

Diseases of the organs of respiration : a treatise on the etiology, pathology, symptoms, diagnosis, prognosis, and treatment of diseases of the lungs and air-passages / by Samuel West.

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A TREATISE ON THE
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AND TREATMENT OF DISEASES OF THE LUNGS
AND AIR-PASSAGES.

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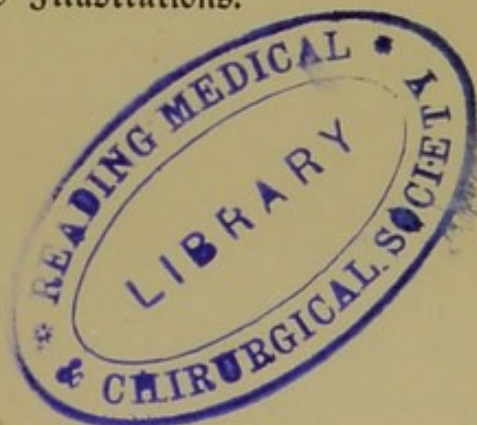
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PREFACE.

MEDICAL works have to a great extent, in recent years, taken the form of collections of essays on special subjects by different authors. This has its advantage, for such sub-division of labour is the obvious and easy way of dealing with the ever-increasing mass of medical literature. Yet these composite productions, even where they gain in literary completeness, lose what is of at least equal importance, the individuality and consistency which a single writer gives to his work.

In no department of medicine has the progress of science and the growth of literature been more rapid than in that which forms the subject of this treatise, and it may seem rash for an author to attempt to deal with it single-handed. Lightened as the labour is by the many complete monographs recently written on special parts, the task is still a heavy one. Time alone, if nothing else, would have brought this fact home to me, for many years have been spent upon the work beyond the time originally allotted to it. Still, I do not regret the delay, for it has enabled me to write from a wider experience, and to put to a longer test the conclusion on disputed points to which I had been led. The part which bacteria play in the production of disease and its symptoms, and the complications resulting from mixed infections, have altogether changed our conception of morbid processes. The progress of knowledge in this field of research has been so rapid, that constant revision has been necessary to keep what had been written up-to-date, and even while the book was passing through the press, certain sections have had to be recast.

I have throughout not hesitated to express my own opinions, as I think I may fairly claim the privilege of doing—after an experience of so many years' duration both at a special chest hospital and at two general hospitals; but I have tried to avoid dogmatism, knowing well that personal experience may be peculiar or even misleading. Wherever the views advanced differ in important respects from those generally accepted, as, for instance, in the case of pneumothorax and

of broncho-pneumonia, I have set forth in detail the grounds of my conclusions, so that others may be able to form their own judgment.

I have throughout endeavoured to acknowledge all good and recent work, and to give authors the credit due to them, but it has not been my intention to supply complete bibliographies. The references given indicate where fuller information of a bibliographical kind can be obtained. If I have omitted names which were entitled to mention, I trust the omission will be lightly dealt with, considering how large the literature of the subject is.

The book is divided into sections, and not chapters, and the relative importance of its parts is indicated by types of different kinds. This is found to render reading and reference easier. It does not, however, follow that those sections which stand in small type are of little importance; they are in many cases, as in the article on pleural tension, of special rather than of general interest. Clinical cases are all printed in small type, as well as much other matter, which is added to illustrate or explain the conclusions stated in the text.

In the introduction, it has not been my object to give a complete account of the normal anatomy and physiology of the respiratory organs. I have dealt only with certain parts which are of importance, as having a distinct pathological and clinical bearing. Thus I have described the lymphatic system of the lung and pleura more fully than is usual, and have treated at some length the physical conditions of the pleura and lung, upon which the right understanding of many clinical phenomena depends.

In dealing with physical signs, I have reduced the technical terms employed to their simplest dimensions. The difficulties of Auscultation and Percussion are largely of our own making, and arise from the misuse of words, the terms being either not strictly defined, or used deliberately by different writers in different senses. The result is confusion, not in respect of facts, but of words. I have dealt shortly with the two chief offenders—Bronchial Breathing and Râles. My own terminology is, I trust, simple and consistent; at any rate I have not added to the difficulties by coining any new terms.

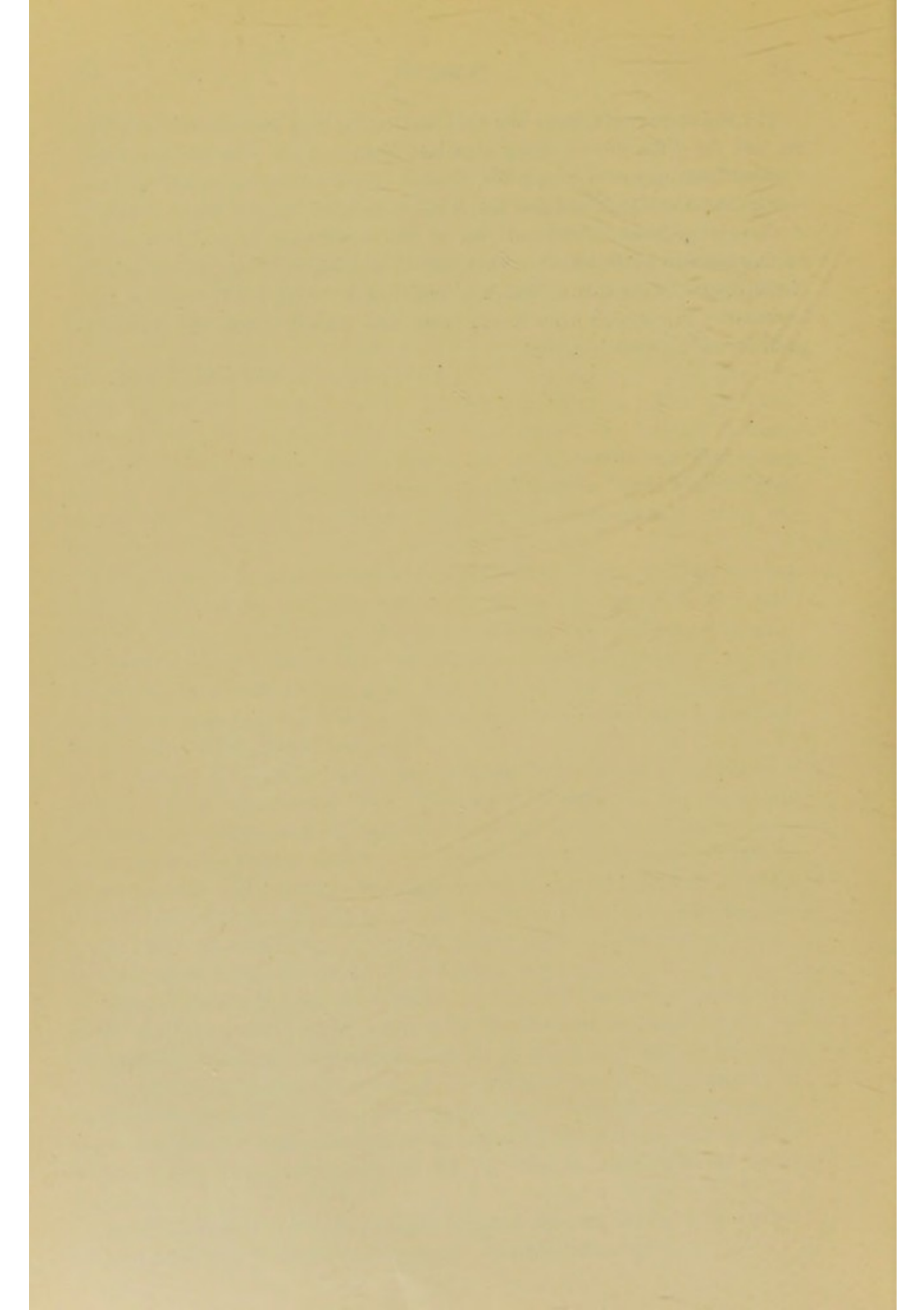
Many of the illustrations are original. To Dr. F. W. Andrewes, my colleague at St. Bartholomew's Hospital, I am much indebted for the use of some fine microscopical drawings; to Professor Stewart, of the Royal College of Surgeons, for permission to photograph some of the preparations in that museum; to Professor Hamilton of Aberdeen, and to the publishers of Ziegler's Pathology, for the reproduction of some standard and well-known illustrations, and to many friends at the other London hospitals for the ready access allowed me to the museums.

As the usefulness of a book is largely increased by a good index, I have taken pains to make this full and complete, so that reference may be easy.

If I had known beforehand the time and labour this book would have cost me, and the difficulties of doing sustained literary work with the many and constant interruptions of a busy life, I much doubt whether I should have had courage to undertake it, and now that it is accomplished I cannot but be conscious of many deficiencies. Yet I am glad of the opportunity it has given me of putting together much scattered work, and of bringing to a focus the experience of many years in the special branch of medicine in which I have been so long interested. In writing I have learnt much, and I trust others may find some profit in reading what is written.

SAMUEL WEST.

15 WIMPOLE STREET,
CAVENDISH SQUARE, W., *May* 1902.



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DISEASES OF THE ORGANS OF RESPIRATION.

1. INTRODUCTORY.

THE object of respiration is to supply the oxygen necessary for the oxidation processes of the body as well as to remove the carbonic acid formed within the body. This is the function of the lungs.

The process of respiration consists in the interchange of gases between the blood and air.

The essential structure of the lungs is extremely simple, viz., a thin membrane richly supplied with blood-vessels, exposed on both sides to the air, the blood being kept in movement and constantly renewed by the heart and the other forces of circulation, and the air by the bellows-like action of the thorax.

Defects of respiration may therefore be produced in three ways:—

1. By defects in the supply of blood to the vesicles of the lungs.

2. By defects in the supply of air to the vesicles of the lungs.

3. By defects in the respiratory organs themselves.

Most of the so-called diseases of the respiratory organs are associated with definite anatomical changes in them which constitute their pathological anatomy.

ANATOMY.—The respiratory organs of man consist of the lungs, with their pleura, and the air-passages leading to them.

The air, drawn through the nose or mouth and pharynx, enters the larynx, where the air-passages proper commence, and passes in succession through the trachea, bronchi and bronchioles to reach the vesicles.

The trachea extends from the larynx to a point within the thorax opposite the third dorsal vertebra (*cf.* fig. 4), where it bifurcates to form the two

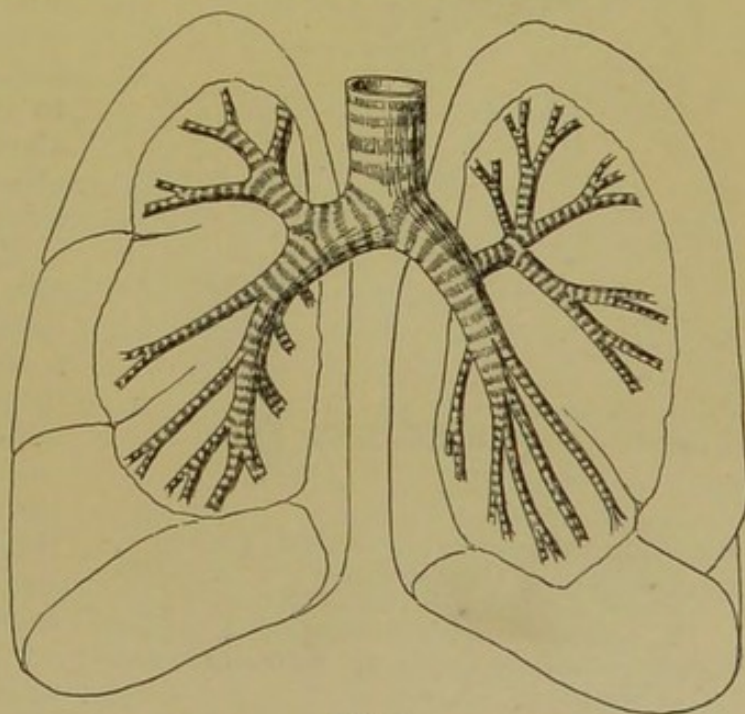


Fig. 1.

Trachea and bronchi, showing the mode of division. The bronchi of the upper lobe are seen to take origin almost at right angles to the main bronchus (*Testut*).

main bronchi, one for each side. Within the lung the bronchi divide rapidly, for the most part dichotomously, and they do not anastomose with each other.

The bronchi are named according to their sizes, large, medium, and small. They generally divide 5 to 8 times before the terminal bronchi are reached. Bronchi of 1 millimetre ($\frac{1}{25}$ inch) in diameter or less are called small bronchi or bronchioles.

The bronchioles also divide; the larger branches lie between the lobules of the lung, the smaller enter the lobules and terminate; they are accordingly described as sublobular, intralobular, and terminal.

The terminal bronchioles expand into a funnel-shaped space, the vestibule, from which small air-ducts (alveolar canals) lead to the infundibula, and upon the walls of these the vesicles are arranged like grapes upon a stalk.

A group of terminal bronchioles with their ultimate expansions form a lobule; groups of lobules form the lobes into which the lungs are subdivided.

The whole lung is bound together by a network of connective tissue extending from the pleura to the root, where the bronchi and vessels enter, the smallest meshes of which correspond with lobules, and the larger with the areas of distribution of the larger bronchi. In this connective tissue the blood-vessels, lymphatics, and nerves run.

Each lung thus formed is irregularly pyramidal or conical in shape, with the base downwards, hollowed below to rest upon the diaphragm, and, on the inner side, to adapt itself to the pericardium; the apex rises above the clavicle outside the thorax about an inch and a half on each side.

The lungs are divided into lobes, the upper conical and the lower quadrilateral. The right lung has a small middle lobe and the left a deep notch anteriorly to receive the apex of the heart.

The right lung is the shorter and the left the narrower, but, on the whole, the right lung is slightly larger than the left and weighs more in the proportion of about 22 to 20.

The weight of the lungs varies much according to the amount of blood and air they contain, the

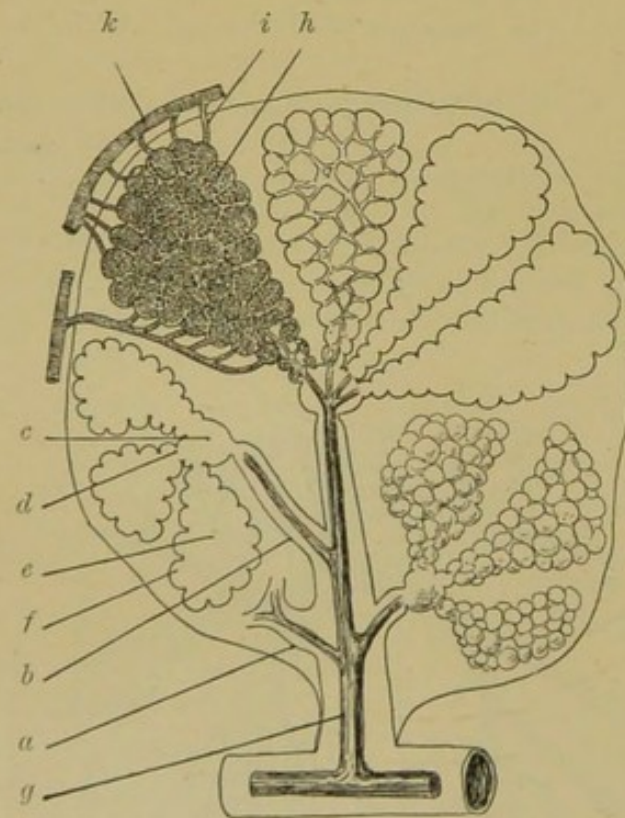


Fig. 2.

a, intralobular bronchus; *b*, terminal bronchus; *c*, vestibule; *d*, alveolar canals; *e*, infundibulum; *f*, alveolus; *g*, branch of pulmonary vein; *h*, capillary network over alveoli; *i*, radicles of pulmonary vein; *k*, pulmonary vein (*Testut*).

specific gravity of an absolutely airless lung being 1056, as for instance in the foetus before birth; after respiration it varies greatly, from .746 to .345, or, in the condition of maximum distension, only .126.

The lungs bear to the body-weight the proportion, on the average, of about 1 to 37 in the male and of 1 to 43 in the female, but the limits of variation are considerable, viz., between 1 to 35 and 1 to 50. In the male the average

weight of the two lungs is 44 ozs. (right 24, left 21), and in the female 32 ozs. (right 17, left 15).

The lung is spongy, or porous on section, floats in water, and crepitates when squeezed between the fingers. It is rosy pink in colour in the infant, but in the adult darker and more or less mottled with dark pigment.

The description of the minute anatomy of the lung may be conveniently divided into two parts: (1) that of the air-passages from the larynx to the sublobular division of the bronchial tubes, (2) that of the intralobular bronchioles with the infundibula and vesicles in which they end.

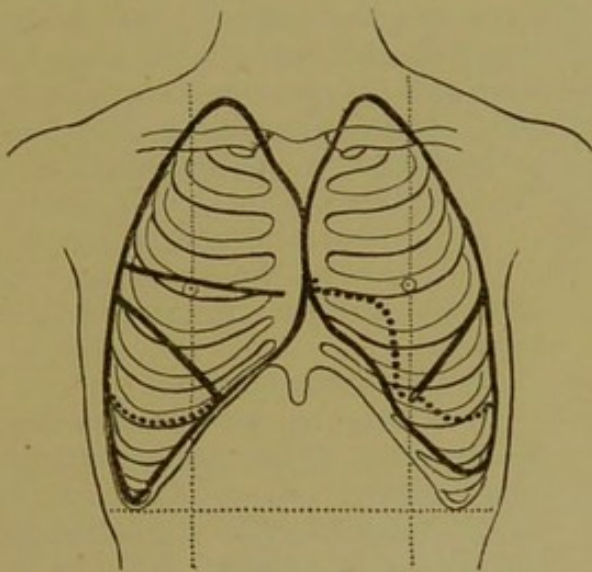


Fig. 3.

Diagram to show the front view of the lungs
in situ with its lobes.

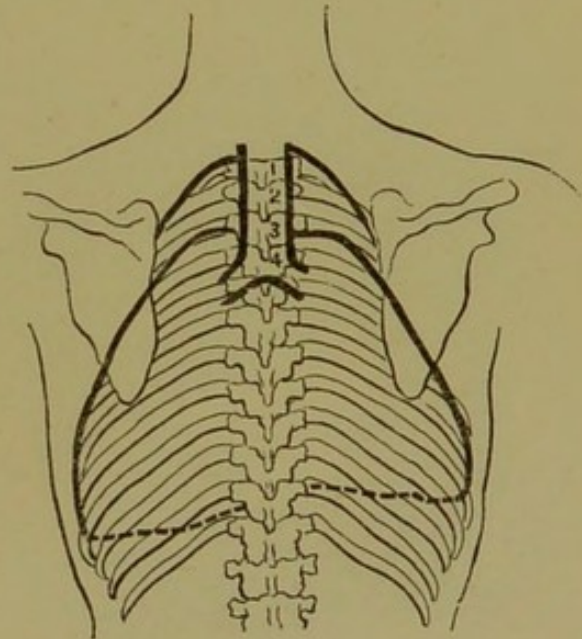


Fig. 4.

Similar diagram to show back view.

The Air-Passages.—The air-passages are constructed upon the same essential plan throughout, though the constituents vary in relative prominence and importance in different parts.

The most striking constituent of the larger tubes is the cartilaginous framework found in them. This subserves the purely mechanical function of keeping the tubes open during the varying pressures of respiration; without it the tubes might be sucked in on inspiration and the entrance of air prevented.

This resistant framework is most developed, as would be expected, where the exposure to pressure is greatest, viz., in the larynx and trachea. In the larynx it is highly specialised and furnished with a complicated arrangement of muscles to subserve the further function of phonation, and to provide a sphincter for the protection of the air-tubes against the entry of foreign bodies.

In the trachea it consists of C-shaped rings, closed at the ends behind by a fibro-elastic membrane containing much muscular tissue, which also unites the separate rings to each other. Where the trachea bifurcates these rings fuse to form an irregular mass of cartilage completely surrounding the dividing tubes, and presenting a prominent projecting ridge within at the point of bifurcation (*cf.* fig. 16).

As the tubes become smaller the cartilages become more irregular in shape and arrangement and fewer in number, and they finally disappear entirely in the bronchioles.

The cartilages of Wrisberg and Santorini and part of the arytenoids are formed of the yellow elastic cartilage, but the rest are all of the common hyaline variety.

The Bronchi.—A medium-sized bronchus presents on section the following microscopic structure (fig. 5):—

A. The epithelial coat.

1. A single layer of columnar ciliated epithelial cells with some goblet cells (*e*).

2. Several strata of transitional cells mostly pyriform in shape.

3. A single layer of flattened squames, constituting the so-called Débove's membrane. Some of these cells form the foot-plates in which the tapering ends of the transitional cells are, by some authors, said to end. These cells play a most important part in the regeneration of the epithelium.

4. A basement membrane 11 μ in thickness, homogeneous, and perfectly distinct from Débove's membrane.

B. The internal fibrous coat.

5. A layer of areolar tissue containing much elastic tissue arranged longitudinally (*d*). This coat is richly supplied with branches of the bronchial artery, and in it the lymphatics arise as irregular plasma-spaces, lined with endothelium.

C. The muscular coat.

6. A continuous layer of interlacing bundles of non-striped muscular fibres (*c*) arranged circularly (the bronchial muscle), between which numbers of lymphatic spaces and vessels exist.

