases, one of them being circumscribed empyema of the right apex.

7. **Duration.**—This was measured by the pyrexia, which occasionally preceded the evidence of hepatisation, and more often ceased before the signs of consolidation had disappeared. In 118 cases the duration was not ascertainable.

The shortest cases lasted three days; and these mild, but certainly not "abortive," cases occurred in children or youths, as the following detailed statement shows:

### Duration of Pneumonia

<table>
<thead>
<tr>
<th>Days</th>
<th>Ages of Patients</th>
<th>Cases.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Three</td>
<td>3, 5, 6, 8, 9, 12, 14, 22, 27</td>
<td>11</td>
</tr>
<tr>
<td>Four</td>
<td>{ 5, 6, 7, 7, 10, 11, 11, 12, 12, 13, }</td>
<td>21</td>
</tr>
<tr>
<td>{ 14, 14, 17, 19, 19, 20, 21, 21, 22, 26 }</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Five</td>
<td></td>
<td>30</td>
</tr>
<tr>
<td>Six</td>
<td></td>
<td>68</td>
</tr>
<tr>
<td>Seven</td>
<td></td>
<td>62</td>
</tr>
<tr>
<td>Eight</td>
<td></td>
<td>39</td>
</tr>
<tr>
<td>Nine</td>
<td></td>
<td>29</td>
</tr>
<tr>
<td>Ten</td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>Eleven</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Twelve</td>
<td>(in four recovery delayed by complications)</td>
<td>8</td>
</tr>
<tr>
<td>Thirteen</td>
<td>(fall of temperature by lysis)</td>
<td>3</td>
</tr>
<tr>
<td>Fourteen</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Fifteen</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Seventeen</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Twenty-one</td>
<td>(delayed by complications)</td>
<td>3</td>
</tr>
<tr>
<td>Above three weeks (delayed by complications)</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

316

The most frequent duration of the disease was about a week; 32 patients were actually ill for only three or four days, 199 (nearly two-thirds of the number) for five to nine days, and only 17 for more than twelve days.

8. **Mortality.**—The total number of deaths was 111, a high per-
per centage (25.5) out of 434. But this general statement is of little value. Hospital mortality in this as in most other diseases is higher than that of private practice, because the previous habits and conditions of the patient are less favourable. But again, general cases seen in consultation are usually severe, and sometimes hopeless. Of 73 of the latter class 25 were fatal—more than a third.

Deaths among 362 Hospital Patients (86)

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Highest observed Temp.</th>
<th>Herpes</th>
<th>Locality</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>F.</td>
<td>47</td>
<td>103.4</td>
<td>Absent</td>
<td>Right base</td>
<td>Ulcerative endocarditis and meningitis</td>
</tr>
<tr>
<td>M.</td>
<td>31</td>
<td>102.4</td>
<td></td>
<td>Double</td>
<td>In course of phthisis</td>
</tr>
<tr>
<td>M.</td>
<td>43</td>
<td>104</td>
<td></td>
<td>Double</td>
<td>In course of phthisis</td>
</tr>
<tr>
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<td>49</td>
<td>103.2</td>
<td></td>
<td>Double</td>
<td>Carcinoma of stomach</td>
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<tr>
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<td></td>
<td>Right apex</td>
<td>Hæmoptysis; gumma of liver</td>
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<tr>
<td>M.</td>
<td>43</td>
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<td></td>
<td>Left base</td>
<td>In course of morbus Brightii</td>
</tr>
<tr>
<td>M.</td>
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<td></td>
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<td>Gangrene of lung; chronic disease of spine</td>
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<tr>
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<td></td>
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</tr>
<tr>
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<td>51</td>
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<td></td>
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<td>Gangrene of lung; pericarditis</td>
</tr>
<tr>
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<td>43</td>
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<td></td>
<td>Right apex</td>
<td>In course of morbus Brightii</td>
</tr>
<tr>
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<td>Laryngitis</td>
</tr>
<tr>
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<td>2</td>
<td>104</td>
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<td>Right base</td>
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</tr>
<tr>
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</tr>
<tr>
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<td>Morbus Brightii (lardaceous)</td>
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<tr>
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<td></td>
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<td>Morbus Brightii; pericarditis</td>
</tr>
<tr>
<td>M.</td>
<td>46</td>
<td>...</td>
<td></td>
<td>Left base</td>
<td>Pericarditis; delirium</td>
</tr>
<tr>
<td>M.</td>
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<td>...</td>
<td></td>
<td>Right apex</td>
<td>Intemperate; hyperpyrexia</td>
</tr>
<tr>
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<td>109</td>
<td></td>
<td>Right base</td>
<td>Intemperate</td>
</tr>
<tr>
<td>M.</td>
<td>41</td>
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<td></td>
<td>Right apex</td>
<td>Delirium tremens</td>
</tr>
<tr>
<td>M.</td>
<td>35</td>
<td>103</td>
<td>Present</td>
<td>Left base</td>
<td>Gangrene of lung; ulcerative endocarditis</td>
</tr>
<tr>
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<td>46</td>
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<td>Absent</td>
<td>Double</td>
<td>Tumour of brain</td>
</tr>
<tr>
<td>F.</td>
<td>42</td>
<td>101.8</td>
<td></td>
<td>Left base</td>
<td>Intemperate; ulcerative endocarditis</td>
</tr>
<tr>
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<td></td>
<td>Right apex</td>
<td>Delirium</td>
</tr>
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<td></td>
<td>Right base</td>
<td>Delirium</td>
</tr>
<tr>
<td>M.</td>
<td>39</td>
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<td></td>
<td>Left base</td>
<td>Delirium</td>
</tr>
<tr>
<td>M.</td>
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<td>...</td>
<td></td>
<td>Left base</td>
<td>Pleuritic effusion</td>
</tr>
<tr>
<td>M.</td>
<td>58</td>
<td>102</td>
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<td>Right apex</td>
<td>Pericarditis</td>
</tr>
<tr>
<td>M.</td>
<td>27</td>
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<td></td>
<td>Right base</td>
<td>Delirium</td>
</tr>
<tr>
<td>M.</td>
<td>26</td>
<td>102.4</td>
<td></td>
<td>Right base</td>
<td>Pericarditis; thrombosis of pulmonary artery</td>
</tr>
<tr>
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<td>Left base</td>
<td>Delirium</td>
</tr>
<tr>
<td>M.</td>
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<td>Delirium tremens</td>
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</table>

1 The total mortality for all ages and both sexes, without for the most part exclusion of secondary cases, was 191 in 1060 (Coll. Invest. Report—private cases), 281 in 2618 (Huss hospital and private at Stockholm), and 192 in 1010 (Coupland—Middlesex Hospital).
### Deaths among 362 Hospital Patients—continued

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Highest observed Temp.</th>
<th>Harpes.</th>
<th>Locality.</th>
<th>Complications.</th>
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<tr>
<td>M.</td>
<td>42</td>
<td>104·2</td>
<td>Absent</td>
<td>Left base</td>
<td>Intemperate</td>
</tr>
<tr>
<td>M.</td>
<td>36</td>
<td>105·8</td>
<td>&quot;</td>
<td>Double</td>
<td>Intemperate; gangrene of lung</td>
</tr>
<tr>
<td>F.</td>
<td>63</td>
<td>102·4</td>
<td>&quot;</td>
<td>Right base</td>
<td>Pericarditis</td>
</tr>
<tr>
<td>M.</td>
<td>49</td>
<td>104</td>
<td>&quot;</td>
<td>Left base</td>
<td>Intemperate; gangrene of lung</td>
</tr>
<tr>
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<td>107·8</td>
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<tr>
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</tr>
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<td>104·4</td>
<td>&quot;</td>
<td>Right apex</td>
<td>Intemperate; pericarditis</td>
</tr>
<tr>
<td>M.</td>
<td>22</td>
<td>103·4</td>
<td>&quot;</td>
<td>Double</td>
<td></td>
</tr>
<tr>
<td>M.</td>
<td>39</td>
<td>97·8</td>
<td>&quot;</td>
<td>Double</td>
<td>Admitted 7th day, moribund</td>
</tr>
<tr>
<td>M.</td>
<td>8</td>
<td>104</td>
<td>&quot;</td>
<td>Double</td>
<td>Pericarditis; empyema</td>
</tr>
<tr>
<td>M.</td>
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<td>103·6</td>
<td>&quot;</td>
<td>Left apex</td>
<td>Delirium tremens</td>
</tr>
<tr>
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<td>Admitted comatose</td>
</tr>
<tr>
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<td>Diabetes</td>
</tr>
<tr>
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<td>Double</td>
<td>Intemperate</td>
</tr>
<tr>
<td>F.</td>
<td>18</td>
<td>105</td>
<td>&quot;</td>
<td>Double</td>
<td></td>
</tr>
<tr>
<td>M.</td>
<td>4</td>
<td>104·6</td>
<td>&quot;</td>
<td>Double</td>
<td></td>
</tr>
<tr>
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<td>3</td>
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<td>&quot;</td>
<td>Double</td>
<td></td>
</tr>
<tr>
<td>M.</td>
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<td>Present</td>
<td>Left base</td>
<td>Endocarditis</td>
</tr>
<tr>
<td>M.</td>
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<td>103·6</td>
<td>Absent</td>
<td>Left base</td>
<td>Influenza; pericarditis</td>
</tr>
<tr>
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<td>&quot;</td>
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<td>Delirium</td>
</tr>
<tr>
<td>F.</td>
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<td>Right apex</td>
<td></td>
</tr>
<tr>
<td>F.</td>
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<td>105·2</td>
<td>&quot;</td>
<td>Double</td>
<td>Pregnancy; empyema</td>
</tr>
<tr>
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<td>Right lung</td>
<td>Pericarditis</td>
</tr>
<tr>
<td>M.</td>
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<td>&quot;</td>
<td>Right lung</td>
<td>Hodgkin's disease</td>
</tr>
<tr>
<td>F.</td>
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<td>Delirium</td>
</tr>
<tr>
<td>M.</td>
<td>48</td>
<td>103·6</td>
<td>&quot;</td>
<td>Double</td>
<td></td>
</tr>
<tr>
<td>M.</td>
<td>63</td>
<td>101·2</td>
<td>&quot;</td>
<td>Right base</td>
<td></td>
</tr>
<tr>
<td>M.</td>
<td>45</td>
<td>105·4</td>
<td>&quot;</td>
<td>Right apex</td>
<td>Pericarditis; delirium</td>
</tr>
<tr>
<td>M.</td>
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<td>Double</td>
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</tr>
<tr>
<td>M.</td>
<td>52</td>
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<td>Double</td>
<td>Intemperate</td>
</tr>
<tr>
<td>M.</td>
<td>32</td>
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<td>&quot;</td>
<td>Left base</td>
<td></td>
</tr>
<tr>
<td>M.</td>
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<td>Acute nephritis</td>
</tr>
<tr>
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<td>Left base</td>
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<td>Present</td>
<td>Left base</td>
<td></td>
</tr>
<tr>
<td>M.</td>
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<td>104</td>
<td>Absent</td>
<td>Double</td>
<td>Pericarditis</td>
</tr>
<tr>
<td>M.</td>
<td>4m.</td>
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<td>&quot;</td>
<td>Right lung</td>
<td>Delirium; laryngitis</td>
</tr>
<tr>
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<td>Double*</td>
<td>Laryngitis</td>
</tr>
<tr>
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<td>Absent</td>
<td>Double</td>
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</tr>
<tr>
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<td>&quot;</td>
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<td>Fatty liver</td>
</tr>
<tr>
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<td>Left apex</td>
<td></td>
</tr>
<tr>
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</tr>
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<td>Empyema</td>
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</tr>
<tr>
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</tr>
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<td>&quot;</td>
<td>Right lung</td>
<td></td>
</tr>
<tr>
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<td>43</td>
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<td>&quot;</td>
<td>Double</td>
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</tr>
<tr>
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</tr>
<tr>
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<td>54</td>
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<td>&quot;</td>
<td>Double</td>
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</tr>
<tr>
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<td>&quot;</td>
<td>Right apex</td>
<td></td>
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<td>104</td>
<td>&quot;</td>
<td>Left base</td>
<td>Laryngitis</td>
</tr>
<tr>
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<td>104</td>
<td>Present</td>
<td>L. lung and R. apex</td>
<td>Intemperate</td>
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</table>
Deaths among 73 Private Cases (25)

*(Herpes absent in all)*

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Highest observed Temp.</th>
<th>Locality.</th>
<th>Complications.</th>
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</thead>
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<td>103</td>
<td>Left base</td>
<td>Intemperate; pregnant</td>
</tr>
<tr>
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<td>101</td>
<td>Right base</td>
<td>Intemperate</td>
</tr>
<tr>
<td>M.</td>
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<td>...</td>
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</tr>
<tr>
<td>F.</td>
<td>30</td>
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<td>Delirium</td>
</tr>
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<td>F.</td>
<td>15</td>
<td>104</td>
<td>Left base</td>
<td></td>
</tr>
<tr>
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<td>Double</td>
<td></td>
</tr>
<tr>
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<td>66</td>
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<td>Right base</td>
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<td>Phthisis</td>
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<td>Icterus</td>
</tr>
<tr>
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</tr>
<tr>
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</tr>
<tr>
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<tr>
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<td>General bronchitis</td>
</tr>
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<td>Right base</td>
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<tr>
<td>M.</td>
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<td>General bronchitis</td>
</tr>
<tr>
<td>F.</td>
<td>75</td>
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<td>Right mid-lung</td>
<td>Delirium</td>
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<td>62</td>
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<td>Emphysema</td>
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<tr>
<td>M.</td>
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<td>Left base</td>
<td></td>
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<td>Mania</td>
</tr>
<tr>
<td>M.</td>
<td>49</td>
<td>106</td>
<td>Left base</td>
<td>Delirium</td>
</tr>
</tbody>
</table>

Death-rate of Pneumonia at several Ages

Under 5, 5 deaths out of 20 cases. Four months (pericarditis), 24, 3 (double).