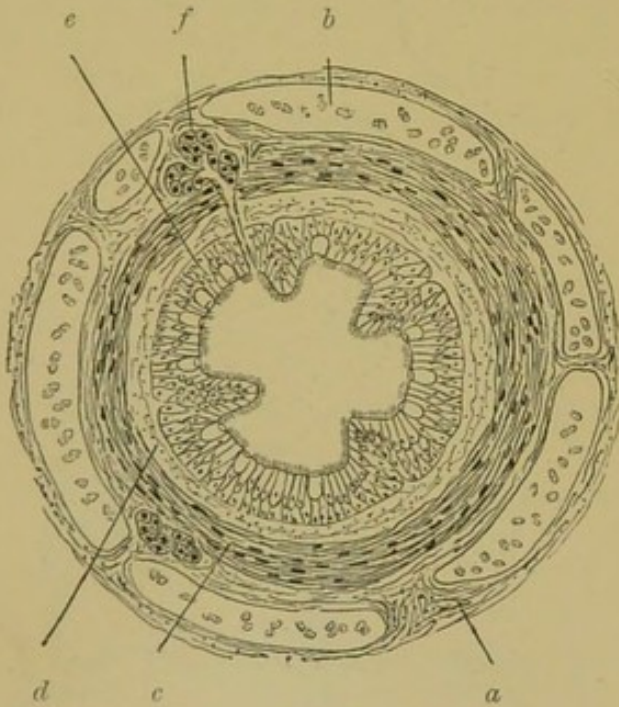


Fig. 5.

Section of a bronchus (from *Testut*, modified).

a, fibrous tissue; *b*, cartilages; *c*, muscular layer; *d*, submucous layer; *e*, epithelial layer, showing ciliated epithelium and goblet cells; *f*, bronchial gland.

D. The external fibrous coat or adventitia.

7. Loose bundles of white fibrous tissue with elastic fibres (*a*) arranged concentrically with the lumen and continuous with the adventitia of the pulmonary vessels and with the interlobular septa of the lung.

This coat contains the acini of the mucous glands, blood-vessels, numerous lymphatics, the cartilages, nerves and nerve ganglia.

The mucous glands are racemose, and lie partly between and partly upon the cartilages, the ducts pierce the muscular coat and open upon the surface of the mucous membrane; their basement membrane is continuous with that of the bronchus, and is lined near the orifice with columnar ciliated cells. The branches of the pulmonary artery and vein usually lie upon opposite sides of the bronchus.

The cartilages are irregular plates of the common hyaline kind, and external to them are the nerves and nerve ganglia, in close relation with the branches of the bronchial vessels.

The lymphatic vessels are abundant, and much adenoid tissue is present.

The Trachea.—The structure of the *trachea* does not differ essentially from that of the bronchus just described, except in the further development of the cartilages as C-shaped rings, and in the arrangement of the muscular fibres between the ends of the rings to form the trachealis muscle and complete the tube. This muscle has both longitudinal and circular fibres, but the latter are the most numerous.

The mucous glands are larger and more numerous, and open by larger orifices. The lymphatics are abundant, and there is a rich plexus of nerves beneath the trachealis muscle derived from the vagus, recurrent laryngeal, and sympathetic.

The small and terminal bronchi.—As the bronchi get smaller the cartilages and glands disappear; the epithelium becomes reduced to a single layer of cubical ciliated cells; and finally, in the terminal bronchi only the following coats are distinguishable:—(1) a single layer of small polyhedral granular cells without cilia; (2) a circular muscular coat of unstriped fibres; and (3) a fine adventitia of elastic fibres arranged chiefly in longitudinal networks. The muscular coat in the small bronchi is twice as thick, relatively to the total thickness of the walls, as it is in the larger bronchi. The terminal bronchi are regarded as having a respiratory function, because of the change in their structure, and because of their being supplied by the pulmonary artery.

The infundibula and air-vesicles.—In the *infundibula* and *air-vesicles* the following further changes occur. The granular cells no longer form a continuous layer but are broken up into groups, separated by flat squamous cells. These cells become more and more numerous as the alveoli are approached, and form in them a continuous lining.

The number of granular cells visible depends upon the distension of the lung; they are few or absent when the alveoli are completely distended, and more numerous when collapsed. In the foetus, *i.e.*, before respiration, they are all of the granular kind.

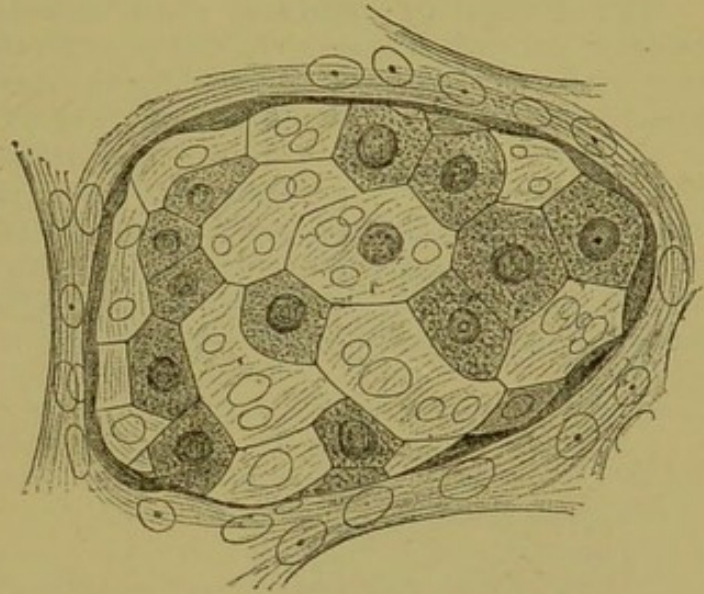


Fig. 6.

An alveolus from the lung of a new-born child, stained with nitrate of silver to show the epithelium. Some of the cells are much more distinct and granular than the rest (*Schultze*. $\times 500$).

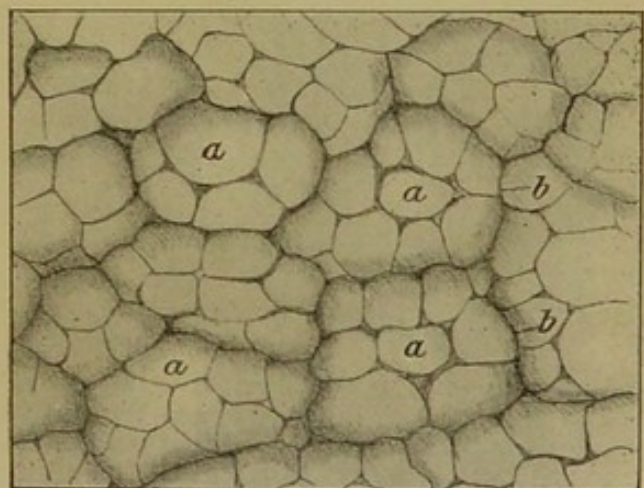


Fig. 7.

Portion of the outer surface of the cow's lung (*Kölliker*. $\times 30$). *a*, pulmonary vesicles filled artificially with wax; *b*, the margins of the smallest lobules.

The muscular coat is continued on to the infundibulum and small air-ducts, but upon the alveolar walls it exists only as a few scattered cells here and there near the air-ducts.

The elastic fibres are continued on to the alveoli and form its framework.

The alveoli or vesicles, which measure about 0.20 mm. ($\frac{1}{120}$ -inch) in diameter, are packed close alongside of each other, and assume, in consequence, an irregular polyhedral form; though, for the most part, grouped in bunches on the infundibula they occur also isolated and sessile.

They are composed of thin structureless membrane lined internally, as already described, by a continuous layer of flat squamous cells with a few granular cells interspersed, and supported externally by numerous fine, occasionally branching, elastic fibres continuous with the external elastic coat of the bronchioles. To these fibres the elasticity of the alveoli is due. A few scattered muscle fibres are found near the entrance of the air-ducts.

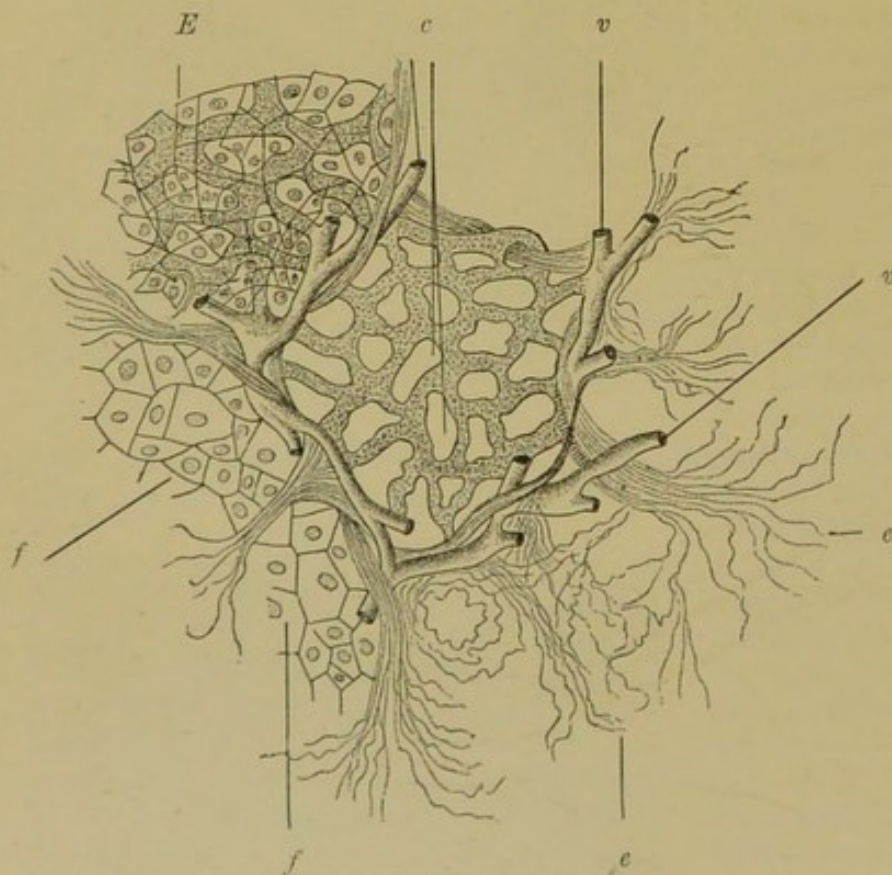


Fig. 8.

Semi-diagrammatic representation of the air-vesicles of the lung (*Landois and Stirling*). *v*, blood-vessels at the margin of an alveolus; *c*, capillaries; *E*, relation of the squamous epithelium of an alveolus to the capillaries in its wall; *f*, alveolar epithelium shown alone; *e*, elastic tissue of the lung.

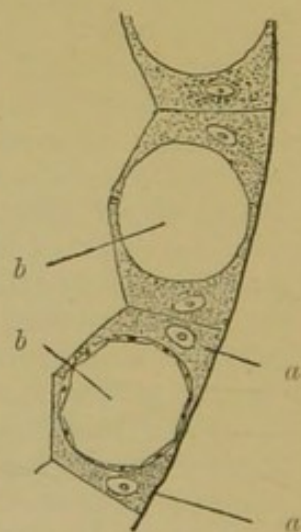


Fig. 9.

Diagram of alveolar wall, to show the capillaries surrounded by epithelial cells; in one the wall of the capillary is indicated by cells. *a*, epithelial cells; *b*, capillaries.

The blood-vessels form freely anastomosing loops round the vesicles, and from these loops a fine network of thin-walled capillaries spreads over the vesicles, a single layer of capillaries lying between any two contiguous alveoli, and, when distended, projecting into the lumen of each.

Among the elastic fibres there exists a network of branched connective tissue cells, contained as usual within lacunæ, which form the radicles of the lymphatics. Between the flattened squamous cells are minute pores, *pseudo-stomata*,

which communicate with the lacunæ. Through them the lymphatic cells, a few of which are always found on the surface of the air-vesicles, migrate and carry with them into the lymphatics any foreign substances which may have found their way as far as the alveoli.

Pseudo-stomata are peculiar cells of connective tissue type existing between the cells of the bronchial mucous membrane and of the alveoli and sending out processes, which anastomose with the similar cells of the sub-epithelial layer. In the ordinary condition these cells completely fill up the space in which they lie, but when the alveolus or bronchus is distended actual channels or real stomata are formed.

The number of alveoli has been calculated to reach the enormous figure of 725,000,000 with a superficial area of about 90·8 square metres, which is 100 times greater than the body surface (·8 to ·9 square metres).

The lymphatics of the lung.—The lymphatics of the lung constitute a superficial and a deep system. The superficial (subpleural) lymphatics lie immediately beneath the pleura and form a network, the efferent trunks from which run along the pulmonary ligaments to the root of the lung, communicating on their way with the deep lymphatics.

The deep lymphatics remain within the substance of the lung and accompany the branches of the vessels (perivascular) and of the bronchi (peribronchial). These two sets of vessels anastomose freely with each other as they run towards the root of the lung, where they end in the bronchial glands.

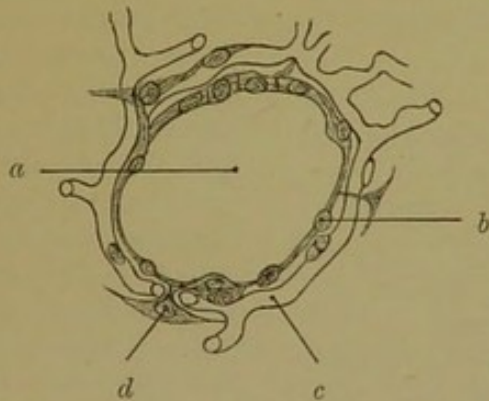


Fig. 10.

Transverse section of an alveolus from the lung of a guinea-pig (*Klein*). *a*, alveolar cavity; *b*, lining epithelium; *c*, capillary blood-vessels injected. They are represented as uninjected and not nearly so numerous as in the actual preparation, in order to make the drawing not too complicated; *d*, interalveolar connective tissue corpuscles sending processes between the epithelial cells of the alveoli, pseudo-stomatous tissue.

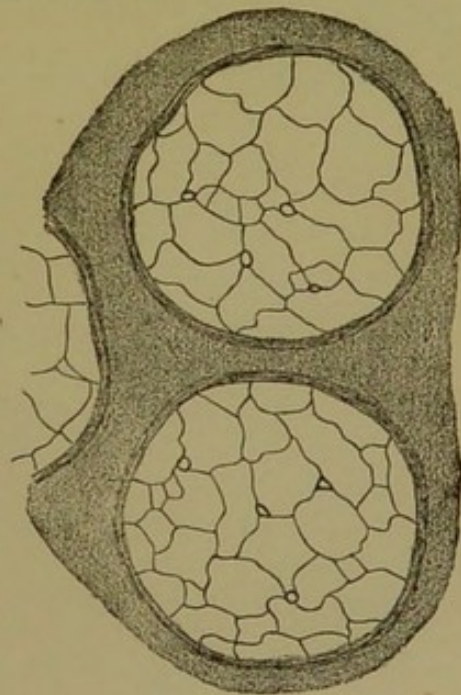


Fig. 11.

Section through a guinea-pig's lung, showing lining epithelium of the alveoli and between them small holes, pseudo-stomatous canals (*Klein*).

The lymphatic vessels follow the connective septa of the lung, and all contain valves.

The lymphatics take origin—

1. In the walls of the alveoli.
2. In the mucosa of the bronchi; and
3. In the pulmonary pleura.

1. In the walls of the alveoli the radicles consist of irregular lacunæ round the branched connective cells, which communicate with the sub-pleural plexus on the one hand and with the perivascular and peribronchial vessels on the other,

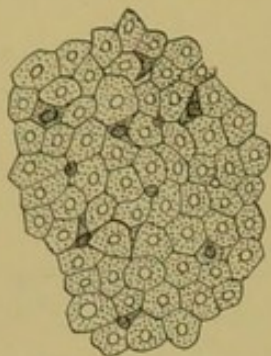


Fig. 12.

Surface view of epithelium of bronchus of a rabbit, showing the epithelial cells, viewed from the surface and appearing, therefore, as a mosaic of polyhedral cells, and between them nucleated branched cells (Klein, ix. A).

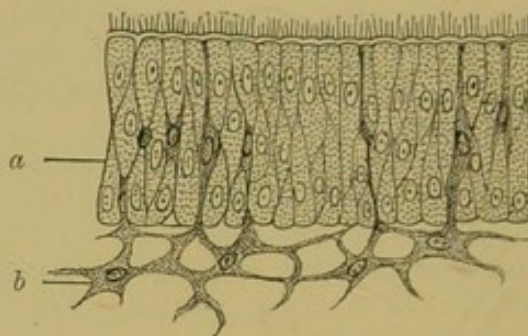


Fig. 13.

The same, seen in profile. *a*, epithelium with intra-epithelial nucleated cells, pseudo-stomatous cells in connection with; *b*, nucleated cells of sub-epithelial mucous membrane.

and by means of the *pseudo-stomata* they are also in communication with the interior of the alveoli.

2. In the walls of the bronchi they originate in the sub-mucosa and unite to form

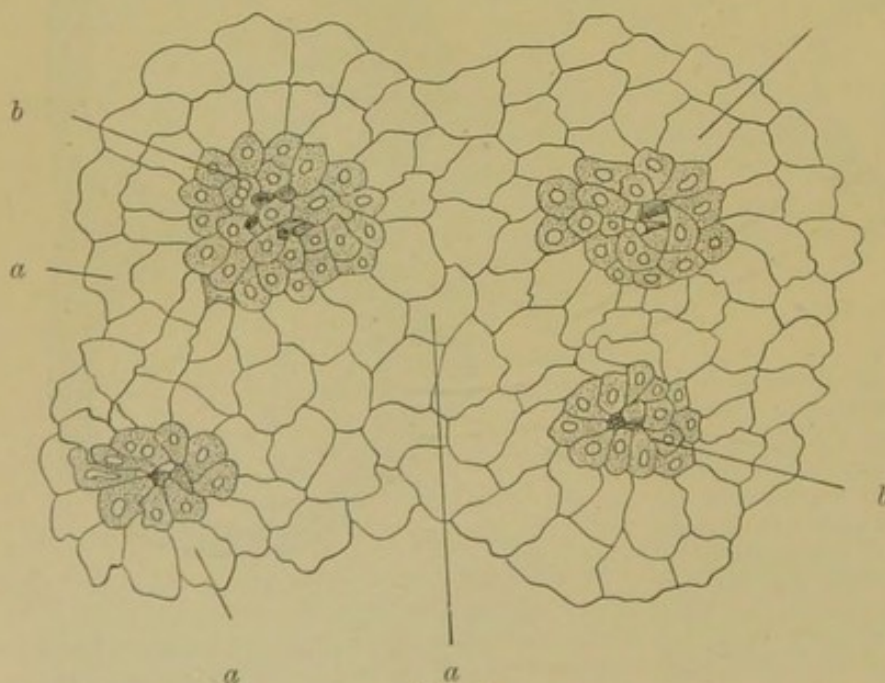


Fig. 14.

Surface view of the endothelium of the pulmonary pleura of the rabbit in the condition of chronic pleuritis (Klein). *a*, general flattened epithelium; *b*, germinating endothelium round stomata, these latter being plugged with fibrinous material, stained dark with nitrate of silver.

the peribronchial vessels which freely anastomose with the perivascular trunks. These radicles communicate also by means of *pseudo-stomata* with the interior of the bronchi.

3. In the pulmonary pleura, they arise in spaces between the connective tissue bundles and are in connection, by stomata, with the pleural cavity.

The free communication of the lymphatics of the lung with the cavity of the pleura by means of stomata and with the in-

terior of the alveoli and of the bronchi by *pseudo-stomata* provides for the rapid absorption of fluid or for the removal of fine solid substances by the

lymphatics. This process is aided by the movements of respiration, which distend the stomata and lymphatics on inspiration and compress them on expiration; this alternating opening and closing, aided by the arrangement of valves in the vessels, directs a constant stream of lymph from the periphery towards the root of the lung.

The pleura.—The *pleura* forms a complete investment for each lung (the visceral layer) and also a lining to the interior of the thorax (the parietal layer). The two layers are continuous only at the root of the lung and for a short distance from the root of the lung down to the diaphragm (the *ligamentum latum pulmonum*). Between the two pleural sacs the mediastinal space is left in which the heart and other important structures are found. The pleura is in intimate relation with the pericardium on each side.

The pleura does not quite reach down inferiorly to the attachments of the diaphragm nor do the lungs quite fill up the whole pleural cavity, except on deep inspiration. There is a small space left on ordinary inspiration (the complementary space) where the two layers of the pleura are in contact.

The structure of the pleura is that of other serous membranes, viz., a fibrous basis covered with endothelium.

The cells, in the parietal layer, are flattened squames, but in the visceral layer they are in places granular, as in the alveoli. The number of these granular cells varies, as in the alveoli, with the distension of the lung. Upon the surface of the membrane numerous stomata exist surrounded by germinating epithelium.

The blood-vessels.—The *blood-vessels* of the lung belong to two different systems, the pulmonary and the bronchial.

The *pulmonary arteries* accompany the bronchi, but divide more frequently, especially towards their terminations.

It is usually stated that they do not anastomose with each other. This is not correct, for Zuckerkandl has proved that the anastomosis between the branches of the bronchial and pulmonary arteries is very free, and that some of the anastomosing branches are quite large; of the bronchial tubes the large divisions are supplied by the bronchial artery only, and the smallest by the pulmonary only, but the whole intermediate portion receives a supply from both sets of vessels, which freely anastomose.¹

The terminal arterioles supply one or more contiguous lobules and do not anastomose with each other. They form round each vesicle a ring which freely anastomoses with those of the adjacent vesicles supplied by the same arteriole.