Between 5 and 10, 1 death. 66.

" 10, 15, 2 deaths. 45.

" 15, 20, 5. 48.

" 20, 25, 9. 45.

" 25, 30, 10. 38.

" 30, 35, 8. 29.

40, 31 (double), 32, 33, 33 (double). 33 (intemperance), 34.
Between 35 and 40, 13 deaths out of 33 cases. 35 (double), 35 (intemperance), 35 (double, intemperance), 36 (intemperance), 36 (intemperance), 36 (intemperance), 37 (cancer), 37 (double, intemperance), 38 (intemperance), 39 (double), 39* (intemperance), 39, 39.

,, 40 ,, 45, 13 ,, ,, 27 ,, 41 (pericarditis), 41 (intemperance), 42, 42 (intemperance), 42 (morbis Brightii), 42 (tumour cerebri), 43, 43 (syphilis), 43 (double) bis, 43, 13 (pericarditis), 44 (morbis Brightii).

,, 45 ,, 50, 18 ,, ,, 27 ,, 45 (pericarditis), 45 (pericarditis), 45, 45 (intemperance), 45 (hyperpyrexia), 45 (morbis Brightii), 46 (endocarditis), 47 (endocarditis and meningitis), 47 (diabetes), 48 (pericarditis), 48 (double) ter, 48 (intemperance), 49 (morbis Brightii), 49 (intemperance), 49 (double), 49 (hyperpyrexia).

,, 50 ,, 53, 4 ,, ,, 5 ,, 51 (bronchitis), 52 (double, intemperance), 54 (endocarditis) bis.

,, 55 ,, 60, 10 ,, ,, 15 ,, 55 (bronchitis), 55, 56 (diphtheria), 57, 57, 58 (morbis Brightii), 58 (pericarditis), 58 (double), 58 (diabetes), 59.

,, 60 ,, 65, 5 ,, ,, 12 ,, 62, 62, 63 (pericarditis), 63, 63 (morbis Brightii).

,, 65 ,, 70, 4 ,, ,, 6 ,, 66, 66, 66 (pericarditis), 68.

Over 70, 3 ,, ,, 5 ,, 72, 75, 75 (morbis cordis).

Causes of death.—That age is a grave element in the prognosis of pneumonia is clear from the last table. Excepting infants, there are few deaths under 15. After this age, the mortality rises steadily with the age of the patient, although even after 70 the prognosis is not always fatal. It will also be noted that the deaths at the earlier ages are generally accompanied by one of the untoward complications to be mentioned presently; whereas most of the fatal cases in the later decades are uncomplicated.

In all cases, single or double, and at all ages, the important causes of death were as follows:—

Intemperance, with or without delirium tremens, was present in a marked degree in 16 of the fatal cases, and this is probably below the truth.

In 10 other fatal cases delirium, more violent and particularly more diurnal than usual, was noted; and although only 4 cases were distinguished as well-marked delirium tremens, many of the other delirious patients were intemperate.

Pericarditis occurred in 14 cases, all of which proved fatal: endocarditis in 7, and meningitis in 5.

Bronchitis was fatal in only 3 cases, all old people.

In 3 fatal cases there was pleuritic effusion, and in only 1 of these was it purulent; so that all the cases followed by empyema without other complications ended in recovery except this one.
Hyperpyrexia—a temperature over 106°—occurred in 6 patients, of whom 2 recovered (under 107°), and 4 died.

Of fatal cases of secondary pneumonia 8 occurred in the course of Bright's disease; 5 in course of chronic tuberculosis; 2 in diabetes, and the 6 others happened in cases of cancer, lymphadenoma, tertiary syphilis, cerebral tumour, and valvular disease of the heart.

REFERENCES


CATARRHAL PNEUMONIA

SYNONYMS.—Pulmonary catarrh, Broncho-pneumonia, Lobular pneumonia, Disseminated pneumonia, including Vesicular pneumonia.

DEFINITION.—This disease is more difficult to define than is lobar pneumonia. In some cases it closely resembles the latter both clinically and anatomically, while in others the two diseases offer few points of resemblance. Lobular pneumonia, as an anatomical lesion, includes such different conditions as acute pulmonary catarrh in a child after measles, the chronic broncho-pneumonia of rickets, the hypostatic
pneumonia of fever (in part), the caseating pneumonia of phthisis, and the lobular suppuration of pyæmia. The last two conditions form part of the pathology of tuberculosis and of septic embolism respectively. Hypostatic pneumonia is a secondary condition probably beginning in the bronchi, certainly secondary to prolonged passive congestion, and made up of lobular hepahtisation, often confluent and sometimes primarily lobar, with edema, and collapse. The remaining kinds of pulmonary inflammation, which are included under the head of Catarrhal Pneumonia are better dissociated from lobar or fibrinous pneumonia, and named acute or chronic pulmonary catarrh.

Of the disease thus restricted we may say that it befalls children far more frequently than adults; that, though sometimes acute in its origin, the signs of its invasion are never so abrupt or well marked as those of lobar pneumonia; that its course is either subacute or chronic, and that it never terminates by crisis; that it is accompanied by bronchitis, and is probably always secondary. It is much more directly connected with breathing cold air, or air laden with dust and other mechanical impurities, than is lobar pneumonia, and seldom occurs in an epidemic form. It is often secondary, not only to bronchitis, but also to measles and whooping-cough, and to chronic conditions of ill health, particularly rickets. As a rule it affects both lungs, and that in irregular patches, without preference for either apex or base. Although often accompanied by pleurisy, this may be absent.

Anatomy.—As one of its names implies, catarrhal pneumonia affects the lungs lobule by lobule, and these lobules are often found scattered over both lungs, whence the name “disseminated pneumonia.” More often several adjacent lobules are affected together so as to form patches in different parts of the lung. These patches again may coalesce, and thus after death from pulmonary catarrh a considerable area of continuous solid and hepahtised lung is found which closely resembles the lobar hepahtisation of fibrinous pneumonia. Pulmonary catarrh does not affect the base more frequently than the rest of the lung; nor is there reason to believe that it has a predilection for the apices, except when it is part of the effects of tuberculous invasion. Occasionally only part of a lobule may be affected; smaller, separately inflamed areas may, in some cases, be distinguished by the naked eye, and this kind of disseminated pulmonary catarrh may be distinguished as “vesicular pneumonia.”

Microscopic examination shows that the disease originates in a catarrhal inflammation of the mucous membrane of the bronchi; this spreads to the last bronchioles, which open into the lobules—a condition described as “capillary bronchitis.” When the lobule is itself examined it is found filled with epithelium and with smaller cells which have the characters of leucocytes. There are no blood-discs, and fibrin is absent or scanty. The larger epithelioid and smaller inflammatory cells fill the air-vesicles and intralobular passages, and as they accumulate expel the air and convert each infundibulum, and at last the whole lobule, into a small solid mass.
Along with hepatised lobules there are almost always found, particularly in young children, some lobules which have undergone collapse; they are airless, but not soft; shrunken, not swollen; and empty, that is, not stuffed with inflammatory products. These collapsed portions are seen as depressed, dark patches on the surface of the lung, and particularly at its lower edges. They were formerly confounded with the patches of lobular pneumonia which they so often accompany; but from these they are entirely distinct, and it is doubtful whether such collapsed portions of lung are capable of undergoing the process of lobular inflammation.

Bacteriology.—The encapsulated diplococcus (or pneumonococcus) of lobar pneumonia is often found in the alveolar contents of the affected lobules; but it is often absent, and with it or in its stead may be seen various other micrococci. There is no reason to believe that pulmonary catarrh depends upon, or even is constantly associated with the presence of any one specific microbe.

Etiology.—Catarrhal lobular pneumonia is almost always associated with bronchitis, and is usually secondary to it. It is in children often accompanied with signs of rickets; and it is a frequent sequel of measles and whooping-cough, less frequently of scarlatina, small-pox, or enteric fever. It may follow as the result of burns, and complicate infantile "dysentery." Why it is comparatively rare in adults is difficult to say. It does not appear to be a specific infective disease, and its relation to true lobar or fibrinous pneumonia rather obscures the etiology of the latter than lights up its own.

Clinical symptoms.—The symptoms and signs of pulmonary catarrh are far less striking than those of acute lobar pneumonia. There are no rigor, no sudden rise of temperature, to mark its onset; we suspect rather than ascertain its presence when a case of bronchitis in a child or an aged patient is accompanied with fever; and comparatively slight physical signs are sufficient to confirm our suspicions.

The onset of pulmonary catarrh is gradual; the fact that bronchitis is extending to the smaller tubes is not shown by any trustworthy physical sign, although we may find a sonorous, deep-toned rhonchi-replaced or accompanied in certain parts of the chest by a high-pitched sibilus; or the large, coarse, toneless rattles produced by mucus and air in the trachea and larger bronchi may be replaced by mucous rales of the same quality, but smaller, that is to say with more numerous explosions in each inspiration. These physical signs may or may not be present. Our recognition of the presence of what is called capillary bronchitis depends upon the symptoms of dyspnoea—upon cyanosis, increased rapidity of breathing, and sucking in of the soft parts about the thorax with each inspiration. When inflammation has gone still farther and affected the lobules, there is often, as we should expect from the above anatomical account, no diminution of resonance on percussion; for the solidified lobules are scattered, and resonance spreads, while dulness does not. If several inflamed or collapsed lobules are near
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enough to form an airless patch, we may then recognise on light per-
cussion a note shorter, of higher pitch and of diminished tone, com-
pared with that afforded by the rest of the chest; but probably the
earliest physical sign of the presence of lobular catarrh will be a con-
sonating, more or less musical quality in the previously toneless inspiratory
rôle.

If the affected lobules become so numerous and close as to form a large
solid area in one part of the lung, we obtain more decided dulness on per-
cussion; and the râles become finer and more consonating until they
approach very nearly the characteristic fine crepitation of lobar pneumonia.
These last signs are not only slow in appearing, or absent throughout the
whole case, but they do not advance steadily from the base upwards.
They appear most often in the middle of the back, internal to one or the
other shoulder-blade, sometimes at one base, sometimes in the armpit,
and occasionally at the apex; but they frequently shift in position, and
are very seldom symmetrical on the two sides. This absence of definite
local distribution is an important element in distinguishing catarrhal
from fibrinous pneumonia, and also from phthisis; but unfortunately
it affords no evidence against the presence of diffused pulmonary
tuberculosis.

In a further stage of consolidation the dulness may be as marked
and extensive as in lobar pneumonia. Tubular breathing will then be
heard, and marked bronchophony, not with the child's ordinary voice but
with its cry.

The pyrexia which shows the presence of lobular and vesicular
pneumonia is usually moderate in degree, even in children; and rarely
approaches the height seen in lobar pneumonia, in septicemia, or in
tuberculosis. The course of the fever is irregular without constant
evening accessions, but remittent rather than intermittent.

The skin of a patient suffering from catarrhal pneumonia is often
dry and hot, but it never has the pungent feel characteristic of fibrinous
pneumonia; and in many cases, especially in children, it is covered with
sweat. This is particularly the case when the broncho-pneumonia is
tuberculous; but the symptom is not only inconstant in itself, it is
greatly modified by the use of poultices, steam-kettles, and other modes
of treatment.

The urine is often scarcely affected, and when febrile in other
characters very seldom contains albumin.

The pulse is frequent and usually soft, more frequent in proportion
to the temperature than in lobar pneumonia.

The respirations increase in frequency in proportion to the extent of
the lungs involved. In mild cases they are not above thirty, but in
severe cases rise to fifty, and even considerably higher. The forced
muscles of respiration are usually brought into play, and the degree in
which they are used furnishes another valuable evidence of the severity
of the disease. The nostrils dilate with each inspiration, as in many of
the lower animals; and the inspiratory movements of the diaphragm are
followed by expiratory contractions of the muscles of the abdominal wall. In young children, of whom we chiefly speak in this description, respiration is mainly abdominal, and in dyspnœa the muscles which move the thorax have less play than in adults; but in these patients the want of resistance of the tissues causes much earlier and usually more marked movement of the soft parts than in adults. With inspiration a deep depression is seen to form above each clavicle, and another between the trachea and the manubrium. The epigastrium is drawn in, and even the lower ribs and ensiform cartilage yield to atmospheric pressure, particularly when the bases of the lungs are collapsed and airless.

Orthopnœa may be seen when extensive catarrhal pneumonia affects an adult, but is less common than in dyspnœa from cardiac disease, bronchitis, or lobar pneumonia; and in children it is decidedly less common than in dyspnœa from laryngitis or from empyema.

The important symptom of insufficient aeration of the blood, which consists of blueness of the surface, is present in all severe cases of pulmonary catarrh. It is usually first seen in the lips and the ears, then in the fingers and toes, next in the face, and finally over the whole surface of the body and the mucous membrane of the mouth. While the frequent and forced respirations show the want of air, the cyanosis just described shows how ineffectual these forced and frequent efforts are in supplying the want. Thus the rapidity of the pulse indicates the increased efforts of the heart to keep up the pulmonary circulation, and the paleness of the skin proves the deficiency of supply to the systemic capillaries, and gives us a hint of the similar failure of circulation in the lungs.

In adults affected with catarrhal pneumonia the expectoration is usually scanty, and consists of thin mucus without much admixture of air. In most cases, however, there is already present the frothy mucopurulent expectoration of preceding bronchitis. The rusty sputum of fibrinous pneumonia, whether prune-juice, or saffron, or greenish in tint, is probably never seen in cases of lobular pneumonia. In cases with the symptoms and course of catarrhal, not fibrinous, pneumonia, I have occasionally observed scanty and nearly airless sputa, of a pinkish colour from the presence of blood; and streaks of blood probably derived from the veins of the trachea or upper air-passage may sometimes be seen, as in ordinary cases of bronchitis which do not extend to the lobules. True rusty sputa are as nearly pathognomonic of fibrinous pneumonia as any symptom can be; and pure hæmoptysis under similar circumstances is almost as characteristic of phthisis; but the expectoration offers us no help in the often difficult question between uncomplicated pulmonary catarrh and disseminated tuberculosis of the lungs.