The capillaries from these rings arise and form a network with very fine meshes over the whole alveolus. The capillaries are very small and measure only $\frac{1}{2500}$ to $\frac{1}{3000}$ inch in diameter. They lie immediately beneath the epithelium and form but a single layer between two contiguous alveoli, so that when distended they project into each. Some fine vessels are supplied to the terminal bronchi where they anastomose with those derived from the bronchial artery.

The *pulmonary veins* are derived from the capillary network and lie on the opposite side to the artery. The veins freely anastomose with each other as they grow larger and are joined by branches from the bronchial veins. They then travel along the septa to the root of the lung, the artery being usually above, and the vein below, the bronchial tube.

The pulmonary veins have no valves, and are said to be smaller in capacity than the arteries, water being given off in the lungs. The area of the pulmonary capillaries is also stated to be less than that of the systemic capillaries, so that the blood stream through them must be more rapid.

¹ *Sitzungsber. d. k. Akad. d. Wissensch.* (Vienna), vol. lxxxvii.

The pulmonary veins carry arterial blood and the arteries venous blood.

The *bronchial arteries* arise from the aorta or intercostals, and number one, two, or three for each lung. They accompany the bronchi as they divide.

They are distributed to the lymph glands at the root of the lung, to the walls of the blood-vessels, to the interlobular septa, to the bronchial mucous membrane, being continuous in the smallest branches with the plexus derived from the pulmonary artery, and lastly to the visceral pleura. Here they may be distinguished from the pulmonary vessels of the superficial alveoli by their tortuous courses and their open arrangement, by lying outside the lobular tissue and by ending in the superficial set of bronchial veins.

The smaller bronchial veins anastomose with the pulmonary veins. The bronchial veins open usually on the right side into the vena azygos and on the left into the intercostal.

A few small vessels reach the lung and pleura along the ligamentum latum pulmonum from the lower intercostals.

The Nerves.—The *nerves* of the lungs are derived from the anterior and posterior pulmonary plexus, and consist of branches from the vagus and the sympathetic. They accompany the bronchi, lying outside the cartilages, and being in close relation with the branches of the bronchial arteries. They contain medullated as well as non-medullated fibres, and numerous small ganglia.

Their mode of termination in the lung is not yet ascertained. Some fibres pass to the bronchial muscle, some to the blood-vessels, and possibly some to the mucous glands.

It is upon the bronchial muscle that the lung-tonus to a great extent depends, and the nerve which governs this is the vagus.

When the vagus of one side is divided, the bronchi of that lung dilate; if the peripheral end be then stimulated, they contract; if the central end be stimulated, the other vagus being intact, contraction also occurs, but not to the same extent, so that the vagus contains both centrifugal and centripetal fibres controlling the bronchi. Lastly, asphyxia, which causes contraction if the vagi be intact, has not this effect if the vagi have been divided.

The root of the lung and the mediastinum.—The lungs are free to move in all directions within the thorax, save at the root where the vessels and bronchi enter, and along the mediastinum where they are attached by the ligamentum latum pulmonum.

The root of each lung is composed of the bronchus and large blood-vessels, with the nerves, lymphatic vessels, and glands connected together by areolar tissue, and surrounded by a sheath of the pleura.

The root of the right lung lies behind the superior vena cava, and a part of the right auricle, and below the vena azygos, which arches over it to enter the superior cava.

The order of the vessels is, from above downwards and from behind forwards :
Bronchus, artery, vein.

The root of the left lung passes below the arch of the aorta in front of the descending aorta, but on this side the artery lies above the bronchus.

Each bronchus and each pulmonary artery divides into two branches, an upper and a lower, before entering the lung, the lower being the larger. On the right side the upper division of both artery and bronchus gives off a separate branch to the middle lobe. The right bronchus is larger than the left.

The size of the bronchi vary in direct proportion to the size of the lung and also to the respiratory capacity.

The phrenic nerve descends over the root of each lung, and the pneumogastric nerve lies behind.

The nerve-plexuses lie in front and behind the root and main bronchi, and are called anterior and posterior accordingly, the posterior being the larger. They are formed chiefly of branches from the vagus and the sympathetic.

The lymphatic glands are grouped for the most part round the bifurcation of the trachea and the bronchi.

THE PHYSICAL EXAMINATION OF THE CHEST.—It is hardly necessary to devote much space to this subject, upon which so many comprehensive treatises have been written. Various parts of it will be dealt with as occasion arises hereafter. There are but two points which may with advantage be considered now, for they cannot be introduced conveniently elsewhere. I refer to the use and meaning of the terms *bronchial breathing* and *râles*.

BRONCHIAL BREATHING.—Auscultation is not really a difficult subject. It requires some little preliminary instruction, and after that its mastery is only a question of attention and practice; yet there is no doubt that it often appears confusing to students. The difficulties, however, are chiefly of our own making, and lie not in things but in words; for the facts of auscultation, their significance, and their relation to pathological lesions, are well known and understood, while the confusion lies in the technical terms used to express the phenomena observed. Among these technical terms the chief offenders are two, namely, *bronchial breathing* and *râles*.

Technical terms are convenient, but to be really useful they must be strictly defined and accurately used, otherwise they introduce confusion instead of simplicity, and that is the case in auscultation. If we could only get rid of technical terms, and merely describe in simple language what we actually hear, there would be no confusion and little difficulty.

Now why, if the facts are so plain and so easy to comprehend, is so much time and energy spent in discussing the meaning of terms? It is for the simple reason that the different terms are regarded as indicating different pathological lesions. These lesions are the important conditions to recognise, and the use of a wrong term would, therefore, imply a wrong conclusion as to the pathological condition. Unfortunately, the same term has not always the same significance, that is to say, it does not always, in the mind of every teacher or examiner, correspond with the same pathological lesion, and it is because this difference of usage of terms is not recognised and taught, that so much confusion is introduced into the subject.

I propose first of all to consider what the facts of auscultation are; what their meaning is; and why it is important to give certain of them special names. I shall then be able to show where the confusion comes in, and how it will be possible to avoid it.

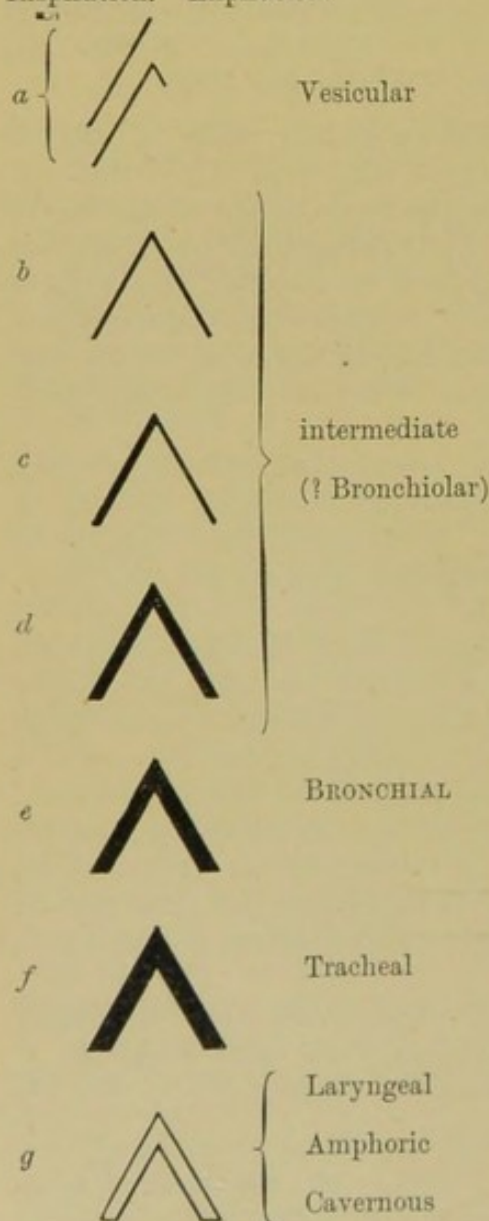
The Breathing Sounds.—As the air passes in and out of the larynx and air tubes it makes sounds which are carried throughout the air tubes by the column of air contained in them. These sounds are very obvious if we listen over the larynx, and they have a certain loudness and a certain character. If we listen over the trachea we hear the sounds as before, but with somewhat diminished loudness and different character. Again, over the bronchi—that is, beneath the manubrium sterni in front, or behind right and left of the second or third dorsal vertebra—the breathing sounds are still heard, but are further diminished in loudness and altered in character. Finally, if we listen in the

axilla or at the base behind—that is, as far as possible away from the air tubes—the breathing sounds are entirely different from those heard in the other places specified. The breathing sounds heard in these various parts have since Laennec's time been called laryngeal, tracheal, bronchial, and vesicular respectively.

Pathologically, these terms become of significance when they are heard in places in the chest where they should not normally be heard at all. If, in places where vesicular breathing ought normally to be heard, vesicular breathing disappears, and in its stead some other kind of breathing is heard, the conclusion

DIAGRAMMATIC REPRESENTATION OF THE BREATH-SOUNDS.

Inspiration. Expiration.



may be drawn, subject to a few reservations to be dealt with later, that that portion of the lung is in a condition of disease; and, the more abnormal the breathing is, the greater the pathological changes which have taken place. This is the real reason why so much importance is attached to these terms, for they are regarded as the measure of the pathological change.

Over the larynx, inspiration is heard as well as expiration; both are loud, noisy as it were, and both have a peculiar character, such as might be produced in a large hollow space as we know the larynx to be. On account of this hollow character this kind of breathing when heard elsewhere is often called, not laryngeal, but amphoric or cavernous. Over the trachea, inspiration and expiration are both heard. The sounds are still loud and noisy; though not quite so loud as over the larynx, they are somewhat raised in tone, and have lost

some of their hollow character. Over the positions described where bronchial breathing may be heard, both sounds are still audible, but with a still higher pitch and a much harsher character. When, in disease, instead of vesicular breathing other sounds are heard, they are called *laryngeal*, *tracheal*, or *bronchial*, according as they approach most nearly to the characters of the breath sounds heard in these respective places.

Now let us turn to vesicular breathing and analyse what it is that we hear in those parts of the chest which are far removed from the large bronchial tubes. Here there is little noise: the sounds are muffled, blowing, soft, almost sighing in character, and what is more remarkable is that they are no longer double. We hear the blowing murmur with inspiration, but with expiration we either do not hear anything at all, or we hear the blowing murmur only for a short time during the early part of expiration. Thus vesicular breathing is characterised first by the absence of noise, secondly by its blowing character, and thirdly by the absence, or diminution in length, of the expiratory sound.

All the forms of breathing may be represented diagrammatically by lines, the varying thickness of them indicating the amount of noise. Thus vesicular breathing may be indicated by a thin line for inspiration, with a very short thin line for expiration, if it be audible at all (see diagram *a*); bronchial breathing by two thick lines (diagram *e*); tracheal breathing by two thicker lines (diagram *f*). Of course, between the diagram which represents vesicular breathing and that which represents bronchial breathing various transitional forms might be drawn, and these would correspond with the transitional forms of breathing which are actually observed. Laryngeal breathing (*g*), or, as it is called, "cavernous breathing," may be represented by two double lines enclosing a space, instead of two thick solid lines, thus indicating its hollow character.

Now, just as the air tubes pass without a break from one end to the other, the larynx into the trachea, the trachea into the bronchi, the larger bronchi into the small bronchi, and the small bronchi into the bronchioles and vesicles, the lines of division between them being purely arbitrary, so it is with the different kinds of breathing to which the different names are given.

The earliest pathological change from healthy vesicular breathing is that expiration becomes audible throughout its whole length (*b*); the next change is that inspiration becomes louder than it should be (*c* and *d*). By - and - bye both inspiration and expiration are equal in loudness and intensity, and subsequently come to have the character to which the name bronchial breathing is given.

Between bronchial and vesicular breathing we have many transitional stages, and the question is how far the term "bronchial" shall extend. No one who has ever heard true or typical bronchial breathing such as is met with in acute pneumonia can fail to have been struck by it or is likely to forget it. It is so characteristic and peculiar, and it means so much pathologically, that it is evident a special name is required for it.

It seems a pity to use the same name which we employ to designate this remarkable phenomenon for a variety of other forms of breathing which have not the same pathological significance. And this is the gist of the whole matter; for while

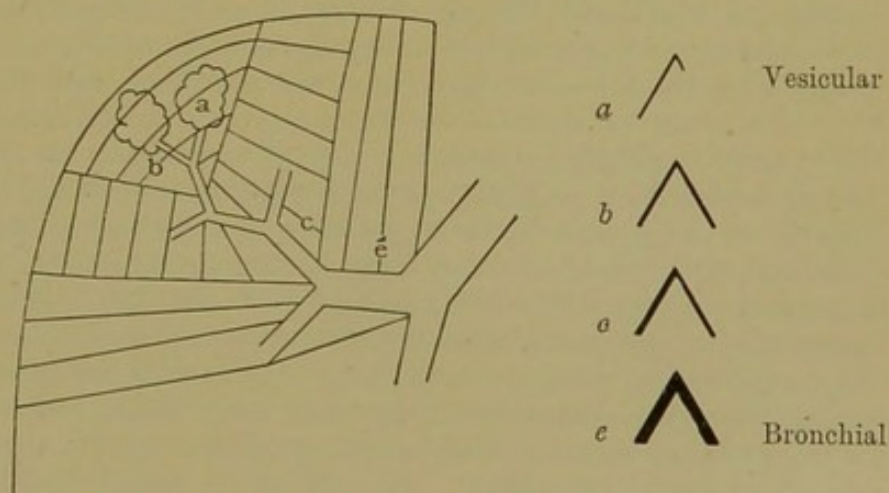


Fig. 15.

Showing the relation assumed to exist between the altered breathing sounds and the amount of pathological lesion.

some strictly limit their use of the term to that very peculiar form of breathing most frequently heard in pneumonia, others employ it as a general expression for many kinds of pathological breathing, from the slightly modified vesicular up to the typical bronchial breathing. The importance of the question is at once apparent if we look at it from a pathological point of view. In the one case, we indicate a massive, considerable consolidation of the lung, and, in the other, a consolidation which may be comparatively trifling.

Now if, instead of using technical terms, we described in simple ordinary language the phenomena we hear, all this difficulty and confusion would at once vanish. Thus we might put into words what we really have to observe in fact. We have first to listen whether expiration can be heard; if so, whether it is as long and as loud as inspiration. Then, whether inspiration or expiration has any abnormal characters in quality and tone. These changes can all be described in plain and simple language, and without the introduction of technical terms at all. In this way it is easy to make the subject intelligible and interesting, and to avoid all the confusion which technical terms introduce.

Now let us turn for a moment to the cause of the respiratory sounds, and consider where these sounds are produced; why it is they are modified in different parts of the air-passages; and how it comes that vesicular breathing is so different from all the other kinds. The sounds of breathing are produced chiefly at or about the glottis, and they are diminished in intensity, that is, they become less and less loud the farther away we pass from the glottis; thus distance from the seat of production accounts for the diminished noise we hear in different parts of the chest. As we pass farther and farther away from the larynx, the sounds become not only less loud, but rise also in pitch; these changes are to be connected with the size of the tubes over which we are listening.

In vesicular breathing we have another and new factor added. In the vesicles of the lungs the air column is, as it were, broken up by a number of partitions into compartments or spaces of different sizes. In this way the sound brought by the air-tubes is so dispersed and dissipated that much of it becomes lost; the vesicular structure of the lung may be likened to a feather pillow, and the vesicles muffle the sound in a similar way. The result is that so long as the vessels are healthy but little of the breathing sounds are audible at all; we hear inspiration only, and little or nothing of expiration. As soon as the vesicular structure of the lung becomes more homogeneous—that is to say, the irregular air spaces become solid—the muffling of the sounds is less, the breathing sounds in the smaller tubes are heard, and consequently expiration becomes audible. As the consolidation extends, inspiration and expiration become more distinct, and, finally, when the consolidation becomes massive and extends, we may suppose as far as the large bronchi, the sounds produced in them are audible, and we get typical bronchial breathing. Thus it comes about that the degree of alteration in the respiratory sounds is an index of the amount of consolidation, as is represented in fig. 15.

If, on ordinary quiet breathing, the sounds produced by the passage of air through the larynx are transmitted to the vesicles of the lung, it follows that they will be transmitted with greater distinctness when the breathing at the larynx is loud, and, when it is very noisy, the sounds heard over the vesicles of the lung may resemble in character those audible over some of the air-tubes. It might be then difficult to say whether the cause of this exaggeration of the breath sounds lay in the larynx or in the vesicles of the lung.

Even simple increase in the rapidity of breathing produces more noise, and then both inspiration and expiration may become audible over the vesicles of the lung. This very frequently happens in children, and was named by Laennec “*puerile*,”

a term which is still retained, but which is often applied not to the breathing of children only, but to similar kinds of exaggerated or loud breathing in adults.

In children and in adults, puerile breathing may very closely resemble some of the pathological kinds of breathing previously described, but in most cases it retains something of that muffled character which has been described as peculiar to vesicular breathing.

If patients are told to take a deep breath they frequently begin to make noises with the mouth, pharynx, or larynx. This must be prevented, for the noises thus produced may mask the other changes in the character of the breathing for which we are looking.

The most serious difficulties are introduced when disease of the larynx or trachea has led to stenosis or narrowing of the tube, so that the air has to pass through a constricted aperture. It must, therefore, pass at a greater rate, and in doing so will produce a greater noise. This noisy laryngeal and tracheal breathing has received the name of "stridor." Stridor complicates diagnosis very considerably, for the sounds being loud and transmitted far and wide are audible over the vesicles of the lung. Under these circumstances the diagnosis of the condition of the vesicles of the lung by auscultation becomes extremely difficult, if not impossible, and mistakes may be made in either direction: either disease of the vesicles may be diagnosed when none exists, or the absence of disease inferred when considerable disease is present.

The last difficulty that needs to be mentioned is in connection with rhonchus. This sound is often of considerable intensity, so that it is frequently heard even far away from the chest. It is also often double, that is to say both inspiration and expiration are audible. As a rule, however, rhonchus is easily distinguished from other forms of pathological breathing by its sonorous or musical character; but if these are absent, and there is nothing but the increased loudness and harshness of respiration, rhonchus may come to resemble bronchial breathing very closely, and I have seen instances in which it would be very difficult by the ear alone to say which was present. However, this difficulty is not likely often to arise.

Now, I think we are in a position to see the whole matter. "Bronchial breathing" introduces confusion in our ideas, because it is used in different senses by different persons. Although all agree in calling the extreme form "bronchial breathing," there is great difference of opinion as to the term to be applied to the intermediate forms, and some attempt to solve the difficulty by calling them all bronchial. It would be well if we had names for those intermediate forms which lie between bronchial breathing and vesicular, but none have been accepted. They have been called "intermediate," "undefined" (*unbestimmtes*), but these terms are unsatisfactory, indefinite, and not in general use. As our terminology is in the main anatomical, we might carry it a little further, and while speaking of laryngeal, tracheal, and bronchial breathing, add to the list "bronchiolar" breathing. There is an objection to the introduction of a new term into auscultation, and I am not inclined to advocate it. Still if we could agree upon some such intermediate term, we should do away with much of the confusion which now exists.

Râles.—I now turn to the second term which is a source of confusion—namely, "*râles*." The sounds heard on auscultation fall into two groups: first, modifications of sounds heard in the healthy chest; and, secondly, new, added, or adventitious, sounds never heard in health at all. The second group consist of rattling, bubbling, crackling, and rubbing sounds. The rubbing sounds are called "friction," and the rest have been named "*râles*."

Thus, *Râles* is a general term, with several varieties. Laennec described five different kinds of *râles*, namely : 1, crepitant ; 2, mucous ; 3, sonorous ; 4, sibilant ; 5, dry crackling. This is really a good classification, and if it had been adopted in its entirety, as Laennec intended, there would be little confusion. Unfortunately, this is not done.

1. *The Crepitant Râle*.—This is fine crackling sound, such as is heard when the hair is rubbed between the fingers over the ear, and is now more commonly known by the name of "fine hair crepitation." It is heard most frequently in the early stages of pneumonia, but it is also met with in some other conditions of lung disease.

It is usually explained as being due to the opening out, by the entrance of air, of collapsed air vesicles which have stuck together, the crackling sound being produced on the separation of the two surfaces.

It would be well to place the fifth variety of *râle*, viz., *dry crackling*, also under this heading—a noise which Laennec compared to the crackling of a dry bladder, and described as met with occasionally in emphysema. It is not a fine, but a large, coarse crackling (*râle crépitant sec à grosses bulles ou craquement*).

Between these two forms of crepitation Laennec placed another variety which he called "subcrepitant," not quite so fine as the fine-hair crepitation, nor yet so coarse as the other form. This was observed frequently in cases of early phthisis. Laennec, however, did not say, as subsequent writers have done, that this subcrepitant *râle* was pathognomonic of phthisis. Nor is the statement true, for this *râle* may be heard in many conditions of the lung which are not of tuberculous nature, and notably in bronchopneumonia.

This term "subcrepitant *râle*" is one which is not required ; it is very indefinite, difficult to comprehend and to explain, and therefore would be best formally banished from our nomenclature, as indeed it now almost is in practice.

All these forms of *râle* Laennec thought not to be due to fluid, and he therefore called them "*dry râles*."

2. *The Mucous Râle*.—Mucous *râles* are crepitant, crackling sounds also, but regarded as due to the bursting of air bubbles in fluid. They have therefore been called "*moist râles*." These *râles* vary in character according as they make more or less noise, or are produced in larger or smaller tubes, and so they give the impression of being caused by large, medium-sized, or small bubbles bursting. They are therefore described as small, medium, and large-sized crepitation. The largest of all is often called "bubbling," and in its coarsest form is known as "the death rattle," from which the name "*râle*" was originally taken. All these mucous *râles* alike are now termed "crepitation."