In children, not only in infants, but often up to the age of eleven or twelve, there is a remarkable inability to expectorate. Phlegm is coughed up into the larynx and then swallowed; and though some children as young as seven or eight (once a boy of only five) have learnt how to get rid of it, and others may be taught the art, we must reckon
upon the absence of this valuable help in diagnosis. When by natural or forced vomiting a child empties its air-passages of accumulated secretion, mucus and muco-purulent matter may be seen in the vomit; some of it perhaps lately swallowed, and some ejected directly from the larger bronchi. In such cases the presence or absence of rusty sputa may be observed.

In aged patients it is not uncommon to find the same inability to expectorate as in children.

As the disease goes on, the patient's appetite suffers; he becomes pale and thin; his nights are constantly disturbed by cough, and his strength gradually fails.

Course, complications, and prognosis.—The duration of acute pulmonary catarrh is undetermined. Infants may die in a few days from want of power to get rid of the secretion which obstructs the air-passages; and in aged persons catarrhal pneumonia, or so-called capillary bronchitis, often occurs as the last stage of chronic bronchial catarrh, and proves fatal in two or three days. But, with the exception of the two extremes of life, patients suffering from catarrhal pneumonia seldom die within a week or even a fortnight from the date of recognition of the disease. Most often its special symptoms and signs gradually disappear, and the condition of simple bronchitis in which the disease began remains at its conclusion. In children, particularly, this also gradually subsides, as a rule, and complete convalescence is established.

Of the complications to be feared, the most frequent and formidable is tuberculosis, either in its generalised form or in that of chronic phthisis. A large proportion of cases of bronchitis and broncho-pneumonia in children are associated with the presence of caseous lymph-glands, cervical, mesenteric, or mediastinal; and the bacilli which already exist in the lympharia may readily infect the lungs and other organs.

Another important complication of catarrhal pneumonia in children is empyema, and this must be sedulously looked for, or it may escape notice. In infants the most frequent complication is extensive collapse of the lung, which, as above stated, almost always accompanies lobar pneumonia at an early age, and often determines a fatal event.

On the whole the prognosis is most affected by the age of the patient. Broncho-pneumonia in little children and broncho-pneumonia in the aged are very fatal diseases. In children above two or three years old the forecast is much better, but it is still decidedly worse than in cases of lobar pneumonia occurring at the same age. In adults uncomplicated pulmonary catarrh is rare, and usually dependent upon some special form of irritation. In such cases the prognosis depends upon the nature of the irritant and the probability of its being withdrawn.

Pulmonary catarrh, which is secondary to measles or whooping-cough, is more serious than that which occurs without these precedents. Again, when it occurs, as is so often the case, in a child affected with rickets, it is more likely to be intractable or fatal than when this condition is absent. Of the symptoms of the disease, the extent of lung
involved in inflammation or collapse, the frequency of the pulse, and above all the degree of dyspnoea estimated by the symptoms above detailed, are the most important elements of prognosis.

The most dangerous symptoms are those of suffocation, and this is the most frequent immediate cause of death.

Next in importance to the lividity and the forced and rapid breathing, which show pulmonary obstruction, are the weak and frequent pulse and pallor which point to failure of the heart.

In little children pulmonary collapse is often as extensive as pulmonary catarrh. After the second year is passed, this complication is less frequent and dangerous, and in adults it scarcely occurs except at the posterior edge of the lower lobe. The most serious complication of all is the presence of tubercle.

Diagnosis.—The recognition of broncho-pneumonia in most cases is not difficult; but certain mistakes are apt to occur, and in some cases it is impossible to decide upon the exact nature of the pulmonary inflammation present, except by the progress of the case.

From lobar pneumonia the diagnosis, founded on the onset and course, as well as on the symptoms above enumerated, is only difficult when many inflamed lobules coalesce to form a single large patch towards the base of the lung. Here dulness on percussion, tubular breathing, and crepitant râles will simulate primary lobar inflammation; and when such a case is seen for the first time it is almost impossible to avoid the mistake. In children we have not the help afforded by the sputum, and the physical signs are not so definitely localised as in adults.

On the other hand, true fibrinous pneumonia in children is sometimes called lobular merely because of the patient’s age. The shortness of its course will correct the error. In elderly people the difficulty is to distinguish broncho-pneumonia from “capillary” bronchitis—from bronchitis, that is, affecting the smallest bronchioles, but leaving the lobules free. Probably the two affections are often present together. When rhonchus and sibilus are present over the whole of both lungs, with little or no elevation of temperature, and with marked cyanosis, it is unlikely that the bronchitis is complicated by pneumonia. When one lung is decidedly more affected than the other, and particularly if partial dulness, bronchial breathing, or crepitation appear in patches which shift their position, and if these symptoms are accompanied by pyrexia, we may be sure that lobular pneumonia is present.

The most common and important difficulty of diagnosis is between lobular pneumonia and acute tuberculous of the lungs—not phthisis, for in its symptoms and localised physical signs this disease is almost always characteristic; if it be overlooked or mistaken in its early stages, it is not confounded with lobular pneumonia. No doubt, a chronic broncho-pneumonia confined to one apex would be difficult or impossible to distinguish from early phthisis; but the existence of such an affection, apart from the actual presence of tubercle, is extremely rare.
in an adult. Acute broncho-pneumonia of one apex with pyrexia, frequent pulse, sweating and general bronchitis, may, however, closely simulate phthisis in a child. When the previous condition and mode of onset are unknown, it is difficult if not impossible to distinguish this from phthisis; in fact, the physical signs are the same. The far more frequent difficulty is to decide upon the presence of disseminated tubercles in the case of children who do not throw off a pulmonary catarrh, but week after week grow paler and thinner and more feverish; or, again, in the case of adults who, long subject to bronchitis, begin to show the pyrexia and physical signs of broncho-pneumonia in addition. In such cases the bronchitis and lobular pneumonia are actually present whether tubercle be there or not. The presence of the latter must be recognised by other than auscultatory signs. In adults there is no likelihood at this stage for blood and nummular sputa and fragments of elastic tissue to be present in the sputa, but we may find the bacilli of tubercle—a discovery which at once decides the question. In children this means of diagnosis is absent; and we depend rather on the height and irregularity of the pyrexia, on the rapidity and degree of emaciation, and the amount of sweating. The same symptoms help us in the recognition of acute miliary tuberculosis in an adult. In the latter case, however, the question more often lies between tuberculosis and bronchitis.