The larger forms of crepitation are easy to recognise by the ear, and are obviously due to the bursting of bubbles in fluid. The small crepitation has been likened to the crackling of salt when thrown on the fire, or the bursting of very fine soap bubbles in lather. This small-sized crepitation is by no means easy to distinguish from the crepitant *râles* just described, and which are now called "fine-hair crepitation." Nor is it easy sometimes to distinguish it from a form of friction in which the sound, instead of being one of rubbing, has this crackling character.

All the sounds alike may be modified by the condition of the lung which surrounds their place of production. Thus, if the lung be solid, they may acquire a sharp, clear character, and appear as if produced immediately beneath the ear. In this respect they may be compared with the clear transmission of the voice sounds which is called "pectoriloquy." Again, if the crepitations are produced in a cavity, they may not only be large and bubbling, but, in addition, have a

ringing, hollow character, strictly comparable with amphoric breathing or amphoric reasonance.

3 and 4. *Sonoro-sibilant Râles*.—The third and fourth variety of *râles*, namely, the sonoro-sibilant *râles*, are now universally called “rhonchus” and “sibilus” respectively, the former being a snoring or sonorous, the latter a hissing, piping, or whistling sound; and they both, as the epithets applied to them imply, frequently have a musical character.

It is now evident that Laennec's classification may be arranged somewhat differently. The sonoro-sibilant *râles* (the third and fourth varieties) are now called “rhonchus” and “sibilus.” All the other kinds of *râles* are comprehended in the general term “crepitation,” other descriptive terms being added to it to indicate what kind of crepitation it is.

Now, I think it becomes clear where the confusion is introduced in respect of the term “*râle*.” The term is sometimes used strictly as a general expression, as Laennec used it with all the varieties which he described. Others, finding that “rhonchus” and “sibilus” are in such common use, and forgetting that they form but the “sonorous” and “sibilant” groups of Laennec's *râles*, use it as simply representing the remainder of Laennec's varieties of *râles*—namely, those which we describe for the most part as “crepitation.”

“*Râle*,” therefore, like “bronchial breathing,” nowadays has two meanings, and it is impossible to say beforehand which meaning it is intended to have. It may be used as a general expression, in the same way that Laennec employed it, or it may be used as meaning nothing else than crepitation. It is evident, therefore, that we do not want the term “*râle*,” and I think it would be well if we could get rid of it.

To the other terms, “mucous *râles*,” “moist *râles*,” and “dry *râles*,” there are other objections. Terms in auscultation should simply be descriptive; they should state facts and facts only, and not mix up facts with theories or inferences drawn from them. But this is just what these terms do. The mucous *râle* is a *râle* of a crackling character; this is a fact. It is due, it is supposed, to the bursting of bubbles in fluid, the fluid being mucus; these two implications are opinions only. It is evident that the fact may be correct and the theories or inferences wrong.

Again, with the dry *râles* the sounds are crepitant, not bubbling. This is descriptive of their character. It is assumed that they are not due to the passage of bubbles of air, which is an opinion. The dry *râles* fall into two kinds, the “crepitant” and the “sonoro-sibilant.” These are easily distinguished by the ear. Rhonchus and sibilus are called “dry” sounds, and are supposed to be due to secretion which lines the tube, and thus narrows its lumen; yet if the secretion be a little more in amount and lie not upon the walls of the tube, but get into the cavity of the tube, bubbles may be produced, and thus crepitation arise, so that actually the same secretion may produce a moist or a dry *râle* somewhat at random.

Again, in respect of some of the forms of fine crepitation, the ear recognises the sounds readily, and they are described correctly as small crepitation, but the ear alone is quite unable to decide in many cases what the cause or mode of production of these sounds is. The same fine crepitant sound may have four modes of origin. It may be due (1) to the opening out of collapsed air vesicles, the crepitant *râle* of Laennec; (2) to the crepitation of emphysematous lungs, the “dry crackling crepitation” of Laennec; (3) to the bursting of minute air bubbles, the small mucous crepitation of Laennec; or (4) to pleuritic friction. According to the method of production we might call three of these “dry” *râles*, and the other “moist”; but it might be quite impossible

by the ear alone to decide to which cause the particular crepitation was due.

It is dangerous for anyone, whether student or teacher, thus to mix up fact and inference in a common term, and it is fatal to clear teaching. I may make my meaning clear if I take another example. Nothing is commoner in the examination of the heart than to hear a murmur described as "mitral systolic." Now, what does this expression really involve? It means that there is a murmur audible at the apex, systolic in time, transmitted in certain directions—that is, to the axilla and to the interscapular space, associated with certain changes in the heart itself and in the character of the heart sounds elsewhere, and because of these facts we infer that it is produced by regurgitation through the mitral valve. The proper place for this conclusion would be at the end of the description of the case, where the diagnosis comes, and not in the commencement of it under the physical examination, where we have only to describe the facts observed, and not the conclusions drawn. At any rate, what is a more common experience with students than to hear them say that there is a mitral systolic murmur, the simple fact being that there is a systolic apex murmur, while their opinion that it is due to mitral disease, tacitly expressed by the phrase employed, is quite as likely to be wrong as right? This tendency, therefore, to mix up facts and inferences in a common term should be discountenanced.

What we require, in an account of the physical examination, is a statement of facts observed, not of inferences drawn. As long as the terms employed are descriptive only they are useful; as soon as they involve an inference or a theory they become dangerous, for the facts may be right and the conclusion or theory wrong.

The term "*rôle*" thus becomes a source of confusion, first because it is used in different senses either as a special term or as a general one; and, secondly, because the addition of such descriptive terms as "moist" and "dry" imply a theory about which there may be great difference of opinion. It would be well, therefore, if we could banish "*rôle*" altogether from our vocabulary. We do not want it, and a term which is redundant is often the cause of confusion.

2. MALFORMATION OF THE RESPIRATORY ORGANS.

The respiratory organs develop as buds from the intestinal track, and the diaphragm as a membranous outgrowth between the lungs and the Wolffian bodies. Arrest of the development may occur, in any degree, but grave defects are incompatible with life. Malformations are more of teratological than of pathological interest. If a child is to live, the defects can only be of slight degree.

Congenital Atelectasis.—The most important defect is that in which some of the alveoli do not expand after birth, but remain in the foetal state. If this is not so extensive as to be incompatible with life, connective tissue changes take place in the atelectatic parts, associated with more or less bronchiectasis.

If the affection involve a whole lobe the bronchi may become saccular and the condition be produced which has been described as "the Gruyère Cheese" or "Turtle" lung. Usually, however, this is the result of post-natal disease, especially broncho-pneumonia.

If the change be not so extensive, hypertrophy of the rest of the lung occurs, which compensates for the defect, so that no symptoms may be produced.

Mere irregularity in the division of the lungs into lobes or in the number or mode of division of the bronchi is chiefly of anatomical interest, and is seldom of clinical importance, for it need not and does not generally affect health.

Congenital defects of the diaphragm lead to the passage of some of the contents of the abdomen into the thorax, and constitute one of the chief varieties of diaphragmatic hernia, under which heading they will be considered.

3. GENERAL CLASSIFICATION OF DISEASES OF THE RESPIRATORY ORGANS.

The simplest and most convenient classification is anatomical. Accordingly, the diseases of the respiratory organs may be grouped in the following way:—

1. Diseases of the air-passages.
2. Diseases of the lungs.
3. Diseases of the pleura.

To these may be added another group, viz.:—

4. Affections of the respiratory mechanism.

In this last group some are neuroses, *e.g.*, asthma, spasmodic croup, and whooping-cough, and find their natural place in connection with diseases of the air-tubes, while others depend upon neuro-muscular defects, which have for their result paralysis of the chief respiratory muscles, viz., the intercostals and diaphragm.

This general classification will be followed as far as possible.

4. DISEASES OF THE AIR-PASSAGES.

The affections of the air-passages are closely linked together by the fact that the chief symptoms they produce are due to the mechanical impediment offered to the passage of air into or out of the lungs.

5. RESPIRATORY OBSTRUCTION.

Though the nose, mouth and pharynx form the commencement of the air-passages, affections of these parts rarely give rise to marked obstruction.

Obstruction in the Nose.—If the air finds difficulty in passing through the nose, breathing is carried on through the mouth instead, and the patient has no trouble beyond some little dryness of the mouth, slight discomfort in eating, and perhaps a nasal tone of voice.

In suckling infants the discomfort may be so great as to lead them to refuse the breast for the time (*e.g.*, snuffles).

Obstruction in the Pharynx.—*Acute Tonsillitis* often leads to such enlargement as almost to completely close the entrance to the pharynx; the breathing is then carried on through the nose, but respiratory difficulty rarely arises unless the parts about the glottis become involved.

Chronic Tonsillitis.—Children with chronic enlargement of the tonsils generally breathe noisily, especially during sleep, but they rarely suffer from any difficulty in breathing unless the chronic tonsillitis be associated, as it sometimes is, with chronic pharyngitis and adenoids. Then the breathing may not only be noisy but also short, the voice throaty or nasal, and the hearing impaired from catarrh of the Eustachian tubes.

This obstruction is thought by many authors to be sufficient to produce deformity of the chest, and the pigeon breast often met with in such cases is then referred to this cause; but such children are subject to catarrh of the air-tubes as well, and are frail, poorly developed, and often rickety, so that the evidence in support of this view seems weak.

Grave obstruction in the pharynx rarely arises, unless from the impaction of food or some foreign body in such a position as to obstruct the glottis.

With these and such-like exceptions, it is not until the air-tubes are reached that the common and more serious forms of obstruction are met with, and when respiratory obstruction is spoken of it is usually implied that the air-tubes are the seat of the affection.

The gravity of any particular case will depend upon the seat of the obstruction, upon its nature and amount, and upon the rapidity of its development.

Obstruction in the larynx or trachea is always grave, for it interferes with the entrance of air into both lungs. When one lung only is affected the symptoms may be slight, and even the main bronchus of one side may be completely obstructed without urgent symptoms arising. In these cases much will depend upon the rapidity of development, and thus a small amount of obstruction which has developed rapidly will often produce a greater effect, *i.e.*, more marked symptoms, than a much larger amount which has developed slowly owing to the time which is given in the latter case to the respiratory organs to adjust themselves to the altered conditions.

THE SYMPTOMS OF RESPIRATORY OBSTRUCTION.—

The symptoms fall into two groups:—

1. Those of obstruction in whatever way produced;
2. Those of the disease to which the obstruction is due.

The former can be conveniently considered now, the latter subsequently, as each particular affection is dealt with.

The signs of obstruction are much the same in all forms, and of these the two most characteristic are dyspnœa and stridor.

Dyspnœa may present every degree from slight shortness of breath evident only on exertion up to actual suffocation. It is liable to sudden paroxysmal exacerbations, in one of which the patient may die suffocated. Sometimes the paroxysms are frequently repeated and resemble asthma in character.

The effect of position upon the dyspnœa is often remarkable and of importance in diagnosis. For example, when the obstruction is in the larynx, the patient will probably prefer to lie upon the back with the neck extended, and the respiratory excursions of the larynx up and down will be considerable; if, on the other hand, the obstruction be in the trachea or bronchi, the head is usually bent forward, orthopnœa is the rule, and the respiratory excursion of the larynx is slight. If the obstruction be on one side, the patient not infrequently lies on that side in order to give the other lung free play.

The respiratory rhythm is altered, an attempt being made under the control of the pneumogastric to compensate for the obstruction by prolonging that phase of respiration to which obstruction is offered. When inspiration and expiration are both impeded, both may be prolonged, and thus the respiration rate be much reduced below the normal.

Though the movements of the thorax may be excessive, the expansion is impaired on one or both sides, according to the seat of the obstruction. Inspiratory recession is constant and, in children, where the ribs are yielding, may be extreme.

The voice may be feeble from want of breath and, if the obstruction be in the larynx, may be hoarse or even lost entirely; but, if below the larynx, it is not affected unless the vocal cords are paralysed.

Stridor is very common and almost pathognomonic. It may be constantly present both with inspiration and expiration and also with cough, but it is sometimes not evident until the patient exerts himself or lies in an uncomfortable position. When well marked it may produce vibrations distinct enough to be felt by the hand.

Cough varies much. It is generally a prominent early symptom and is at all times liable to be paroxysmal and violent, but it may be almost absent. The cough is most likely to be severe when the obstruction is either in the larynx or near the bifurcation of the trachea.

Cyanosis is a common symptom where the dyspnoea is great, but in severe cases it is succeeded towards the end, especially in children, by an ashy or leaden pallor which is of grave omen.

The *pulse* is generally rapid and its ratio to the respiration rate altered, owing to the relative slowness of breathing. Towards the end it may become irregular and fluttering, the right side of the heart engorged and dilated, and the veins in the neck distended and pulsating. Not infrequently death happens suddenly at the last from cardiac syncope, *i.e.*, from paralysis due to over-distension.

The patients complain of few symptoms except shortness of breath and oppression in the chest. Pain is frequently absent. It is sometimes felt at the seat of obstruction, but is referred at other times to parts far distant, and may be complained of even after the cause of obstruction has been removed.

The pathological results of obstruction.—These vary greatly according to its seat duration and cause.

If a small or medium-sized bronchus be affected, the corresponding portion of the lung becomes collapsed and subsequently inflamed, while the surrounding parts become emphysematous. The occluded bronchus may even be completely obliterated and reduced to a fibrous cord, and the corresponding part of the lung pass into a state of fibroid induration.

When a larger bronchus is obstructed the lung tissue becomes airless and oedematous, a condition which has been described as solid oedema. In the portions so affected necrotic changes not infrequently occur, and may lead to the formation of a cavity, or inflammation may develop and end in abscess or in gangrene. As the obstructed parts of the lung collapse the rest dilates, and where the whole of one lung is involved, the opposite lung, if healthy, passes into the condition of complementary emphysema or hypertrophy.

When both lungs are involved, advanced changes, such as some of those described, are clearly impossible, but the lungs are found generally oedematous, with patches of collapsed tissue, especially at the bases, and with more or less inflammatory consolidation. Pneumonia, however, in these cases more often depends upon the passage into the lung of some portion of the obstructing body or of some irritating or infective secretion, and it is often found *post-mortem* when it has not been suspected during life.

In the air-tubes, besides the inflammation and ulceration which are liable to occur at the seat of obstruction, dilatation is the lesion most likely to be met with. It is nearly always a local lesion limited to the neighbourhood of the obstruction, and though most common on the distal side, may be present on the proximal side as well, but anything like extensive bronchiectasis is rare, except in very long-standing cases or after the impaction of a foreign body.

Prognosis of obstruction.—*The prognosis*, as regards life, depends chiefly upon the amount of obstruction and upon the relief which may be expected from treatment; hence it is more favourable where the obstruction is high enough to be relieved by tracheotomy. In respect of cure, the prognosis depends entirely upon the cause and duration, and upon the secondary lesions which have been produced.

The diagnosis of obstruction.—This is easy; the only difficulty arises in determining its seat and its cause.

If the obstruction be in the larynx the voice is likely to be affected, and, when the dyspnœa is considerable, the head will be thrown back, and the movements of the larynx on respiration be violent. If there be no external signs, laryngoscopic examination will probably reveal the nature of the affection.

If the obstruction be below the larynx, the voice will not be affected except as the result of recurrent-nerve paralysis, and this the laryngoscope will reveal; orthopnœa is the rule, the head is bent forward and the laryngeal movements are slight.

If the obstruction be in a bronchus, the vocal vibrations, vocal resonance, and breathing sounds will be diminished or absent over the affected part, the movements restricted and the part of the thorax corresponding may be somewhat contracted.

In fatal cases of obstruction the end usually comes gradually, with the signs of carbonic acid poisoning and failure of the right side of the heart, the patients dying comatose. In other cases the fatal result is brought about by some complication, as by pneumonia or by œdema of the lung or larynx, and sometimes sudden collapse occurs which is probably of cardiac origin. All cases alike are subject to sudden paroxysms of suffocative dyspnœa, the causes of which have been referred to.

THE CAUSES OF OBSTRUCTION.

The air-tubes, like other tubes, can be obstructed in three ways—

1. *By plugging within, i.e., by Occlusion.*
2. *By changes in the walls, i.e., by Stenosis.*
3. *By pressure without, i.e., by Compression.*

The relative frequency of these causes varies in different parts of the air-tubes. Thus stenosis is common in the larynx and rare in the bronchi, while compression is rare in the larynx and common in the trachea and main bronchi.

Occlusion, or plugging from within, is in nearly every case due to a foreign body, and although the occlusion caused by the membrane in diphtheria might seem to place diphtheria under this category, still its natural relations are with the other forms of inflammation.

Stenosis, or obstruction caused by changes in the walls, is very frequent in the larynx, but not common in the trachea. In the larynx it may be due to

some form of laryngitis, to abductor paralysis or fixed crico-arytenoid joint, to new growth or stricture; in the trachea, to stricture, tumour, or some form of inflammation.

Compression from without is little likely to affect the larynx, but in the trachea and bronchi it is one of the commonest causes of obstruction. Thus, in the neck, the trachea may be compressed by a goitre or other enlargement of the thyroid, by inflammation or new-growth in the parts about, by a foreign body in the œsophagus: within the chest, by aneurysms of the aorta, tumours and other mediastinal affections, by tumours or abscesses in connection with the bones of the thorax, by tumours of the œsophagus, pericardial effusions, and even some enlargements of the heart.

These various causes are all set out in the annexed table, and will be discussed in the order given.

The three commonest forms, which may be taken as types of the different groups, are membranous laryngitis, mediastinal tumour, and foreign body.

CAUSES OF OBSTRUCTION TO THE AIR-TUBES.

1. <i>Within.</i>	OCCLUSION.	Foreign bodies.
2. <i>In the walls.</i>	STENOSIS.	Some inflammatory affections of the larynx (with which tubercle and syphilis may be included). Œdema of the larynx. Spasm of the larynx. Abductor paralysis. Fixed crico-arytenoid joints. Stricture { (α) of the larynx. (β) of the trachea or New growths } bronchi. Tubercle. Syphilis. Lupus, etc.
3. <i>Without.</i>	COMPRESSION.	
	In the neck.	Goitre and other affections of the thyroid. New growth. Inflammatory swellings.
	Within the thorax.	Thoracic aneurysm. New growths and other affections of the mediastinum. Enlarged glands: inflammatory, tubercular, neoplastic. Large thymus. Cancer of œsophagus. Affections of bones, especially sternum, spine, and clavicle. Emphysema of mediastinal tissues. Pericardial effusion. Dilated left auricle.

6. FOREIGN BODIES.

Foreign bodies of the most varied kinds have been found in the air-tubes. Dusts, however, and fluids like blood or pus, though in one sense foreign bodies, are not included under the term, which is restricted to such substances as would in common parlance be spoken of as bodies, *i.e.*, masses of a certain size.

Foreign bodies gain access, as would be expected, in nearly every case from the mouth or pharynx. They are very frequently derived from the food, and yet they rarely pass into the air-tubes during the act of swallowing unless the parts about the glottis are gravely diseased so that it cannot properly close, or unless its muscles are paralysed or its sensibility impaired. When any of these things happens, as in malignant or other ulceration of the larynx, after diphtheria, or tracheotomy, in bulbar paralysis or central nerve lesions, or again where the patient is unconscious, as in coma, or under the influence of narcotics or anæsthetics, foreign bodies may pass easily into the larynx either during swallowing or vomiting, and have in not a few cases been the cause of death either from suffocation or from the inflammation which follows. Except under such circumstances foreign bodies gain access to the air-tubes as it were by surprise, and are sucked in by a sudden inhalation or gasp while the glottis is widely dilated for respiration, and therefore off its guard. It is most frequently with the sudden inhalation that precedes a laugh or cough, or with a deep inspiration on running or violent effort, that the accident occurs. Children furnish most of the cases owing to the inveterate habit they have of putting things into their mouth, sometimes even going to bed with them in the mouth and sucking them into the air-tubes during sleep.

Besides the common articles of food, there have been found coins, fruit-stones, pins, needles, nutshells, pieces of bone, wood and coal, peas, shells, beads, marbles, ears of corn, grasses, small toys, and even living leeches, etc. During vomiting also, the contents of the stomach may pass into the tubes as well as things which must have come originally from the intestine, such as lumbrici or even thread-worms.

Again, cases are described in which, during operations on the parts about the throat, teeth which have been extracted, pieces of tonsil, uvula or polypus which have been excised, or again some of the instruments used, as for example a laryngeal brush, the stem of a tracheotomy cannula, the needle of a syringe, etc., etc., have fallen into the tubes.

In another group of cases the foreign body gains access to the tubes in some other way, by external violence, as a bullet, pin or dart; from a tracheotomy wound, *e.g.*, a piece of dressing, a feather, etc., or by the ulceration into them of a caseous or calcareous gland. Portions of a necrosed cartilage, or of a body impacted in the œsophagus, like the vertebra of a fish, may be found, or, lastly, hydatids from the lung or pleura may be present.

In one remarkable case, a man coughed up a piece of one of his own ribs about three inches long; this is of historical interest as having been recorded by Pibray, the first surgeon of Henry the Fourth.

Finally, ulceration or malignant disease may open a fistulous communication with the œsophagus, and thus admit food of all kinds.

Some substances undergo change after impaction: for example, particles of food decompose and become septic, setting up violent inflammation in the parts about them: others, again, like seeds, imbibe moisture and swell and thus become

very firmly impacted; they have been known even to germinate; thus a bean has been found swollen to twice its natural size, and a maize seed has commenced to sprout after being lodged for three days.

It is unnecessary, having indicated the chief sources whence the foreign bodies may be derived, to attempt to give a complete list of all that have been described. Miscellaneous as they are, they admit, however, of being arranged in one or two groups which are of clinical importance, viz., according to their size, *i.e.*, whether large or small, and according to their shape, *i.e.*, whether jagged or rounded. A large body will stick in a large tube and lead to great obstruction, while a small one may pass on into a small tube and possibly give rise to no symptoms at all. On the other hand, a jagged body may be entangled in the larynx and will in all probability stick there, and then, besides the mechanical obstruction it offers, it will give rise to violent irritation, evoking spasm and leading to inflammation.

Foreign bodies having gained access to the air-tubes may stick in the glottis or in the larynx, or may pass on into the trachea or bronchi.

Of the two bronchi they lie more frequently in the right. This is remarkable, considering that the right bronchus comes off from the trachea almost at a right angle, while the left is much more oblique and lies more directly in the course of the trachea. The explanation is to be found in the greater size of the right bronchus, and in the position of the inter-bronchial septum. This septum extends from the bifurcation of the trachea some little distance upwards, much more to the left than to the right, and thus protects the mouth of the left bronchus, so that a body falling to the end of the trachea is directed by the septum away from the left bronchus and into the right.

Various statistics are published to show the relative frequency of foreign bodies in different parts of the air-passages. (Thus Bourdillat¹ found that out of 156 cases, 80 were lodged in the trachea, 35 in the larynx, 26 in the right bronchus, and 15 in the left bronchus. Durham,² however, regards the larynx and the right bronchus as the commonest seats.) Statistics, however, are of little more than general interest, and are hardly of practical use in any given case, for so much depends upon the size, weight, and smoothness of the foreign body as to the part of the air-tubes where it is likely to lodge.

SYMPTOMS.—The symptoms are in most cases conclusive, viz., a sudden attack of dyspnoea preceded by the disappearance of the foreign body from the mouth, and the sensation of its passage into the windpipe.

The causes of the dyspnoea are two: (1) Mechanical obstruction to the passage of air, and (2) laryngeal spasm set up by the irritation. The former is of course persistent and the latter transient.

The dyspnoea due to mechanical obstruction varies with the size and character of the body and with the place of its impaction; thus a large body lodging in the



Fig. 16.

Trachea and bronchi opened from behind to show the different size of the two bronchi and the ridge extending upwards from the bifurcation towards the left side.

¹ *Gaz. med.*, 1861 and 1868.

² *Holmes' System.* Art. on subject.

glottis, larynx, or trachea may produce almost immediate death from suffocation, while a small body impacted in a large bronchus may lead to little dyspnœa, and, in a small bronchus, perhaps to none at all.

The dyspnœa is of the usual inspiratory type, and, if extreme, may be attended with very marked inspiratory recession of the soft parts of the thorax, or of the lower ribs in young children.

Laryngeal spasm is a very important factor in the production of the initial dyspnœa in most cases. Severe spasm may be excited by the passage of even a very small body through the larynx, but it is more likely to arise if the body become impacted there. In many cases the spasm quickly passes off, and with it, of course, so much of the dyspnœa as was due to it. Where the body is small and passes quickly through the larynx, spasm may be completely absent, and whatever dyspnœa arises is then due to mechanical obstruction alone. In cases where the body is impacted in the larynx, and yet is too small to offer much, if any, obstruction, the initial spasm may pass off quickly and recovery appear to be complete, yet spasm may recur, perhaps, many times, and sometimes take on a recurrent, almost asthmatic, type. In other cases there is no further spasm, but after a few hours the dyspnœa slowly returns and gradually increases until it becomes extreme. This is the result of acute laryngitis or inflammatory œdema set up by the irritation. With the dyspnœa is associated more or less cyanosis according to the amount of obstruction.

Cough is generally more or less severe, and may be so violent and paroxysmal as to be the most distressing symptom, and where there is also laryngitis it may assume a croupy character and thus add to the difficulty of diagnosis. The cough is strictly conservative, and directed to expel the foreign body, and as soon as this has been effected the symptoms usually subside as rapidly as they came on.

The voice is not affected unless there be laryngitis, or the foreign body be so placed as to interfere with the movements of the vocal cords.

There is at first no expectoration, unless there be a little streaky hæmoptysis from the violent coughing. In the subsequent stages, the sputum has the characters of the resulting inflammations; thus it may be bronchitic, purulent, or even fetid, and if a vessel become involved, there may be profuse and even fatal hæmoptysis.

Pain is usually absent, unless the soreness beneath the sternum or the aching in the muscles due to the cough be included under that term. Sometimes, however, there are sensations which enable the patient to refer the obstruction to its proper place; but, on the other hand, the sensations often continue, especially in the larynx and in nervous persons, long after the foreign body has been got rid of.

The symptoms vary very greatly in intensity according to the seat of the impaction. They are most severe when the impaction is in the larynx on account of the violent spasm and coughing excited, and because of the great and rapid swelling to which the larynx is liable.

The physical signs, like the symptoms, vary according to the seat and amount of obstruction. If there be much obstruction in the larynx, there will be the usual marked respiratory excursion of the larynx, with more or less laryngeal stridor, and perhaps some affection of the voice. If there be stridor, whether due to an obstruction in the larynx or in the trachea, it will so modify the physical signs in the chest as to make it very difficult to draw any conclusion from auscultation as to the condition of the lungs.

If the foreign body stick in the bronchus and completely occlude it, the voice and breath sounds will be abolished over the corresponding parts of the lungs,

but if the occlusion be not complete a kind of stridor may be heard which may resemble rhonchus, save that it does not vary so much from time to time as rhonchus does.

If a large bronchus be plugged and the lung collapse, the parts of the thorax corresponding may be flattened, and measure less than on the opposite side.

The foreign body may change its place, either as the result of coughing or of some change of posture from the larynx to the bronchus, and *vice versa*, or from one bronchus to another. When freely movable it may be heard or even felt rattling in the trachea, and is thus frequently coughed up into the larynx, where, if it be not at once expelled, it excites the same kind of paroxysmal dyspnoea as at first. It is this which constitutes one of the chief risks as long as the foreign body continues in the air-tubes.

Just as the change in position of the foreign body may produce fresh dyspnoea, so it may relieve that already existing. Dyspnoea, due to a foreign body, suddenly relieved may mean that the body has been expelled, that the spasm has passed off, or that the body has passed lower down into one of the smaller bronchi. Which of these causes the relief is due to, can only be settled by careful examination.

The general condition of the patient, beyond that which is the result of the dyspnoea, will depend greatly upon the complications to which the foreign body has led.

PATHOLOGICAL CONSEQUENCES.—As a rule, all fear of after effects is removed when the foreign body is got rid of early, but if it remain impacted for long, pathological changes are almost certain to follow, either locally at the seat of impaction in the tubes themselves, or distally in the lungs beyond.

The local changes vary somewhat according to the seat of impaction. In the larynx, as a rule, inflammation rapidly supervenes, and may lead to so much swelling as to produce suffocation, unless relieved by tracheotomy.

A ring may lie for a long time in the larynx or trachea without producing symptoms or local changes of importance, and be removed after many years' impaction with complete recovery. On the other hand, it may excite chronic inflammatory induration; the stenosis thus produced may after removal sometimes slowly subside and disappear, but more frequently persists with all its disastrous consequences. In the ventricles of the larynx, a foreign body may be impacted for many years without producing symptoms or any grave local lesion.

This happened with a piece of gold in a case recorded by Watson in his *Clinical Medicine*. On the other hand, Dessault records a case in which a cherry stone in that position caused death from chronic laryngeal disease two years after impaction.¹

In the trachea, an angular body might lead to ulceration of the walls or to perforation, with the formation of an abscess around, and the entrance of air into the tissues of the neck, or of a communication with the œsophagus, or even with one of the large blood-vessels. Thus Rokitansky records a case in which the innominate artery was penetrated by a dart lodged in the trachea.

In a bronchus, it is true, a foreign body may lie for some time without producing change at all, but more commonly it excites local bronchitis with a mucopurulent and sometimes a fetid discharge.

Andrew² records the case of a child, in which a piece of nutshell had lodged in the right bronchus. This produced a profuse thin watery mucopurulent discharge, which continued for two or three days, and ceased immediately on the expulsion of the foreign body.

¹ *Œuvres char.*, ii. 258.

² Cavendish Lecture, *Lancet*, 1899, p. 1177.

If ulceration occur, the inflammation will probably extend beyond the bronchus to the adjacent parts of the lungs, and the resulting pneumonia is not unlikely to suppurate or gangrene. The inflammation may spread to the mediastinum and form an abscess there, or if the bronchus be perforated, may give rise to mediastinal emphysema, which, spreading along the cervical fascia to the subcutaneous tissue of the neck on the one hand, may then extend widely over the body, or, on the other hand, may communicate with the pleura and produce pneumothorax.

If the impacted body be of a non-irritating nature, the changes are of a much more chronic character, and attended with an abundant production of connective tissue, by which it may become almost completely encapsulated. These local lesions may subsequently become the seat of tubercular infection, which may extend to the lung and lead to phthisis.

The distal changes, *i.e.*, the secondary changes at a distance from the obstruction and not in immediate connection with it, are best studied in those cases in which the foreign body is lodged in a small bronchus. If the obstruction be complete, the corresponding parts of the lung collapse, and may subsequently pass into a state of more or less acute pneumonia, which, if the foreign body be septic, as in the case of particles of food, will probably suppurate or even gangrene. In other cases the inflammation is of a more chronic character and attended with a good deal of interstitial change, which may even lead ultimately to the complete obliteration of the alveoli, and the conversion of the obstructed parts into an almost solid fibrous mass, in which the bronchi may be more or less dilated. More often irregular cavities develop in it, which may have originated in bronchiectasis, but which extend by ulceration and inflammation round them until the case closely resembles chronic phthisis, in gross anatomy as well as in clinical features, save that the lesions are not tubercular. With such indurative changes, the corresponding part of the chest will be contracted, and may measure much less than the opposite side.

Advanced changes, such as these, are hardly possible except where a portion only of one lung is affected, although there are a few cases in which the obstruction has been in the main bronchus, and the whole of the corresponding lung has been destroyed.

When suppuration takes place round the impacted body, it may escape from the place of its impaction and come to lie free within a cavity in the lung or even in the pleura, as in the remarkable case recorded by Carpenter,¹ where, in a fatal case of empyema, there were found lying loose in the pleural cavity some false teeth which the patient had swallowed thirteen years before.

Sometimes pyæmic symptoms arise and kill the patient, as in a case in which the cause was discovered to be a bean which had been long impacted in a bronchus, and the presence of which was not suspected.²

Some bodies make their way out of the air-tubes by simpler processes than those described. Needles, blades of grass, ears of corn, or burrs have thus travelled, until they reached the subcutaneous tissue, from which they have been removed by simple incision.

COURSE AND COMPLICATIONS.—A foreign body may become impacted and produce immediate suffocation; this is fortunately rare: it may be immediately expelled; this is happily the rule: or after remaining a longer or shorter time in the air-tubes it may be spontaneously expelled or be removed by operation, with or without previous tracheotomy. If it remain impacted, it will

¹ *Guy's Hosp. Rep.*, 1st Ser., 353.

² *B. M. J.*, 1880, 519.

set up local or distal changes in the bronchi or lungs, of which the patient not infrequently dies in the end, and that sometimes even when the foreign body has at last been expelled or removed, by reason of the mischief previously caused. Lastly, but also most rarely, it may remain impacted for years, and produce no lesions or symptoms of consequence.

Thus, a piece of bone has been found in the lung six years after its impaction, and similarly a nail, without any lesion or anything in the history to fix the date of entrance. Again, a nail has been known to lie in a bronchus for twelve months before any symptoms developed.

Further, it is possible for the body to lie for long periods, even many years, in the tubes and yet be spontaneously expelled with perfect recovery.

A piece of coal¹ has been coughed up after 101 days, and a tin whistle² after 104 days. Watson records a case in which after repeated attacks of profuse hæmoptysis, spread over a period of seven years, a lady at last expectorated an ear of barley and made a perfect recovery. Pieces of bone have been expelled after six years' and after fourteen years' impaction, but the most remarkable case of this kind is recorded by Gross, in which a piece of bone was coughed up after being lodged in the tubes for sixty years.

Lastly, the body has been successfully removed by operation after long periods.

Lefferts³ removed a ring from the larynx of a child of six and a half years of age which had been impacted there for four years, *i.e.*, from the age of two and a half years. A piece of bone which had been in the trachea for nine years was successfully removed by Mr Watson Cheyne.⁴ In giving an account of the case Dr. Bunch and Mr. Lake gave a table of all the recorded cases up to date, 31 in number, in which recovery followed, after operation or spontaneous expulsion, where the body had been retained for longer than one year.

PROGNOSIS.—Wonderful as many of these cases are to relate, still they form the curiosities of the subject and are only notable exceptions to the general rule. The risks are great as long as the body remains impacted, first because of the danger of sudden suffocative spasm developing as the result of the body being coughed up into the larynx, if it be not already lodged there; and secondly, because of the almost certain prospect of changes occurring round the impacted body, which will lead to serious disease, if not early death.

DIAGNOSIS.—The diagnosis is in most cases easy, but, where the body sets up spasm of the larynx, the distinction in a young child from laryngismus stridulus may present difficulty, or if the spasm recurs, from whooping-cough or even asthma. Where the body is impacted in the larynx, and has excited inflammation, the resemblance to croup or other forms of laryngitis is very close.

If the initial spasm be absent, the diagnosis is extremely difficult and has to be made by the physical signs and by the history, which in children however is never trustworthy, so that it is a good rule in practice always to bear in mind the possibility of a foreign body in any child who presents unusual chest symptoms and signs.

Where the foreign body is small and septic, the case often presents itself as pneumonia or gangrene of the lung, or even of general pyæmia, and the cause may then only be discovered *post-mortem* by the demonstration of the foreign body.

TREATMENT.—The object of treatment is obvious, *viz.*, to get rid of the foreign body as soon as possible. Fortunately nature does this for us in most cases and the body is expelled by coughing. Attempts have been made to assist nature by exciting sneezing or vomiting, or by inverting the patient and shaking

¹ *Med. T.*, 1884, p. 501.

² *Lond. M. T.*, viii.

³ *Med. Rec.*, 1874, p. 671.

⁴ J. Rickman Godlee, *Lancet*, Sept. 25, 1897; *Med. Chir. Trans.*, 1896.

or slapping the back. Though each of these methods has been successful, they are all more or less risky owing to the danger of suffocation if the body be coughed up into the larynx and be not at once expelled. To avoid this risk tracheotomy is indicated, and it should, as a general rule, be performed with as little delay as possible. Durham says: "If the surgeon hesitate to operate under favourable circumstances, he may be compelled to do so in the greatest haste and under the greatest difficulties." The object of the operation is to facilitate the expulsion of the foreign body, and its effect is that it is either immediately or after a short interval coughed up through the wound. Even if it be not at once expelled, tracheotomy enables it to be searched for at leisure, and removed, if it be within reach, by appropriate instruments. Removal through the mouth, if the larynx be the seat of impaction, is rarely practicable until tracheotomy has been performed, on account of the spasm excited; still a few successful cases have been recorded.¹

The results of cases treated with and without tracheotomy are compared by Mr Durham.

Of 298 cases in which no operation was performed, death occurred in 40·94 per cent., and every case in which the foreign body was not expelled (102) died of the effects sooner or later, while of 338 cases in which tracheotomy or some similar operation was resorted to, death occurred in only 23·08 per cent.

Of 1000 cases collected by Weist, operation was necessary in just one-third (338 cases): of these 245 recovered and 93 died (38 per cent.).²

The combined statistics of Durham, Gross, Weist, and Roe give the following result:—

With operation, . . . 1234 cases with 964 recoveries = 78 per cent.

Without operation, . . . 1417 cases with 1035 recoveries = 73 per cent.³

Although, where the body is small and freely movable, the operation may be sometimes avoided, still there is much room for judgment and skill in deciding in what cases to wait and how long, and even then the patient should be kept under the closest observation, with everything ready for tracheotomy the moment it may become necessary.

Where the foreign body is firmly impacted in a bronchus, considering the serious and almost invariably fatal consequences, all reasonable attempts at its removal by surgical means are justifiable, but the difficulties of operation are considerable.

Impaction of a Fishbone in the Larynx.—Initial Spasm—Dyspnœa absent for four Days, then Severe, necessitating Tracheotomy—Subsequent Removal of the Bone through the Wound—Difficulty in Removing the Tube owing to Paralysis of the Abductors, until they were forcibly dilated and thus set into Action.

An infant nine months old was seized suddenly with severe dyspnœa, due it was thought to swallowing a plum stone. In a very short time the spasm passed off, and there was no manifest dyspnœa for four days, when it returned, and rapidly became so urgent that tracheotomy was performed. This gave relief, and all went well for three days, when fresh dyspnœa set in. When the tube was removed a piece of the dressing was coughed up from the trachea, and on examining with a probe a foreign body was felt in the larynx which was removed with forceps, and proved to be the gill-plate of a small fish, probably a herring.

After this all went well, but the child could not do without the tube, for it did not breathe through the mouth, though it could cry audibly. After this had gone on for some time without improvement the child was anaesthetised and a graduated bougie and then a dilator passed through the larynx both from the mouth and from below. This had the effect for a time of permitting the child to breathe through the mouth, but the tube had to be replaced soon after. The next day, however, on the tube being taken out, air passed freely by the mouth, the tube was then and there finally given up, and in a fortnight the wound was healed.

¹ M. Mackenzie, *Path. Soc. Tr.*, xviii. 27.

² *Trans. Amer. Surg. Ass.*, 1881-3, i. p. 117.

³ Cf. also Preobraschensky, *Wien Klinik*, 1893, No. 8-10. Roe, in *Burnet's Dis. of Ear, Nose, and Throat*.

The case illustrates several points of interest.

1. The initial spasm produced by the impaction of the bone, which quickly passed off and was followed by a period of latency, in which the absence of symptoms led to the belief that the foreign body had been expelled.

2. The sudden development of persistent dyspnoea due to the inflammatory swelling set up by the impacted bone.

3. The closure of the glottis which prevented the removal of the tube and was due to paralysis of the abductors from disuse.

4. The speedy cure of this condition by mechanical dilatation.

A case in which the removal, from the front, of a foreign body in the right bronchus was successfully achieved is recorded by Milton. The patient died subsequently from septic infection, the result of defective drainage. It is probable that with improved methods the results may be more satisfactory.¹

LARYNGITIS.

Laryngitis is chiefly of importance in relation to respiratory diseases so far as it produces impediments to the entrance of air into the lungs. In a slight form it may do no more than impair the voice and cause cough, but in the more severe forms it may be attended with so much swelling as to lead to considerable narrowing of the air-passage and even place life itself in danger from suffocation. It is also often symptomatic and may then yield the first evidence of a grave lesion such as tubercle, syphilis, or tumour. For these reasons laryngitis assumes an importance in diseases of the respiratory organs which it would not otherwise have.

There are two great forms of laryngitis which differ according as the inflammation is attended with the formation of a membrane upon the surface or not, viz.—

1. Catarrhal or simple laryngitis.
2. Membranous or croupous laryngitis.

Besides these there are specific affections of the larynx, which are often spoken of as special forms of laryngitis, of which tubercle and syphilis are the most important.

7. CATARRHAL LARYNGITIS.

The **pathology** of catarrhal laryngitis is simple, and the changes are essentially the same as those seen in inflammation of the trachea and bronchi, viz., congestion of vessels, swelling of the mucous and sub-mucous coat, due to infiltration with serum and small cells, shedding of epithelium, and formation of mucopurulent secretion.

The predominance of one or the other of these changes will depend upon the part of the larynx attacked, for the inflammation is very often limited in extent. Thus, if the vocal cords alone be affected, there will be much congestion but little swelling, and phonation will be chiefly interfered with. This congestion, visible during life with the laryngoscope, disappears after death.

If the softer and looser parts are affected, such as the false vocal cords and the aryteno-epiglottidean folds, the swelling will be the chief feature and may be sufficient to lead to considerable dyspnoea.

The **chief symptoms** are, like the morbid anatomy, simple: (1) huskiness or hoarseness of the voice, and sometimes even complete aphonia; (2) cough; (3) expectoration; and (4) sometimes dyspnoea.

The *affection of the voice* depends either upon the defective or irregular vibration of the true cords, owing to the loss of their elasticity consequent on the congestion and swelling, or to the cords not approximating as they should on phona-

¹ *Lancet*, Jan. 6, 1901.

tion. This latter may be due, on the one hand, to inflammatory changes in the cords themselves or in the muscles or nerves which govern their movements, or, on the other hand, to mechanical causes, such as swelling over the arytenoid cartilages or in the inter-arytenoid space, or mucous and inflammatory secretion lying upon them.

Aphonia, *i.e.*, complete loss of voice, may occur, but, though not uncommon with simple catarrh in women, it is certainly rare in men.

The cough is usually dry, frequent, irritating, and may recur every few seconds both night and day. This is especially the case in children during the first days of measles. In children it is often spasmodic, and may even resemble whooping-cough. It is sometimes deep, bellowing, or barking, a peculiarity which Ziemmsen refers to coarse vibrations set up in the swollen aryteno-epiglottidean folds or false cords.

The *expectoration* is scanty, viscid, mucopurulent and difficult to get rid of, but it varies greatly in amount with the part of the larynx affected, and, as a bad catarrh is rarely limited to the larynx, much of it may come from the pharynx or the trachea. It is occasionally streaked with blood. The spitting of pure blood, even in small amount, is very rare, but there seems to be no doubt that hæmoptysis may occur with laryngitis, for streaks of blood may sometimes be seen upon the vocal cords, and occasionally small varicose vessels are present which may rupture during coughing.