A disease which not infrequent mistakes warn us to be watchful against confounding with broncho-pneumonia in children, is empyema. In both cases we have pyrexia, dulness on percussion, and cough, with dyspnea and without expectoration. In both the child may be pale and thin, and the fingers clubbed; in both its voice may be too weak and high pitched to help us by yielding bronchophony or tactile fremitus; and, lastly, the small area of an infant's chest, the loudness of its breathing, and certain conditions which favour conduction of bronchial breathing through effused liquid, even in an adult, may all combine to obscure the diagnosis of empyema. On the other hand, knowledge of this danger will sometimes lead even an experienced physician to suppose that dulness at the base of the lung with no vocal resonance or crepitation must be due to pleuritic effusion, whereas it is really a patch of inflamed and collapsed lobules.

The height of the temperature is often a guide, but, on the one hand, this does not distinguish empyema from broncho-pneumonia associated with tubercle; and on the other hand we sometimes, though rarely, meet with 'empyema in which, even in a child, the temperature is scarcely above normal. One help we may gain in doubtful cases by listening to the chest when the child is crying; we may then often obtain both bronchophony and fremitus; and, after a prolonged scream, so deep an inspiration is taken that crepitation or tubular breathing, before unheard, becomes distinctly audible.

In doubtful cases of the kind the use of a grooved needle or a hypodermic syringe is most valuable. It will decide a question which cannot
be settled by the most careful and repeated auscultation; and this is
its only legitimate use.

Beside tuberculosis, another general disease is not infrequently
mistaken for broncho-pneumonia, both in children and adults, namely,
enteric fever. Here we have pyrexia, often of irregular course, and
usually, sooner or later, accompanied with pulmonary congestion,
bronchitis, or hypostatic pneumonia. The bowels are not infre-
quently constipated, an enlarged spleen cannot always be felt, and in
children the characteristic rose spots are sometimes absent. When seen
for the first time, and with an imperfect history of the case, a decision
is sometimes impossible. The points to look for are the early or later
occurrence of pyrexia or cough, the course of the temperature, the presence
of headache, delirium, or apathy, and the fulness of the abdomen. If
enterica be present, a few spots, after repeated searches, will generally
be found on the back and loins, if not on the abdomen and flanks;
and repeated trial will seldom fail to decide whether the spleen be en-
larged or not. In doubtful cases pulmonary catarrh is more probable in
the case of children, and enteric fever in the case of adults.

Treatment.—In the early stage of pulmonary catarrh the treatment
is that of bronchitis. We endeavour to relieve cough, pain, and oppres-
sion of the chest, to promote secretion in the affected parts, and to
favour action of the bowels, the kidneys, and the skin. For this purpose
confine the body is usually desirable; but with little children it is
sometimes better to cover the chest with a cotton-wool jacket, and allow
them to lie in the nurse’s arms, or to sit up when they cough. In cold
dry weather a steam kettle is a useful help in addition to an open fire;
and an adult patient will find still greater relief by inhaling steam from
boiling water to which compound tincture of benzoin, eucalyptus, turpen-
tine, terebene or some other aromatic oleo-resin has been added. When
the air is warm and moist there is no object in making it moister. The
same applies to protection by curtains, screens, canopies, and tents. In
the winter season, in large wards or draughty rooms, these appliances are
most valuable; but when there is too free a secretion of mucus, when
the patient is sweating, feverish and restless, a close, hot and damp air
is not the best for the patient to breathe.

As soon as febrile symptoms have subsided and the patient may be
considered convalescent, it is most important for him to breathe the open
air when the weather is at all suitable. Even in winter a child well
wrapped up and carried out for a quarter of an hour at a favourable
time will often show by improved appetite and better sleep the benefit
of fresh air. This is particularly important in the case of children
suffering from broncho-pneumonia after measles or whooping-cough.

When the temperature is high and the skin is very hot, tepid spong-
ing is called for, and the child should be put in a warm bath every
evening. If fever should run high, repeated lukewarm baths are the best
means of reducing it. A mustard plaster on the front of the chest is
often extremely useful in the early stages of the disease; afterwards a
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jacket poultice of linseed is a common and for the most part a good application: it relieves pain, promotes action of the skin, moderates the cough, and comforts the patient. For infants, however, its weight and tightness are as a rule undesirable, and it may be better replaced by a jacket of cotton-wool worn next the skin.

The food of a patient with broncho-pneumonia should be liquid, given in comparatively small amount and more frequently than in health. To young and weakly children a little milk or broth should be given every two hours; but in other cases there is no need for such frequent feeding, and four hours is not too long a time to elapse between each meal; even this is sometimes too short an interval for the digestive powers of an older patient, and it is better to be content with three or four meals in the day.

Diluents should be taken freely; cold water, soda water with milk or fruit syrup, thin barley-water, toast and water, tamarind water, or lemonade made with cream of tartar; this "imperial drink" is grateful to the patient, and is also diuretic and slightly laxative.

Stimulants are not to be prescribed as a matter of routine. Brandy should be given if the pulse be very rapid and the action of the heart weak and irregular.

The most useful drugs in the early stages are ipecacuan, squill and nitre, sweetened with syrup of tolu or oxymel. Occasionally in the early stage of the attack a few drops of antimonial wine are efficacious in promoting secretion in the bronchial tubes and skin. In serious cases with urgent dyspnæa no drug is so valuable as carbonate of ammonia given in doses of one grain for an infant to five for an adult; its pungency may be covered by liquorice, treacle, or syrup.

When there is marked cyanosis, with a small and weak radial pulse and distension of the jugular veins and epigastric pulsation, bleeding to six, eight, or even ten ounces is indicated; and may often save a patient’s life. In the case of children, two or three leeches on the sternum may be applied in similar circumstances.

As soon as the temperature is normal and the physical signs abated, the patient should be removed to another room, and allowed to sit by an open window, if the weather be favourable. Beside drives in an open carriage, it is often desirable that removal to the south coast of England or to the shores of the Mediterranean should follow an attack of bronchitis with broncho-pneumonia. Children should be removed as soon as possible to the seaside, or at least to pure country air.

CHRONIC PNEUMONIA

It is doubtful whether acute lobar pneumonia ever ends in a chronic inflammatory process. At any rate, in the great majority of cases, if the patient recover, the inflamed lung recovers also, and completely. I once had the opportunity of observing the state of a lung in a patient who died accidentally about a fortnight after recovery from acute fibrinous
pneumonia. The previously inflamed part of the organ was still distinguishable; its consistence was firmer, its colour darker, and it contained less air and more serum than the rest of the lungs: but no other traces of hepatisation were present, and it was no doubt functionally active.

*Chronic lobar hepatisation.*—Addison, however, described a condition of persistent consolidation of lung following acute lobar pneumonia, in which the section is no longer soft and granular but homogeneous, smooth and tough; though still solid and airless. He believed that recovery from this condition might take place. He called it “uniform albuminous induration” (1a, p. 28). Charcot and some other modern writers admit the existence of a similar condition, but regard it as a coalescence of inflamed lobules; and due therefore to catarrhal, not fibrinous pneumonia (7). We cannot doubt the existence of an anatomical state such as is described; but it must be extremely rare, and its true nature, origin, and event are at present undetermined.