Dyspnœa is rare in adults, but in children it is very common, and may occur in severe paroxysms, especially during the night-time.

Forms and Ætiology.—Catarrhal laryngitis may be acute or chronic, primary or secondary.

Of primary laryngitis the commonest causes are a cold or the direct action of irritating vapours, as, for instance, scalding steam, chlorine, bromine, ammonia, etc.

The secondary form is due to the spreading of a catarrh, as in influenza or hay fever, or to the extension of inflammation from the neighbourhood, especially from the pharynx. It occurs also in the course of many specific fevers, especially of measles and influenza. It is often symptomatic, *i.e.*, it is the result of some other laryngeal affection, *e.g.*, tubercle, syphilis, or cancer.

Among the chief and common causes which predispose to simple catarrhal laryngitis, or maintain it, are prolonged and excessive use of the voice, cold, drinking (especially of spirits), and smoking.

One attack predisposes to another, especially if recovery has not been complete, and the patient continues exposed to the conditions under which the first attack originated, as in the case of clergymen, vocalists, and public speakers.

What is called chronic laryngitis, very often consists of a succession of acute attacks with incomplete recovery between them.

As acute laryngitis is likely to produce much more grave symptoms in the child than in the adult, it will be well to consider these two classes of cases separately.

LARYNGITIS IN THE ADULT.—*a. Acute.*—In the adult, catarrhal laryngitis is usually a very trifling affection for which patients often think it unnecessary to take medical advice, unless their occupation is such as to make special demands upon their voice.

The inflammation is often limited to the true vocal cords or parts of them, especially the posterior ends. The affected portions of the cords are reddened, have lost their polish, and are somewhat thickened.

The *symptoms* are then slight, the voice is husky, there is a dry cough and tickling in the throat, made worse by using the voice. After a few days, usually less than a week, the inflammation subsides and the symptoms disappear.

In a severer form, the inflammation is more extensive and involves the soft parts, which may be much swollen.

The true vocal cords are more congested and more swollen, and frequently do not approximate as they should on phonation.

Upon their surface may be seen grayish patches due to epithelial degeneration; sometimes small ecchymoses or superficial excoriations, the so-called catarrhal ulcers. These occur on the vocal processes and the inter-arytenoid folds.

The hoarseness, irritation, and cough are more marked; the secretion is viscid and difficult to bring up; a feeling of dryness, soreness, or of burning in the throat is complained of, and pressure over the larynx causes cough or pain. Actual dyspnoea is rare, though in the most acute forms the patients suffer much discomfort and feel as if they might choke.

In the most severe form of all, the case becomes one of œdema of the glottis, which will be specially considered later.

The constitutional symptoms, in the adult, are slight except in severe cases; a little rise of temperature to 100° or 101° at most, with corresponding acceleration of pulse and respiration. In the child, the general symptoms are more pronounced, and, as in other catarrhal ailments, the temperature may reach 103° or 104° for a day or two.

The *prognosis* of catarrhal laryngitis is almost always good.

The *duration* of the more severe cases is from eight to fourteen days, but of the slight cases rarely more than a week; the voice, however, may remain weak, impure, and unequal to the usual work for some time, especially in the cold and raw months of the year.

The *diagnosis* in the adult of the affection is usually simple enough, though it may not be so easy to determine whether the affection is primary or secondary, *i.e.*, symptomatic.

Laryngeal catarrh of unusual duration or its frequent recurrence in an adult whose voice is not subject to special strain should always raise the suspicion of some underlying disease, of which tubercle or syphilis is the most frequent, and new growth is an occasional, cause.

b. Chronic.—Chronic laryngitis in the adult includes two conditions: (1) sub-acute catarrhal laryngitis which has lasted long or often recurred; (2) the condition of thickening and induration to which previous attacks have led.

Chronic catarrhal laryngitis is most frequent in males in middle life and among clergymen, public speakers, singers, hawkers, etc., who use their voices either too much or in a faulty way. It is very commonly associated with chronic pharyngitis, and, like that affection, is often provoked or maintained by excessive smoking or drinking.

It is a common result of mouth breathing, where respiration through the nose is impeded.

The *symptoms* do not differ from those of acute laryngitis, except that they are not as a rule so severe. The voice is hoarse, and may even be lost and easily tired, so that speech requires an effort. Cough need not be troublesome.

Expectoration is often absent, or very scanty (*Laryngitis sicca*). In some rare cases it may be profuse, and has then been described as *laryngorrhœa*; but profuse secretion of this kind is more commonly due to coincident catarrh of the trachea or pharynx.

The *laryngoscopic appearances* vary greatly, from a patch of redness on a part of one or both cords, up to an affection of the whole larynx with much swelling and stenosis.

In long-standing cases, a general thickening of the superficial parts of the larynx may be produced, which was named by Virchow *pachydermia laryngis*.

Small *warty* or *polypoid growths* may also be found in various parts of the larynx, especially in the epiglottis, aryteno-epiglottidean folds, inter-arytenoid space and false cords. They may produce dyspnoea and require removal.

Upon the vocal cords in patients who have to use their voice excessively, small nodes are often found (*singers' nodes*, *chorditis*), symmetrically placed at the junction of the anterior and middle thirds. They are of small size, not larger than a millet seed, interfere with the proper approximation of the cords and cause roughness or hardness of the voice.

In other cases the swelling and induration may be more marked above the vocal cords or below them (*subglottic laryngitis*) and may cause stenosis.

Ziemssen describes a case in which so much swelling occurred that tracheotomy became necessary, after which the thickening disappeared. In most cases of this kind the swelling depends upon syphilitic, tubercular or malignant disease, and in the diagnosis of these conditions age is of importance. Thus in early adult life tubercle is most likely, at a somewhat later period syphilis, and in the middle and later period malignant disease.

Treatment of Catarrhal Laryngitis in the Adult.—In mild cases, the patients will hardly be induced to stay at home. If they get out they should wear a respirator or breathe through a comforter, should avoid talking, especially in the open air, and eschew smoking. At bedtime, some warm demulcent beverage and a hot foot-bath should be given and a flannel wound round the neck, and, if the weather be cold and raw, a fire lighted in the bedroom.

In the more acute and severe cases the patient must be kept indoors altogether and in a warm room if not in bed.

The food should be liquid and taken as hot as it can be swallowed with comfort; hot milk with soda or seltzer water makes a palatable drink.

A hot bath or foot-bath should be given. A diaphoretic mixture, to which may be added 15 to 30 minims of antimonial wine or 30 to 60 minims of ipecacuanha wine, should be administered every three or four hours. A few grains of calomel should be given, followed by a saline purge the next morning, to unload the bowels. Great benefit is also derived in the early stage from small doses of tincture of aconite taken at short intervals.

As external applications to the throat, cold, wet compresses continually applied often give relief, but hot applications in the shape of poultices or fomentations are usually preferred, and in severe cases a couple of leeches on each side of the larynx will do good.

Internally, relief may be given by the sucking of ice, by the use of inhalations either of steam alone or with the addition of Tinct. Benzoini Co. (3 i. to the pint) or in the form of hop and conium vapour or chloride of ammonium fumes.

If the cough be troublesome a lozenge of morphia and ipecacuanha or of cocaine and rhatany should be given; in a severe case, a few minims of laudanum placed on the back of the tongue or a dose of Pulv. Ipecac. Co. at night-time may be required to control the cough.

In chronic cases or cases of delayed or incomplete recovery, anything but the gentlest use of the voice must be prohibited, the general health in every way attended to, a tonic such as strychnia and iron administered internally and the patient sent away for change of air.

Chief reliance, in chronic cases, is to be placed on the direct application of astringent solutions to the larynx by the laryngeal brush, such as chloride of zinc (20 or 30 grs. to the oz.) and nitrate of silver (10 to 20 grs. or more to the oz.). Astringent sprays of alum or tannin are also useful.

If the voice be left weak, electrical treatment will assist recovery.

ACUTE LARYNGITIS IN THE CHILD — CATARRHAL CROUP — CATARRHAL SPASM — SPASMODIC LARYNGITIS — SPASMODIC CROUP.

Acute laryngitis in the child assumes an importance which it has not in the adult, because of the dyspnœa with which it is so liable to be associated, and of the difficulties in diagnosis which may then attend it.

The general symptoms also are usually more pronounced; there is higher fever, a more rapid pulse and more serious illness.

The dyspnœa is often considerable and is liable to paroxysmal exacerbations sufficient to cause serious alarm.

The frequency with which these paroxysms occur in the catarrhal laryngitis of children is referred, by some writers, to muscular spasm, "catarrhal spasm" as it has been called; but, without questioning the part which spasm may play in laryngeal affections in the child, it must not be forgotten that these paroxysms are often of mechanical origin. The glottis in children is of small size and the parts about it much less rigid and resistant than in the adult, and, as the result, a small amount of inflammatory swelling would more easily cause narrowing of the orifice, and this might readily become still further obstructed by the accumulation and drying of viscid secretion round about it, especially during sleep, for that is the time when the paroxysms most frequently occur.

The dyspnœa, during the paroxysm at any rate, if not at other times, is associated with the peculiar, noisy, rasping, stridorous or stridulous sounds which have been long called "croupous" or "croupy."

By the term **Croupous**, the breathing has been described in three different conditions which have to be distinguished from one another, viz., *Laryngismus Stridulus*, *Membranous Laryngitis*, and *Catarrhal Laryngitis*.

From *Laryngismus Stridulus*, the diagnosis of catarrhal laryngitis presents no difficulties. The paroxysms, though severe, are short, occur abruptly, often without warning, and especially at night, are preceded by no local catarrhal signs in the larynx, and frequently recur night after night. In the intervals the children are in their usual health. The clinical affinities of *laryngismus stridulus* are with convulsions and not with laryngeal affections at all.

It is from *Membranous Laryngitis* chiefly that the diagnosis is so difficult and important. It is important because in catarrhal laryngitis tracheotomy is not so likely to become necessary, and the prognosis of the operation when performed is so much more favourable. It is difficult because the actual symptoms may be the same, and the diagnosis must then rest upon the discovery of membrane elsewhere, or upon the history of the case. If membrane be coughed up the diagnosis is plain.

If membrane be present on the fauces, the presumption is strong that the laryngeal symptoms are also due to membranous inflammation, but the absence of membrane on the fauces is not conclusive, for in some cases the membrane is confined to the larynx and contiguous part of the trachea, and in others the membrane may be present in the pharynx but not visible from the mouth.

The history of a bad cold is frequently of some assistance, but on the other hand a chill often seems to determine the actual attack of membranous laryngitis, and catarrhal symptoms, such as a discharge from the nose, are common enough with diphtheria.

The history of exposure to the infection would, in the case of diphtheria, raise a strong presumption in favour of membranous laryngitis: but, in the case of the other specific fevers, on the whole the presumption would be against it; for in

most of the other forms, and notably in the case of measles and influenza, the catarrhal form of laryngitis is common, but, except in scarlet fever, the membranous form is rare. In all cases bacteriological examination should be made, and, if doubtful, this should be confirmed by the inoculation test.

The presence of a foreign body in the larynx may also raise difficulties, especially where history fails, as in the case of very young children. In all cases of acute inexplicable laryngeal dyspnoea in the young, the possibility of the impaction of a foreign body in the larynx should always be present to the mind. Treatment, however, will not be materially affected, for in all such cases tracheotomy will almost certainly become necessary.

Treatment of Catarrhal Laryngitis in the Child.—Catarrhal laryngitis in the child should be always regarded seriously, and even a mild case treated with as much care as would be given to a serious case in an adult.

The child should be confined to the house, and, if in bed, should be placed in a half-tent, and the steam kettle set to work, with the addition of some aromatic and soothing drug, such as the compound tincture of benzoin.

The throat should be wrapped in cotton wool with a little turpentine or spirits of camphor sprinkled upon it to keep up gentle counter-irritation. Sometimes cold compresses wrung out of iced water give relief, but usually hot fomentations and applications are preferred.

The bowels should be relieved with a small dose of Hydr. c Cretâ, and a diaphoretic mixture given containing a little ipecacuanha wine. Antimony and aconite are not so suitable for young children as they are for adults on account of their depressing effects. If there is much fever and the skin seem dry, a hot mustard bath will give great relief. If the cough be troublesome, a few drops of paregoric should be given.

All this is more or less the treatment of an ordinary catarrh.

If the dyspnoea become marked and especially if it be paroxysmal in character, hot applications, such as a hot sponge or hot fomentations to the throat, should be applied, and an emetic is often useful.

If the child be strong and the fever high, to the diaphoretic mixture some antimony or ipecacuanha wine should be added, or an emetic dose of these drugs given. If, however, it is desirable to make the patient vomit, it will be almost better to give a dose of apomorphia (gr. $\frac{1}{20}$ — $\frac{1}{30}$ *sub cutem*), the effect of which is more certain and more transient, and the after effects less depressing.

Where the paroxysms frequently recur, as they do at night, good may be done by keeping the air in the half-tent moist with steam, for the paroxysms are often due to a collection of viscid mucus in the glottis rather than to actual spasm.

If the dyspnoea become continuous, or during a paroxysm become intense, it may be necessary to perform either intubation or tracheotomy. Tracheotomy does not often become necessary for catarrhal laryngitis, but if performed it has a more favourable prospect than with membranous laryngitis. Intubation is the operation which seems especially indicated for these cases of catarrhal laryngitis. As a matter of fact the diagnosis constitutes the chief difficulty, for even in very small children catarrhal laryngitis is but rarely fatal. And in most cases the simple treatment for an ordinary chest- and throat-cold suffices for cure.

LARYNGEAL AFFECTIONS IN THE COURSE OF SPECIFIC FEVERS.—Laryngitis, in some form, is very common indeed in the course of the specific fevers.

In many cases there is simply slight catarrhal laryngitis with the usual signs. At others are found superficial erosions, on the back of the epiglottis as well

as in the larynx itself, and these so-called catarrhal ulcers may extend into the deeper tissues and lead to true ulceration.

In other cases again patches of infiltration may develop in the submucous tissue, which may ultimately necrose and break down. The cartilages may thus become involved and perichondritis with all its disastrous results be the consequence.

Where there is already a septic inflammation of the pharynx, as, for instance, in scarlet fever, the inflammation may spread to the larynx and excite acute inflammatory œdema there and prove rapidly fatal.

The degree of laryngitis likely to be met with varies with the fever to a great extent; fortunately it does not in many cases go beyond the catarrhal stage.

1. In **Measles**, laryngitis is almost the rule. It gives rise to a dry, irritating cough often called "the measly cough," which recurs every few seconds and gives great distress. It is a simple catarrhal laryngitis; it appears with the rash and usually disappears with it. In little children it may be associated with a certain amount of swelling and dyspnoea, but rarely produces symptoms of any gravity.

When membranous laryngitis occurs in measles it is very fatal, and is of diphtheritic origin. Goodall¹ states that of fifteen cases in which it occurred fourteen died.

2. In **Scarlet Fever** the simple catarrhal laryngitis is not so common, but when laryngeal symptoms occur they are nearly always due to the extension of inflammation from the pharynx, are associated with very great inflammatory swelling, and are almost invariably fatal.

In a certain number of cases the inflammation is membranous, but not necessarily diphtheritic, for, in many, the membrane is proved bacteriologically to be due to other than diphtherial infection, a conclusion previously arrived at by many observers on clinical grounds.

Membranous inflammation of the fauces in scarlet fever is not common, and occurs only in from 1.5 per cent. (Goodall) to 3.3 per cent. (Sweeting).

In these cases the larynx is not nearly so likely to become involved after scarlet fever as in true diphtheria, the difference being, according to Goodall, between 3 per cent. in scarlet fever and 21 per cent. in diphtheria.

It is stated that membranous inflammation of the throat and larynx occurring concurrently with the scarlet fever is generally not of diphtheritic origin, but due to infection with pyogenic organisms, such as the streptococci, while that which develops after the fever, *i.e.*, during convalescence, proves almost invariably to be diphtheritic (Klein, Henoch, Heubner).

The prognosis, in all these cases alike, is very grave, for with few exceptions they prove fatal.

3. **Small-pox**.—In small-pox laryngitis is common, and in a number of these cases it is associated with what has been called "the laryngeal rash." It is met with generally at about the end of the first week. It is associated with a considerable amount of general inflammation and swelling, so that great dyspnoea may be produced and tracheotomy become necessary, but the results of operation are disastrous, for hardly any cases recover.

The inflammation is sometimes membranous and may be true diphtheria, but in the majority of cases it is due to some other organism rather than that of diphtheria.

In many of the slighter cases of laryngitis, small membrane-like patches, which are easily detached and consist, as described, of degenerate epithelium and white blood-cells and bacteria, are seen upon the larynx.

¹ *Lancet*, May 1894.

If life be prolonged perichondritis may develop and lead to death.

In a case described by Eve¹ the perichondritis developed during convalescence.

4. **In Erysipelas** laryngitis is fortunately very rare. It hardly ever occurs unless the tissues of the neck are involved. It leads to acute inflammatory œdema and is invariably fatal.

In the erysipelas ward of St. Bartholomew's Hospital during a period of seven years there were only two cases of serious laryngeal affections in the course of facial erysipelas, and then only when the erysipelas had spread to the neck. In both cases tracheotomy was performed, but the patients died. The number of cases of erysipelas which had been under treatment during that time could not have been less than 650, so that the percentage is very small.

5. **Typhoid Fever.**—In typhoid fever all the forms of laryngitis described are met with; first, the simple catarrhal form; secondly, the gray membrane-like patches with superficial ulceration; thirdly, actual ulcers; fourthly, perichondritis with necrosis of the cartilage; and fifthly, diphtheria.

It is in connection with typhoid especially that those small patches of diffuse or nodular infiltration have been seen which have been described as "specific"; these necrose and form ulcers with infiltrated margins, which have been compared with the ulcers met with in the intestines and have been attributed to the same bacillus. In some of these, the typhoid bacillus has been actually demonstrated, but in others the typhoid bacillus is absent, and some other form of pyogenic organism present, especially the streptococcus and staphylococcus, as is the case also in the inflammations of the trachea, bronchi, and lungs which occur in the course of typhoid.

The fact that these inflammations of the larynx are caused by streptococci and staphylococci, and possibly by other organisms so commonly present in the mouth, as well as by the specific bacillus of typhoid itself, seems to suggest that much might be done to prevent these complications by strict and careful cleansing of the mouth and fauces. In bad cases of fever the patients generally breathe through the mouth, which is not properly cleansed as it would be in health by the free secretion of saliva and the constant movements of the tongue. The mouth, therefore, is a good breeding-place for pathogenic organisms of all kinds and teems with them, so that it is not to be wondered at that they should spread and carry infection to the pharynx, larynx, and air-tubes. The risk of these complications could no doubt be greatly reduced by a careful mouth-toilette, the mouth and throat being kept clean and, as far as practicable, disinfected by mouth-washes or sprays.

For the parts which are within reach of the finger or a brush, nothing is better than the old-fashioned remedy, glycerine and borax.

8. MEMBRANOUS LARYNGITIS.

The term membranous laryngitis is to be preferred to any other. It clearly and distinctly represents the fact, viz., that the disease is a laryngitis with a membrane, and while it indicates the difficulty which is constantly recurring in practice, viz., that of diagnosis between those forms of laryngitis which have, and those which have not, a membrane, it does not necessarily raise at the outset the difficult questions connected with its pathogeny.

History.—Although it is highly probable that a disease corresponding with what we now call membranous laryngitis existed in remote ages, still there is no description in early writers

¹ *Path. Soc. Tr.*, 1880.

which would justify the belief that it was recognised by ancient physicians. The passages in Hippocrates, Aretæus, and Celsus which have been supposed to refer to this affection are not conclusive. It is only at the end of the sixteenth century that a clear description of the disease is forthcoming in the writings of Baillou in 1576. In the beginning of the seventeenth century it is accurately described as prevailing in Spain (*garotillo*), Italy (*mala in canna*), and subsequently in other parts of the world (*morbis strangulatorius*).

In 1765 Francis Home gave an account under the name of croup of what he considered to be a new affection in Scotland, and in 1801 this name was adopted by Cheyne in an essay entitled, "On Cynanche trachealis or Croup." Croup became from this time the commonly recognised term.

The death of the grand-daughter of the Empress Josephine in 1807 from croup, and the offer by the Emperor of a large prize for the best treatise on the subject, caused the disease to be closely studied and much written upon.

But under the one term, croup, three forms of affection were confounded—first, membranous laryngitis; secondly, non-membranous laryngitis; and lastly, laryngismus stridulus.

The distinction between these various forms was not recognised until Bretonneau began to write in 1821. He sharply marked off from the rest that form in which membrane was found in the larynx and trachea, and he invented the term diphtheria to denote it. He regarded the affection as a specific disease arising from infection, and established the fact that, in most cases, though not in all, there was a membrane also upon the fauces; but, whether this was so or not, the disease was in his opinion in all cases alike the same in nature.

Other authorities however did not accept the identity of the two affections, but distinguished croup, which they regarded as a primary laryngeal affection, from the membranous laryngitis which developed in the course of laryngeal diphtheria. The question thus raised as to the diphtheritic nature of croup was the subject of constant discussion, and can hardly be said to be yet settled.

In this country for many years the two affections were regarded as essentially distinct. Of recent years the tide of opinion has set strongly in the opposite direction, so that membranous laryngitis is often curtly described as laryngeal diphtheria.

At the present time, while it is admitted that membranous laryngitis is much more frequently diphtheritic than was formerly believed, it is held also that there is a residue of cases, how large is not known, in which the affection is due to some other cause.

The term "croup" has been the cause of so much confusion that it is rightly dropping out of use. When first employed it was used simply as a descriptive clinical term, and referred to the peculiar noisy breathing. A distinction was then drawn between the cases in which a membrane was present in the larynx, and those in which there was none, the former being called true or membranous croup, and the latter false, spurious, non-membranous croup or pseudo croup, while both affections had to be distinguished from the spasmodic affection known as spasmodic croup, which is now called laryngismus stridulus, the latter being not a local disease of the larynx at all, but allied to, if not actually a form of, convulsive spasm of the larynx.

The general confusion has been still further increased by the distinction made between "croupous" and "diphtheritic" as pathological terms applied to inflammatory lesions associated with the presence of a membrane; the membrane in the croupous form being superficial, easily removed, producing no ulceration, and healing without scarring; in the diphtheritic form involving the deeper tissues, not easily removed, and leaving an ulcer which on healing leaves a scar.

The terms "diphtheria" and "diphtheritic" are now restricted to the lesions, whatever their pathological nature, which are produced by the diphtheria bacillus; while "croup" and "croupous," together with the distinctions they implied, are being gradually eliminated from modern medicine. The terms "croupous" or "croupy," however, used in their original clinical sense to describe certain peculiarities of breathing without any implied suggestion as to the cause, fulfil a want and may possibly survive for some time longer.

In most cases membranous laryngitis is, no doubt, of diphtheritic origin, and in the description which follows, it is this form which will be dealt with. It may be called laryngeal diphtheria.

LARYNGEAL DIPHTHERIA.—Laryngeal diphtheria is an acute, communicable disease produced by a specific bacillus (Klebs-Löffler). It especially attacks the throat, where it produces a membrane in which the specific bacilli are found. Not all membranes on the throat are, however, necessarily diphtheritic, although the majority are.

In diphtheritic membranes many other bacteria are found, *e.g.*, streptococci and staphylococci, so that in many cases there is a mixed infection.

The symptoms are chiefly toxic, *i.e.*, due to poisons produced locally by the bacilli and absorbed into the blood.

The bacilli are not widely distributed in the body and are often confined to the part where the membrane is formed, but they may also be found in the deeper tissues, and occasionally even in the blood.

Diphtheria may, in slighter degrees of infection, produce a simple catarrhal inflammation, *i.e.*, inflammation without any obvious membrane; on the other hand, as stated, not all membranous inflammations are necessarily diphtheritic.

Laryngeal diphtheria presents certain peculiarities. In the first place the local symptoms, which in pharyngeal diphtheria are not very pronounced, become predominant in laryngeal diphtheria, because the membrane obstructs the glottis and interferes with the entrance of air into the lung. In the next place, the toxic symptoms are, as a rule, less marked, but this depends partly upon the obstruction in these cases being early fatal, so that there is less time for the absorption of the poisonous substances into the blood, and partly because the parts affected are not so adapted for rapid absorption as the parts about the pharynx. Thus, it is said that glandular enlargements are less frequent, that albuminuria and visceral changes are rarer, and that asthenia is not so constant. These facts are all, more or less, connected with duration of the disease, for in membranous laryngitis, if the patient survive long enough, all these symptoms may occur, just as they do in faucial diphtheria.

Most cases of laryngeal diphtheria are secondary, that is to say, the membrane has developed first on the throat and then spread to the larynx. Some cases, however, are primary, that is to say, the larynx is the part first affected. Occasionally the trachea is the seat of primary mischief—whence the membrane spreads to the larynx. This used to be described as “Ascending Croup.”

Age and Sex.—Laryngeal diphtheria is especially a disease of children under the age of ten years, though not of course entirely, and the period of greater liability is between the ages of two and five years. It hardly ever attacks the new-born, or suckling infants, and is comparatively rare after the age of seven years.

Steiner's figures for 501 cases show the following age distribution:—

1st year.	2nd.	3rd.	4th.	5th.	6th to 13th.
92	128	87	70	50	74
427.					
= 83·8 per cent.					

The statistics of St. Bartholomew's Hospital for a period of some years gives a percentage of 72 per cent. before five years, and with these the figures of West, 68 per cent., closely agree.

There does not appear to be any marked difference between the sexes in respect of liability, though it is sometimes stated that males are especially susceptible.

The malady attacks all children alike, both weakly and strong, but, of course, the mortality is highest among the former.

Pharyngeal diphtheria in children under ten is especially liable to spread to the larynx, and much more so than in the adult.

In little children with pharyngeal diphtheria, it is stated that in one case out of four the larynx becomes involved. Holt places the average in children under two years of age as high as 42 per cent. In many of these cases the laryngeal

symptoms may be the first to attract attention, for the pharyngeal symptoms may be slight.

Many cases of primary membranous laryngitis are proved clinically to be diphtheria by their giving diphtheria to others, and by the sequelæ.

In cases of infection, where the infection can be traced, the incubation period is generally short, viz., two or three days.

As with other specific fevers, a second attack of diphtheria is far from common, and occurs only at a long interval after the first; while a second attack of laryngeal diphtheria is especially rare and very few authentic cases of it are recorded.

PATHOLOGY.—When the larynx becomes involved, whether secondarily or primarily, the affection commences as a laryngitis, but the surface soon becomes covered with a membrane. The membranes are gray or white in colour, but sometimes dark from altered blood. They are deposited in layers, which are continually reproduced in contact with the mucous membrane, and as they become superficial they may be shed. They vary much in extent, in thickness, and in consistency, being sometimes tough as leather, thick, and difficult to detach, and at other times soft, friable, and easily removed. This depends chiefly upon the age of the membrane and its position, and perhaps also upon the intensity of the inflammation.

Chemically the membranes consist chiefly of fibrin, and are soluble in alkaline solutions, and especially in lime water.

Microscopically they are formed of a network of laminated fibrin, in the meshes of which are contained white and red blood cells, epithelial cells derived from the mucous membrane, and bacilli. In the superficial layers of the membrane many forms of bacilli are found, but in the deeper and more recent layers the diphtheria bacilli are found unmixed with other organisms. The deeper tissues may also, as Kanthack has shown, be invaded.

The membrane is, without doubt, formed as in other cases by a plastic exudation from the blood-vessels, and is not due, as was formerly supposed, to a peculiar degeneration of the cells.

The basement membrane is generally well-preserved, though it may be swollen, irregular, and folded, and on it the false membrane directly rests, unless separated from it in places by a few lymph cells or altered epithelial cells.

The changes in the deeper tissues are those of intense inflammation, accompanied with vascular dilatation and diapedesis, and with the extravasation of red blood cells, which may lead to ecchymoses visible to naked eye. These changes sometimes extend down to the muscles.

When the membrane is removed, the surface looks raw and eroded, but actual ulceration is rare unless the deeper layers of the mucosa have been involved in the membrane and removed with it.

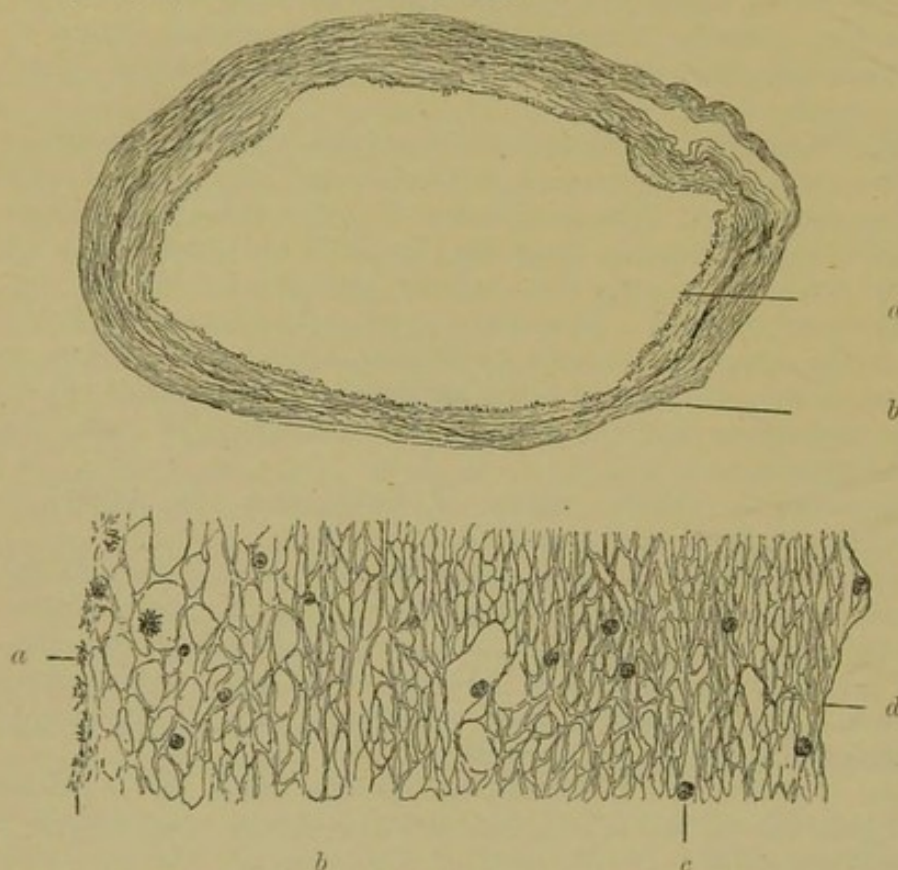
The membrane may be limited to the larynx, but it is usually present in the pharynx also (57 per cent.). In either case it usually spreads to the upper part of the trachea, and may reach down even to the finest bronchi (37 per cent.).

In the larynx, where it may completely close the glottis, it is usually more or less firmly adherent and, if detached, comes away in shreds. In the trachea and larger bronchi it is more loosely attached, and may be expectorated in the form of tubular casts; in the finer bronchi, if membrane be present, it will probably form solid, and not tubular, casts, but as a rule the membrane becomes less and less compact as it becomes distant from the larynx, and often ends in the finer

bronchi as a mucopurulent secretion, so that in the same case every gradation may be traced, from mucopurulent catarrh to membranous inflammation.

Fig. 17.

Section of a tracheal cast, showing laminated fibrin and the position of the bacilli (*Andrewes*). *a*, bacilli; *b*, laminated fibrin; *c*, leucocytes; *d*, indicates the outer surface.



A portion of the same magnified (*Andrewes*). The size of the bacilli is, of course, in both figures much exaggerated relatively to the tissues.

brated case of Watson's, where a complete cast was expectorated seven hours before death, and yet at the *post-mortem* the membrane was found to have been completely reproduced.

The condition of the lung *post-mortem* varies with the duration and intensity of the disease; thus in acute cases the lungs are oedematous and congested, with more or less extensive collapse, and, as in other cases of suffocation, ecchymoses are scattered over the pleura and beneath the mucous membrane of the bronchi. In cases of longer duration, patches of broncho-pneumonia are found with complementary emphysema and with pleurisy over the spots where the pneumonia has reached the surface.

The pulmonary and bronchial glands are usually enlarged. The right side of the heart is dilated, the veins full, and the liver, spleen, and kidneys congested. The lymphatic glands in the neck are enlarged, especially behind the angle of the jaw, if the fauces are involved as well, but if the membrane be limited to the larynx and trachea, glandular enlargement is generally absent.

When broncho-pneumonia develops it is usually excited by the diphtheria bacillus, and, in the alveoli affected, the diphtheria bacilli are often found enclosed in the fibrinous network.

Where the membrane is first formed in the larynx it spreads to the trachea in at least one-third of the cases, but it may start in the trachea first and spread upwards to the larynx, for cases are recorded in which tubular casts have been expectorated some time before laryngeal symptoms developed.

It sometimes happens that no membrane is found *post-mortem*, even where casts or shreds have been expectorated during life; but on the other hand a membrane may reform within a very short time, as in the cele-

The changes in the other organs—viz., the spleen, kidneys, heart, and nervous system—are not, as a rule, found in cases which are fatal early, as most of the cases of laryngeal diphtheria are, but may occur in those cases which die later, after tracheotomy, in the second or third week; in other words, laryngeal diphtheria is fatal before these toxic and septic changes have had time to develop.

During the acute stage there is a febrile leucocytosis which is of some significance. It consists in an increase in the multinuclear neutrophile or finely granular oxyphile cells.

According to Kanthack, high leucocytosis signifies a good reaction and is of favourable prognosis; it decreases during and after the antitoxin treatment; on the other hand a low leucocytosis at the height of the disease is present in nearly all fatal cases. The condition of the blood has not yet been systematically studied during the early stages of convalescence when the anæmia is so pronounced.

SYMPTOMS.—Unless the patient has already been showing symptoms of faucial diphtheria, the attack begins with the symptoms of moderate laryngeal catarrh; the patient is slightly feverish, with somewhat accelerated pulse and respiration; the eyes are suffused and there may be frequent sneezing; the voice is a little hoarse and the cough dry and frequent, recurring in quickly repeated fits of short duration. So far there is nothing to distinguish it from an ordinary feverish catarrh, but in the course of a few hours the voice grows more hoarse, the cough changes its character and becomes short, husky, and muffled, and at the same time dyspnœa sets in with signs of laryngeal obstruction; the inspirations are long-drawn and noisy, the air producing a sibilant sound as it passes through the glottis; soon the expiration becomes also impeded and the breathing stridorous.

It is impossible to describe in words the peculiarities of the stridor and cough in membranous laryngitis, but once heard they are not likely to be forgotten or mistaken.

On examination of the chest, all that is to be detected is inspiratory recession, more or less marked according to the amount of obstruction.

In this stage, the fever increases, the skin is hot and dry, the face flushed and the pulse rapid; the tongue is coated and dry at the centre and tip; there is much thirst; the bowels are confined and the appetite lost.

As the dyspnœa increases, the child becomes more and more restless, tossing itself from side to side and lying only for a few seconds in the same position; the expression anxious and distressed, the alæ nasi dilating, the head thrown back, the larynx moving forcibly up and down with respiration; the inspiratory recession becomes more marked, the veins distended, the heart's action laboured, the skin sweating and the complexion dusky.

The breathing, which was at first loudly stridorous, becomes more sibilant and long-drawn, the voice grows toneless, then whispering, and is finally lost, so that, though the lips move on the attempt to speak, no sound is emitted.

Often after a period of quiet the child springs up suddenly in terror, tearing at its throat and fighting for breath, and when, after a few minutes' struggle, the paroxysm passes off, sinks back in bed exhausted and drops asleep, soon to be awoken again by a similar attack. So one distressing paroxysm follows another, until at last the child has no longer strength to struggle, but lies prostrate, exhausted, dull, drowsy, and difficult to rouse. The breathing grows more shallow and irregular, and the cyanosis deepens, till the final stage of collapse sets in.

This may develop very suddenly and with little warning; the colour then changes, it may be in a few minutes, from the dusky hue of cyanosis to an ashy

or leaden pallor, the child becomes unconscious and may have convulsive fits; the pulse, which has been gradually gaining in rapidity, becomes feeble and fluttering, the respirations grow slower, and at last the patient dies.

The temperature varies much in different cases, being not much raised in some and in others reaching 104° or 105° . The average pyrexia of diphtheria is low, that is, diphtheria is an asthenic fever, both in respect of the temperature and of the condition of the patient.

It is often very irregular, both throughout the day and on different days.

High temperatures are generally associated with pulmonary complications.

The urine presents no special characters, but albuminuria is very common, and that, too, where there is but little dyspnoea, but it may be absent even in fatal cases.

The pulse is almost invariably accelerated even from the first. It is rarely less than 120, but may reach 180 or 200, or be uncountable. A slow pulse is rare, but if, associated with other asthenic symptoms, is of bad omen.

A rapid pulse rate, as it is one of the first signs to appear, so it is one of the last to disappear, and a persistently rapid pulse is an unfavourable sign, though not so ominous as a falling pulse rate while the other symptoms persist or become worse.

Membrane is rarely expectorated by children until, after tracheotomy, it is coughed out through the wound, but, in the adult, casts from the trachea are not infrequently coughed up, even when there is little dyspnoea, and when the chief or only symptom is dysphonia.

In fatal cases the end comes, as a rule, gradually, with the symptoms of carbonic acid poisoning, but death may be sudden, either in one of the paroxysms or from collapse, or the child may have convulsions and die in a fit. In the more protracted cases broncho-pneumonia is likely to develop and prove fatal.

Tracheotomy modifies the course of the disease, but it also introduces a fresh set of risks of its own.

If recovery is to take place the symptoms slowly subside, but convalescence is often interrupted by irregular rises of temperature similar to those so common after broncho-pneumonia, and due probably to the same causes.

DURATION AND COURSE.—The duration of the different stages varies greatly. The first is usually short and rarely lasts more than thirty-six hours before the dyspnoea, which is the characteristic of the second stage, commences. The onset may, however, be so sudden that the first stage may seem to be absent. When collapse sets in the patient has but a few hours to live. The duration of the second stage is very variable, from a few hours to several days, but on the average it does not exceed two or three days.

According to Steiner the first stage averages from one to three days, the second from one to fourteen days, while the third rarely lasts more than from twenty-four to thirty-six hours.

The average duration of the disease is about three to five days, but it may run its course in a few hours.

Thus West¹ records a case in a healthy child of four, which was fatal in fourteen hours. On the other hand cases may be protracted over two or three weeks.

The disease rarely runs a uniform course, but is subject to most deceptive remissions, during which the child seems on the road to recovery; then come the exacerbations which occur most frequently at night and which may be attended with the paroxysms already described.

These paroxysms are referred to several causes, but probably in chief part depend upon the accumulation and drying of secretion round the glottis, or to

¹ *Diseases of Children*, p. 398.

the impaction of a piece of membrane in it; they are also attributed to muscular spasm.

The collapse stage may set in at any moment, often in the most unexpected way, and so long as dyspnoea continues the risk of this exists.

Just as a change for the worse may set in suddenly, so may relief be sudden, when the membrane is expectorated, but, as already stated, the membrane may rapidly reform and the dyspnoea return.

Even when urgent symptoms are past, convalescence is slow, the patient being left weak and ailing, and requiring most careful watching and nursing for some time.

COMPLICATIONS AND RISKS.—In the early stage of the disease the chief dangers are from suffocation owing to the obstruction of the glottis, and from the pulmonary complications to which the inflammation may give rise. In the later stage secondary septic complications may occur, especially if tracheotomy has been performed.

Cardiac collapse is a danger throughout, and may give rise to very alarming attacks or lead to a fatal result, and that quite suddenly.

Lastly, asthenia may be a prominent symptom in the disease throughout, and may lead to death without any actual complication, the patients looking extremely ill and seeming to die because they have not strength enough to get better.

The anaemia and asthenia may last long, even for many weeks, after the urgent symptoms have passed away. These are no doubt toxic effects connected with the blood changes which the disease has produced.

The secondary pulmonary complications depend chiefly upon the extension of the membrane or of the inflammation from the larynx and trachea to the smaller air-tubes. In these cases tracheotomy gives little or no relief, or if it has given relief at first the dyspnoea returns.

Bronchitis and broncho-pneumonia are the chief lesions produced. They are usually excited by the diphtheria bacillus itself and not by streptococcal or other secondary infection (Kanthack), and this occurs also even where there is no direct continuity of the membrane to be traced along the air-tubes to the affected vesicles of the lung.

Collapse and complementary emphysema are the more or less mechanical results of the obstruction to the air-tubes and pass off when the obstruction is relieved.

The onset of pulmonary complications is shown frequently by a rise in temperature, but chiefly by increase of the dyspnoea or by a return of dyspnoea if tracheotomy has been performed previously.

The physical signs are so masked by the laryngeal stridor and by the defective entry of air, that they are of very little use in determining the condition of the lung. Even when dulness to percussion is found, it may not be easy to distinguish between inflammatory consolidation and collapse of the lung or pleuritic effusion.

Causes of death.—In the *early stages* death is brought about chiefly by suffocation or asthenia, or by a combination of the two.

The suffocation is for the most part gradual and due to the obstruction of the glottis produced by the membrane. This condition can be more or less successfully remedied by intubation or tracheotomy. In other cases it is due to the extension of the membrane from the larynx along the trachea and bronchi. For this there is no relief except the expectoration of the casts; the membrane is rarely found to be limited to the larynx, but in fatal cases has usually spread far down the tubes.

In 57 per cent. of fatal cases, according to Northrup,¹ it reached the main bronchi, and in 37 per cent. it extended even to the finest bronchi.

Where the suffocation is sudden, that is to say, where there is a sudden and perhaps fatal paroxysm of dyspnoea, the attack is sometimes attributed to muscular spasm, but it is more likely to be due to the sudden detachment of a portion of membrane and its impaction in the already narrowed glottis.

In the *later stages*, pulmonary complications are the chief source of danger, to which may be added various septic changes, the risk of which is increased by tracheotomy.

Thus, after tracheotomy the wound itself can hardly escape becoming infected and a source of septic absorption. The infection may then travel along the cervical fascia to the mediastinum or to the pleura, and thus lead to empyema, pericarditis, or even mediastinal suppuration, while, in other cases, general septic infection is produced, which is commonly described as pyæmia.

These complications are of course of less frequent occurrence now than they used to be; but in spite of all antiseptics, the risk to some extent must still continue.

In other cases air may be forced along the cervical fascia to the mediastinum, and, by rupturing into the pleura, cause pneumothorax. This is not a very common occurrence, and is probably only met with in cases where there is violent dyspnoea and where the incision through the skin and deep fascia has not been sufficiently free (*cf.* chapter on Pneumothorax).