*Chronic broncho-pneumonia.*—That lobular pneumonia may pass from a sub-acute to a chronic form is no doubt true if we regard the question of time alone without reference to acuteness of symptoms; but there is no evidence that an inflammatory process of a catarrhal kind will continue and spread after the temperature has become normal. The broncho-pneumonia which follows measles, whooping-cough, or diphtheria in children, and the much rarer broncho-pneumonia of adults, or that again which is caused by inhalation of dust (pneumoconiosis), have, so far as at present known, only one event if the patient neither dies in the acute stage nor recovers; and that event is infection by the bacillus of tubercle, and the establishment of pulmonary phthisis.

*Chronic interstitial pneumonia.*—There is, however, another form of disease of the lung to which the name of chronic pneumonia is often given. It is that which was described by Cruveilhier as “induration ardoise,” by Addison as “iron-gray consolidation” (1a, p. 28), by Corrigan as “cirrhosis of the lung” (8), by some French writers as “sclérose pulmonaire.”

1 The origin and limits of the disease are still matters of dispute, and its clinical history does not appear to be precisely correlative with its anatomy. On the one hand, it has often been confounded with the more chronic forms of phthisis; on the other, it is usually associated with dilatation of the bronchi, although the two conditions are not always coincident. It appears sometimes to be a sequel of bronchitis or broncho-pneumonia, and sometimes to begin in successive attacks of pleurisy. Lastly, a similar anatomical condition is occasionally found in cases of syphilis, associated with gumma and probably originating in specific peribronchitis of the trachea and bronchi; this last group of cases is precisely analogous to those of so-called syphilitic cirrhosis of the liver. Clinically it may simulate (tuberculous) phthisis, and was formerly

1 Bayle probably described this condition (as others have done since) under the title of phthisis with melanosis; Anuenbrugger earlier still as scirrhous (that is, induration) of the lung. Corvisart and Chomel also described its anatomy independent of phthisis.
described as a variety of phthisis. The fact that this form of lues does not affect the apices of the lung and thence travel downwards, that it is not associated with other tuberculous lesions and is with those of syphilis, and the absence of bacilli from the sputum are the chief diagnostic points which usually guide us aright, even when hectic, emaciation, hemoptysis, and the phthisical signs of phthisis are most misleading (Path. Tr. 1877, p. 313).

The resemblance, however, which Corrigan justly remarked between his chronic indurating fibrous process in the lung and that which was described by Laennec as cirrhosis of the liver, is an anatomical one. Anatomically we may put the two conditions together, and may compare with them the chronic interstitial nephritis of Bright's disease, which is often styled cirrhosis of the kidneys. We may even extend the comparison to the chronic indurating process in the nervous centres which is now named sclerosis; but in their origin these similar anatomical results differ greatly. By far the most frequent and characteristic forms of cirrhosis of the liver are due to intemperance, but there is no corresponding alcoholic cirrhosis of the lungs.

**Morbid anatomy.**—Pulmonary cirrhosis is most often limited to a single lung. It may begin in any part; but most frequently it starts from the root of the lung and spreads along the peribronchial connective tissue so as to cause on section a radiating appearance of fibrous bands. In other cases it spreads inwards from a patch of local pleuritic thickening. Occasionally it affects the base or the whole lower lobe, or the middle lobe of the right lung; while the rest of the organ remains unaffected. The new fibrous tissue is white, dense, and often so extremely tough as to cut like tendon, or even like cartilage. It is sometimes confined to broad septa or patches, leaving the rest of the lung free; but often it penetrates extensively between the lobules, mapping them out and giving a marbled aspect to a section. The pulmonary tissue itself is darker than the healthy parts of the lung, and varies from a slate colour to an almost black tint. It is firm, and contains less air than usual, but does not sink in water. On microscopical examination, the fibrous tissue is as dense as that of a tendon, though the course of the fibres is less parallel. The pulmonary tissue within a lobule is altered by collapse of some of the air-vesicles, by the lining epithelium being more visible and thicker than normal, by the capillaries being more or less obliterated, and by the thickening of the alveolar wall.

The process is not, therefore, a purely interstitial inflammation as defined by Virchow; it is parenchymatous also. Some pathologists, indeed, consider the intralobular changes as primary and essential; and the interlobular and peribronchial fibrous growth as secondary. But if it be true that pulmonary cirrhosis is seldom the consequence of broncho-pneumonia, and most often takes its rise in peribronchitis or pleurisy, it seems probable that the primary seat of the disease, as of the corresponding changes in the liver, the kidneys, and the spinal cord, is in the interstitial connective tissue.

The affected lung on section shows cavities which can readily be
traced to the bronchial tubes, of which they are 'certainly dilatation.' These sacculus pouches were regarded by Corrigan as the result of traction of the cicatrising fibrous tissue; and, although an opposite opinion has been ably defended by other pathologists, from Laennec downwards, it seems probable that the Dublin physician was correct. There are, no doubt, many cases of primary bronchiectasis, such as those which follow whooping-cough or bronchitis in children, and lead to uniform cylindrical dilatations without consequent fibrous thickening; and, again, such as form the bronchial pouches which have been described as retention-cysts in fetid bronchitis. The sacculus dilatations in cirrhosis are often extremely irregular, and in some parts so closely packed together that scarcely any pulmonary tissue is left between them.

The result is contraction with diminution in bulk of the affected parts of the lung. There may be emphysema in the parts unaffected by cirrhosis, and sometimes large subpleural bullae are seen. On the whole, however, the process is that of contraction. The affected lung becomes smaller as well as denser; and, when one side only is affected, the opposite lung may be hypertrophied and the mediastinum dragged over by the diseased lung.

Beside bronchiectasis, emphysema is also very frequently present, and shows its characteristic signs during life and anatomical appearances after death. Sometimes the hypertrophic pleurisy which has been the starting-point of pulmonary cirrhosis is only part of a general chronic inflammation with thickening of the whole pleura-peritoneal cavity; and lungs, heart, and abdominal viscera are all affected by a similar process (Path. Tr. 1882, p. 172).

Symptoms.—These are often obscure and difficult of interpretation. They are chiefly of a physical kind. More or less dulness on percussion will be present, due probably to thickened pleura rather than to the pulmonary cirrhosis itself. Bronchial breathing may be heard, although this is far from constant; more often the pulmonary murmur is obscured by rhonchus and sibilus; but perhaps the most frequent auscultatory sign is the presence of râles, medium or large, and sometimes gurgling, accompanying inspiration and expiration. Not infrequently deficiency of breath-sounds, combined with the dulness, may raise a doubt whether there be an effusion of pus or serum in the pleura.

Expectoration is commonly abundant, muco-purulent in quality, and often serous. Hæmoptysis is not unknown, even when cases of chronic phthisis are carefully excluded.

On inspection the affected side moves less freely than the sound one, and is ascertained by the cyrto meter to be the smaller. Owing to the same process of contraction the cardiac impulse may be displaced towards the affected side, or may be higher than usual.

There is often no pyrexia; the temperature is never high unless, as occasionally happens, septicemia ensues from ulceration of dilated bronchial cavities. There is dyspnœa, increased on exertion, and the fingers may be clubbed.
NATURAL HISTORY.—Cirrhosis of the lung may come on at any age; but the majority of the patients are under fifty. It is rare in children, but not very rare between 15 and 20. The disease is more common in men than in women. It is often associated with intemperate habits, and sometimes with a similar interstitial fibrosis, degeneration, and shrinking of the kidneys or the liver; or with chronic peritonitis as well as pleurisy.

DIAGNOSIS.—Some definitions of this disease would include all cases in which the physical signs denote a contracted, indurated, and comparatively airless condition of one or both lungs, due to fibrous degeneration of the pulmonary tissue, with the presence of numerous cavities containing pus and mucus. In the majority of such cases, however, this condition is due to tuberculous infection. The tuberculous disease affects both lungs; it begins in the apices and travels downwards. The cavities are excavations due to ulceration—vomicae in the technical sense of the word. In fact the disease is chronic, and sometimes obsolete, tuberculous phthisis. Many of the earlier cases recorded by Andral, Corrigan, and Addison were undoubtedly tuberculous; and the same criticism applies to a majority, at least, of the cases which have been described by the late Sir Andrew Clark and other writers as “fibroid phthisis.”

The long controversy as to the degree in which the names pulmonary tubercle and phthisis are coextensive in signification may now be regarded as settled; and the final verdict is in favour of the doctrine originally taught by Laennec, and against that which distinguished between tuberculous and non-tuberculous phthisis. All phthisis is tuberculous; but, along with tubercle, catarrhal pneumonia, congestion, ulceration, bronchitis, pleurisy and fibrosis are always present. Phthisis of rapid course, with extensive ulceration and congestion, may still be called “pneumonic”; and cases which are long protracted, and perhaps at last cured, with abundant cicatrisation, may still be called “fibroid,” or rather “fibrous”; but these are forms of essentially the same disease.

If all cases of cirrhosis of the lung were, as the late Dr. Moxon put it, “phthisis in the præter-pluperfect tense,” there would be no need for the name; or, if retained at all, it would merely denote an anatomical condition present in various degrees in all cases of chronic phthisis. There is, however, abundant evidence that cirrhosis may be independent of tubercle from beginning to end; and the problem is to distinguish this non-tuberculous disease from the much commoner cases of chronic phthisis which simulate it, as well as from the fibrous degeneration which accompanies syphilis of the lung.

In the first place, phthisis almost always affects both lungs, cirrhosis is as a rule confined to one. Either disease may follow chronic bronchitis or repeated pleurisy; but in phthisis other organs, sooner or later, partake in the disease. Accordingly the presence of laryngitis with hoarseness or aphonia, diarrhoea, symptoms of tubercle of the testes or kidneys, of joints or of lymph-glands, is good evidence that the disease of the lung, however chronic, is tuberculous phthisis. Cirrhosis, on the other hand, is confined to the chest. It is a purely local condition; and
symptoms, in nature and degree, depend entirely upon the physiological effects of the local lesion. No doubt the diagnosis, comparatively easy as it is in the dead-house, is sometimes difficult at the bedside; but the presence or absence of the bacillus of Koch should be decisive.

Next to chronic phthisis, empyema is perhaps the affection most likely to be confounded with cirrhosis of the lung. In both cases the symptoms may be similar, namely, cough, wasting, pallor, pyrexia, dyspnoea; and in both there may be dulness at the base of one lung. In empyema the breath-sounds are usually absent over the dull area, and there may be no expectoration; but bronchial breathing is occasionally heard through pleuritic effusion, and if an empyema have opened into the lung, the expectoration may be of much the same kind as that which, in a case of cirrhosis, proceeds from a bronchial pouch. Moreover, in empyema some amount of contraction of the affected side of the chest is often present. The physical conditions are so similar that it is not surprising to find the physical signs also similar. The diagnosis depends, in most cases, upon a knowledge of the origin and progress of the patient’s illness. In this, as in so many other cases, a right decision does not depend upon a single so-called pathognomonic symptom, but upon a wide survey of probable alternatives, and weighing of the course and probabilities of the individual case along with the actual physical signs present.

After all, in some cases puncture alone can decide the matter, and the test is readily applicable.

Prögnosis.—Cirrhosis of the lung is always a grave but rarely a hopeless condition. The forecast varies with the amount of lung involved, with the duration of the disease, and, most of all, with the degree of general disturbance; loss of appetite, anaemia, wasting, sweating, vomiting, or diarrhoea are unfavourable circumstances. When the patient’s weight is kept up, and he eats and sleeps well, we may hope that even extensive cirrhosis of the lung may gradually lead to contraction and obliteration of cavities, and final cicatrisation of the affected parts with hypertrophy of the opposite lung. Such a complete cure is no doubt exceptional; more often the disease passes into a permanently chronic condition, and the patient dies at last from bronchitis affecting the sound lung, or from some intercurrent affection.

Among the complications to which the patient with cirrhosis is liable may be mentioned—first, dilatation of the right side of the heart and anasarca; next, septicæmia from ulceration of one of the bronchial cavities; more rarely, abscess of the brain from pyæmia of a similar origin; or lardaceous disease, the result of prolonged suppuration.

Treatment.—From the nature of the case, the treatment must be tentative and expectant, following the indications of the patient rather than of the local disease. We endeavour to keep the expectoration from becoming fetid, and to check its amount, to relieve cough, particularly at night, by parergic and other anodynes, and to hasten the process of cicatrisation by occasional counter-irritants, or by strapping the affected
side with plaster. Inhalations of turpentine, thymol, terebene or creasote are often useful in lessening the secretion and correcting the factor. At the same time, by help of mineral acids and biters, particularly quinine and nux vomica, we try to improve the patient's appetite; with the same object we give him varied and abundant food, consulting rather his own caprice than ordinary rules of diet: of stimulants, by far the most useful, if the patient can bear it, is malt liquor, particularly porter. If this cause cough, constipation, or headache, ale or light German beer may be taken with advantage at the mid-day meal. Sometimes wine is better relished and proves more useful; in most cases it is certainly superior to alcohol in the form of spirits; if given at all, brandy is, as a rule, most useful when given as a sedative at night.

Whenever the weather permits it, the patient should be taken out of doors. When this is impracticable, he may sit before a widely open window, warmly wrapped up, and breathing through the nostrils with the mouth persistently closed. Fresh air often proves the most powerful promoter of appetite and of sleep.

Cases of cirrhosis of the lung are greatly benefited by climatic treatment; removal from dust-laden workshops and from foggy towns to pure air is the first step to improvement, and may often cut short the disease in its early stage. A mild and equable climate, such as that afforded by the south-west coast of England and many parts of Ireland, is the best for these patients:

There is no doubt that these cases are among those that derive most benefit by spending successive winters and springs on the Riviera, at Palermo, Corfu, Cairo; or in islands like Madeira, the Canaries, or those of the Southern Pacific. If the patient's means are ample, this arrangement is the best than can be made for his advantage. [Vide art. "Climate in Disease," vol. i. p. 247.]

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REFERENCES


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