Even when urgent symptoms have passed off and no grave complications on the part of the lung have developed, the risks of asthenia and cardiac failure continue for some time.

The cardiac weakness in these cases may be often betrayed by a considerable dilatation manifest on percussion, and sometimes by the presence of murmurs. All these signs may be present on examination and yet betray themselves by no symptoms; at other times patients may have, with or without this obvious dilatation, sudden attacks of cardiac syncope of a very alarming or even fatal character. In a certain number of cases, death occurs unexpectedly during the second or third week after all immediate risk seems to be past, and at a time when the patient may be regarded as convalescent.

Often in the later stages of the attack and for some time during convalescence profound asthenia and anæmia continue. The asthenia is probably to be connected with the leucocytosis which has developed during the later stages of the disease, and which is only recovered from in course of time.

SEQUELÆ.—Of these there are usually none, unless they are such as have been left behind by some of the complications mentioned.

Paralysis seems to be comparatively rare after laryngeal diphtheria, unless there has been considerable pharyngeal diphtheria as well, probably because most of the severe cases of laryngeal diphtheria do not survive. Now that the mortality has been so much reduced by the antitoxin treatment, the frequency of paralysis after laryngeal diphtheria will be probably greater.

In the adult, the local symptoms are less severe, first, because the larynx is larger; secondly, because adults are better able to cough and thus more easily expectorate the membrane and secretion in the tubes; finally, because tracheotomy is less often necessary and when performed has better results. At the same time, asthenia and other toxic symptoms are more common,

¹ *Cf.* Holt, *l.c.*

for the reasons given, viz., that the cases are of longer duration and, therefore, have more time for toxic absorption, and that primary laryngeal diphtheria is much rarer and faucial diphtheria more common, or in other words, that the larynx is less liable to be involved during the course of faucial diphtheria.

On the other hand, primary tracheal diphtheria and chronic diphtheria are rarely met with except in adults.

Primary tracheal diphtheria is rare, that is to say, cases in which casts of the trachea are expectorated without any laryngeal symptoms at all, or in which the laryngeal symptoms have only developed later. The latter has been described as "Ascending Croup."

Steiner records two good examples in children, in one of which laryngeal diphtheria did not develop for a week, and in the other for fourteen days, after the bringing up of tracheal casts.

Chronic diphtheria.—In other cases after ordinary attacks of laryngeal diphtheria, casts may be expectorated from the trachea after all laryngeal symptoms have long passed off. This has been termed chronic diphtheria. In some instances casts have been expectorated on successive occasions during apparent convalescence.

In one case, a tracheal cast was brought up as long as 49 days after the initial attack.

I have recently had a similar case in a child of four years of age, in which the tube could not be dispensed with for many weeks, and in which membrane was brought up (not a cast) 46 days after tracheotomy.

PROGNOSIS.—The prognosis of laryngeal diphtheria in children is always grave, but it has been greatly improved by the antitoxin treatment. It was formerly stated that in children not more than 10 per cent. escaped tracheotomy, and that with tracheotomy not less than 70 per cent. died. It may be stated in a general way that prior to the introduction of the serum treatment two out of every three children upon whom tracheotomy was performed died; whereas now, under the serum treatment, perhaps as many as two out of three recover. (See Serum Treatment, below.)

The **general prognosis** depends upon many conditions.

1. *Age.*—Laryngeal diphtheria is fortunately rare during the first year of life, for it is extremely fatal then. It is stated that of infants under one year of age about 98 per cent. died before the serum treatment, and that under two years the mortality was over 80. So fatal were the results of tracheotomy under two years of age, that many physicians used to hold that at that age the operation took away, by the risks it introduced, even the little prospect of recovery the patient might have. Now, cases of recovery under two years of age are not uncommon.

2. *The character of the epidemic.*—Some epidemics are severe, others slight, and the character of the epidemic may to some extent suggest the course the case is likely to run; but slight cases may occur in grave epidemics, and *vice versa*.

3. *The gravity of the case.*—This, in the early stage, may be measured by the amount of membrane on the throat, for in these cases the absorption is considerable and the constitutional symptoms severe.

4. *The duration of the case before treatment was undertaken.*—It is shown that success after serum treatment depends largely upon the time at which it was commenced, the delay of a day or two or even of a few hours making all the difference in the results.

5. *The duration of the laryngeal obstruction before tracheotomy was performed.*—In other words, whether with serum treatment or not, the sooner

tracheotomy is performed, if it has to be done, the better, for the longer it is delayed, the greater the prospect of the membrane extending to the tubes and of the lungs becoming secondarily affected either by collapse or by pneumonia, and the greater the risk of the strength being exhausted by the prolonged struggle for breath.

6. *The relief given by operation.*—When the relief is immediate, the prognosis is so far good; but, when tracheotomy gives little relief, the membrane has probably extended far into the air-tubes and the dyspnoea is thus only partly laryngeal in origin. The dangers then arise chiefly from the more extensive surface affected and the greater absorption of toxin in consequence, and only in part from the obstruction produced by the membrane.

7. *The extent to which the lungs and air-tubes are involved.*—Inflammation of the lungs may develop early or after a while whether the patient has been operated on or not; but provided the air-tubes are free from membrane, the prognosis is not much worse than that of secondary broncho-pneumonia in general. The prognosis is then determined chiefly by the age, but also by the strength of the patient and the extent to which the vitality has suffered in consequence of the previous illness. If the strength be good, recovery may occur, as in other cases of broncho-pneumonia. The serum treatment should in these cases not be given up, but on the contrary pressed, for the broncho-pneumonia is often itself of diphtheritic origin, *i.e.*, due to the diphtheria bacillus.

Prognosis as indicated by physical signs. (a) *Colour.*—The cyanosis varies with the dyspnoea. If the colour does not improve after tracheotomy, or if after temporary relief the cyanosis return, it is evident that the membrane has involved the air-tubes or that the lungs are otherwise affected.

A still worse sign is the ashy pallor which takes the place of cyanosis, often quite suddenly. This is almost invariably the precursor of death.

(b) *Pulse.*—The pulse is generally rapid and out of proportion to the temperature. Great rapidity is a bad sign, but so also is a considerable fall in the rate. Worst of all is marked irregularity, unless it depend directly upon the amount of dyspnoea, when it may become intermittent with inspiration (*pulsus paradoxus*).

(c) *Respiration.*—As the case nears its end the respirations become shallow, superficial, and rapid, but as the strength fails they grow slower and slower, and with this change a good deal of irregularity occurs also, which sometimes assumes the Cheyne-Stokes type.

(d) *The amount of recession of the lower ribs.*—This is always a bad sign for, if it cannot be relieved by tracheotomy, it must inevitably end in extensive collapse of the lung, and, if life be prolonged, in pneumonia.

(e) *Leucocytosis.*—In mild cases there may be no change in the number of white cells. In all severe cases a marked leucocytosis occurs, and is, as in pneumonia, of favourable import as indicating a good reaction; *per contra*, the absence of it is so far a bad sign.

During convalescence the prognosis varies:

(1) *With the complications* which have developed, *e.g.*, broncho-pneumonia or empyema.

(2) *With the general strength.*—It is not rare to see patients die in the second or third week after tracheotomy from sheer exhaustion without any definite cause. This is no doubt a toxic effect. In some the end is sudden, by cardiac failure, and in this respect the dilatation of the heart, which can easily be made out by physical signs, is a warning of the need of care.

(3) *The difficulties with the tube.*—Children may for a long time be unable to do without one. This depends sometimes upon defective action of the vocal

cords, which do not separate as they should on inspiration, but remain in approximation. They may sometimes be restored to activity by passing a probe through the larynx or by electrical treatment. More often the difficulties depend upon alterations which the tube has produced in the trachea either by vegetations near the incision or by ulceration or erosion, or sometimes even necrosis of a cartilage produced by the rubbing of the end of the tube.

In a case of this kind in which a child, aged about four years, died seven weeks after tracheotomy, having been unable to dispense with the tube, the cause was found to lie, in the first place, in granulations which had formed round the opening in the trachea, not below but above, almost completely occluding the passage to the larynx, and, secondly, in erosions produced by the end of the tube in the wall of the trachea which had led to the necrosis and partial detachment of one of the cartilages.

It is remarkable how completely healing takes place when the tube has been removed, for after the tube has once been got rid of bad results from tracheotomy are very rare.

TREATMENT.—The treatment of membranous laryngitis in its early stages is chiefly symptomatic and is the same as that of catarrhal laryngitis.

Remembering the asthenia which is so likely to develop, the strength should be kept up in every way. Thus, food of a nutritious and easily digestible character should be given at short intervals, and if there is any difficulty in getting food taken, either on account of loss of appetite or of discomfort and pain on swallowing, nasal feeding should be at once adopted. There is nothing which so greatly tends to reduce the complications and mortality of diphtheria in little children, both before and after tracheotomy, as the routine practice of nasal feeding.

Stimulants will be necessary to some small amount; thus ten or twenty drops of brandy may be given every hour to a child of two or three years old, and more, of course, to older children. At the same time a little iron and strychnine will be useful if the digestion can stand it. In cases of collapse, subcutaneous injection of liquor strychninæ and caffèin are of great service.

When the dyspnœa is considerable, before the question of intubation or tracheotomy is raised, emetics have been advocated with the view of dislodging the membrane from the larynx. Emetics, however, especially in children, cause great depression and rarely produce the desired result; they are now practically abandoned, at any rate as part of routine treatment.

Of **surgical operations** the choice lies between intubation and tracheotomy. The objection to intubation is that in introducing the tube the membrane may be thrust down and thus aggravate the dyspnœa or even produce fatal suffocation. Though cases of this kind are recorded, these accidents but rarely occur, and intubation has no doubt this advantage over tracheotomy, that no serious operation has been performed.

Tracheotomy, on the other hand, is not free from objection. There are always difficulties in the management of the tube, for it is small, easily choked, and difficult to cleanse. At the same time greater relief is likely to be given by tracheotomy, because large pieces of membrane which could not be forced out through a small tube can be expelled through the incision at the time of the operation and afterwards whenever the tube is taken out.

Until lately the balance of opinion was strongly in favour of tracheotomy as against intubation, but since the introduction of the serum treatment opinion seems to be veering round again in favour of intubation.

If tracheotomy is to be performed it should not be put off too long, for the longer it is postponed the more and more congested and collapsed the lungs

will become, while the strength of the patient will be greatly exhausted by the constant struggling for breath and by the want of sleep and food.

Serum treatment.—In every case of diphtheria where laryngeal symptoms are present, whether pronounced or not, the serum treatment should be commenced without a moment's delay, for the success of the treatment depends largely upon its having been commenced in good time.

This general rule holds quite irrespective of whether there be different kinds of membranous laryngitis or not, for the majority are diphtheritic, and we have no means of ascertaining, at the time, which cases are, and which are not, diphtheritic, and the serum treatment, so far as we know, is not productive of any harm.

The general effect of the serum treatment is shown locally by the loosening and detachment of the membrane, and generally by the improvement which takes place in the general condition.

It is not shown that the serum treatment has any direct effect upon the sequelæ of the disease, *e.g.*, the occurrence of paralysis, yet there is every reason to believe that it must diminish the risks of this also, though it is difficult actually to prove it by figures.

The use of the serum is sometimes attended by the appearance of rashes, for the most part of an erythematous or urticarial nature, and sometimes by a little swelling of the joints and pains in the limbs. These accidents, however, depend probably upon the serum employed and not upon the antitoxin, the injection of which appears to be perfectly innocuous in itself.

The serum is generally injected into the buttock. After seeing that the needle and syringe have been carefully sterilized, the skin should be washed and thoroughly disinfected. After the injection the puncture should be covered with some aseptic plaster.

The usual dose of the serum injected is one which contains 4000 units. In many of the preparations this is equivalent to about 10 c.c. of the serum. 2000 units is the usual dose for little children, say under two years of age; for adults a larger quantity may be injected, up to 4000 units or more.

The injection should be made once or twice daily, and in bad cases the injections may be repeated at shorter intervals. Larger and more frequent doses are necessary if the case has come later under observation.

The amount of serum used will vary with its strength, with the size and age of the patient, with the amount of membrane, and lastly with the general gravity of the case.

Where there is reason to believe that the membrane has spread down the trachea the serum treatment should be pressed, for where so large a surface is covered by membrane, the amount of toxin produced is large, and its absorption rapid. Even when broncho-pneumonia develops, the serum treatment should still be actively pursued.

The effects of the serum treatment are shown first by a reduction in the mortality from diphtheria in general, secondly by a reduction in the percentage of laryngeal cases occurring in the course of faucial diphtheria, and thirdly by the diminished mortality in laryngeal cases.

(a) *The effect on the general mortality.*—Baginsky¹ has shown, as the result of the statistics of a very large number of cases, that the general mortality before the use of serum was 48·4 per cent., and after its use 15·6 per cent. With this Welch,² whose percentage is 17·3, closely agrees.

Herringham's³ statistics, obtained from St. Bartholomew's Hospital, correspond closely with these, the percentage in children of all ages up to ten years being 52·7 before the use of serum and 16·0 after; of those under five, 70·0 before the use of serum and 44·0 after.

¹ *Die Serum-Therapie der Diphth.*, Berlin, 1895.

² *Trans. of Assoc. of Amer. Phys.*, 1895, vol. x.

³ *St. Barthol. Hosp. Rep.*, 1895.

The mortality from diphtheria is greatest in the very young, and decreases with each year of age, but every age period alike shows a similar decrease in mortality.

Thus Welch states that under 2 years of age the total mortality from diphtheria in general was 66 per cent. before the use of serum and after it 33 per cent.

(b) *The frequency with which the larynx becomes involved in the course of faucial diphtheria.*—In children this seems to be on the average about 1 in 4, i.e., 25 per cent. In little children the percentage is even higher. Holt states it to be 42 per cent.; Ganghofner puts it at about the same rate, viz., 2 in 5.

Where laryngeal symptoms were present Welch states that before the use of serum 92 per cent. required operation, but after the use of serum this was reduced to 66 per cent., showing an improvement of about 25 per cent. Ganghofner's figures also show that with the use of serum recovery in about 25 per cent. takes place without operation.

(c) *The results of operation.*—These show the same improvement.

After tracheotomy, the mortality averaged :—

Before serum treatment.	After serum treatment.
60 per cent. at least.	37.4 per cent. (Baginsky)
70 " "	33.9. (Various sources, chiefly German.)
66 " "	39.8. (Welch)
	43 to 48. (<i>Clin. Soc. Rep.</i> , 1898.)

Tracheotomy has hitherto been generally preferred to intubation, but with the use of serum, it seems that the results of intubation are somewhat better than those of tracheotomy. Thus Baginsky states that with tracheotomy the mortality was 37.4 per cent., and with intubation 25.9 per cent.; with this Welch's figures also agree.

The mortality of tracheotomy varies very greatly with the age; thus before the use of serum almost every child under the age of 1 died, and the total mortality under 2 was about 70 per cent., whereas by the use of serum it has been reduced to 33 per cent. This is, roughly speaking, the experience at St. Bartholomew's Hospital also, for whereas in little children 2 cases out of 3 used to die, now with the use of serum 2 cases out of 3 recover. The following table of Tirard's¹ is instructive as showing the decrease of mortality as the age increases and the improvement which follows the use of serum at all periods.

PERCENTAGE MORTALITY ACCORDING TO THE METROPOLITAN ASYLUM BOARD,
LONDON, IN RESPECT OF TRACHEOTOMY.

	Before serum treatment.	After serum treatment.	Improvement.
Under 1 . . .	80	75	12 per cent. under 2.
" 2 . . .	87 } 83.5 per cent.	68 } 71.5	
" 3 . . .	76	42	26 per cent. over 2.
" 4 . . .	62	49	
" 5 . . .	71	42	
5 to 10 . . .	62 } 68 per cent.	36 } 42	

This table also shows that, under 2 years, the mortality, which before serum treatment was 83.5 per cent., fell after serum treatment to 71.5 per cent., equivalent to a diminution in the mortality of 12 per cent.; and that, above 2 years, i.e., from the ages of 2 years to 10 years, the mortality which previously was 68 per cent. fell to 42 per cent., equivalent to a diminution in the mortality of 26 per cent.

In fact the introduction of the serum treatment has robbed laryngeal diphtheria of literally half its terrors.

Treatment during convalescence.—Diphtheria is a serious ailment and produces effects upon the general health which are often out of proportion to the apparent gravity of the initial disease. Convalescence will therefore be protracted and difficult. The children are often left anæmic and feeble for a considerable time, and require the greatest care. They must be kept as far as possible at rest, supported with tonics and good food. Even where no actual paralysis occurs the muscular power is greatly reduced, and fatigue must be carefully avoided. The necessity of rest is shown by the frequency with which dilatation of the heart is met with during convalescence.

¹ Tirard, *Diphtheria*, 1897.

This may produce no actual symptoms and yet be easily detected by physical examination. If neglected, however, so that the children are allowed to get up and exert themselves too soon, the ordinary signs of cardiac weakness will present themselves, *e.g.*, palpitation and shortness of breath, and recovery may be seriously retarded.

The most useful tonics during convalescence are quinine, iron, and strychnia, with occasionally, if the heart be very feeble, a little digitalis.

Quarantine.—An important question presents itself during convalescence as to how long the children should be kept in quarantine and regarded as a source of danger to others.

This general rule may be laid down, that under no circumstances should they be permitted to mix with other children under about three weeks from the termination of their illness, even if apparently well. Diphtheria bacilli of a virulent kind may be found in the throat for a considerable time after faucial diphtheria, as the following table shows, and the risks are probably much the same with laryngeal diphtheria.

In 605 cases examined for the purpose by the New York Health Department after the membrane had disappeared, virulent bacilli were found for 3 days longer in 50 per cent. of the cases.

"	7	"	"	30	"
"	12	"	"	10.6	"
"	15	"	"	6	"
"	21	"	"	2	"
from 4 to 9 weeks				2	"

Tobiesen also found virulent bacilli in the throat of patients at the time of their discharge from the Hospital in 50 per cent.

Many of the statements made upon this point must be received with caution, now that it is known that the morphological test alone is not sufficient for the recognition of the diphtheria bacillus, and that it must be confirmed by the inoculation test. There can be no doubt that many convalescents from diphtheria have been wrongly condemned as still infective upon the result of the morphological test alone.

The course of convalescence will be modified by the occurrence of any complications during the acute illness.

If broncho-pneumonia has occurred, the patients will require the same care as after broncho-pneumonia from any other cause. Diphtheritic broncho-pneumonia, if not fatal at the time, does not seem to lead to any specially disastrous results. Of course, if an empyema has developed, the course of convalescence will be determined largely by this, and so in the case of other complications.

It has already been stated that paralysis does not seem to be so common after laryngeal diphtheria as after faucial diphtheria, and it is remarkable, seeing how often the palate becomes paralysed after faucial diphtheria, that it should be so very rare for the vocal cords to be affected and the voice to be impaired during convalescence.

NON-DIPHTHERITIC MEMBRANOUS LARYNGITIS.—It is known that membranous laryngitis can be produced by various simple traumatic causes as well as by forms of bacteria, other than the Klebs-Löffler bacilli.

Of the **traumatic causes**, scalds from inhalation of steam are the commonest.

But besides these the affection has been described as resulting from the irritation of a foreign body in the larynx, as occurring in the course of malignant and tubercular disease of the larynx, and in connection with both acute and chronic pneumonia; after tracheotomy for syphilitic laryngitis, and after a cut throat (Fagge¹). Most of these cases date from pre-bacterial days, and are not therefore altogether conclusive.

¹ Fagge, *Guy's Hosp. Rep.*, 1897. Parker, *Clin. Soc. Trans.*, 1875.

The most remarkable case of the kind is that recorded by Dr. T. Whitehead Reid, where a lady, on recovering from a faint, took a deep breath and sucked in at the same time Eau-de-Cologne. After suffering greatly from dyspnoea, she was relieved by the expectoration on the fifth day, 92 hours after the accident, of a cast of the larynx and trachea and part of the left bronchus. She continued to spit up pieces of membrane until the seventh day and then recovered.

Membranous laryngitis has also been produced experimentally by the direct application of irritants and caustics such as *Liquor Ammoniae*.

All these cases are of more interest than importance, for the great majority of cases of idiopathic membranous laryngitis are undoubtedly of bacterial origin.

Of the **bacterial forms**, it has been shown that, besides the diphtheria bacillus, the pseudo-diphtheria bacillus, streptococci, and possibly also other pyogenic cocci may produce a membranous inflammation of the fauces; while the pneumococcus and possibly others may produce a membranous inflammation of the air-tubes.

We may presume that what is true of the fauces is true also of the larynx; for instance, it is proved that the membranous inflammation of the pharynx and of the larynx which occur in connection with scarlet fever is often of streptococcal and not of diphtheritic origin. This was an opinion long held by many clinical observers, but one which has only been recently confirmed by bacteriological investigation.

There is also a clinical difficulty which has to be met, and which cannot with safety be entirely disregarded because it does not fit in exactly with current bacteriological theories. The difficulty is this: in epidemics of faucial diphtheria the larynx becomes involved in something like 25 per cent. of the cases; we might expect, therefore, that if membranous laryngitis was simply laryngeal diphtheria, there would be at the same time a very large number of pharyngeal cases, say two or three times the number at least; for instance, if there were a hundred cases of membranous laryngitis admitted to a hospital, this ought to correspond with, at least, two or three hundred cases of pharyngeal diphtheria. Many of these cases would be mild, no doubt, and not come under observation, but still, making all allowance for these, there ought to have been an epidemic of diphtheria, such as did not prevail.

From these considerations it seems to follow—

1. Either that primary laryngeal diphtheria is a very much more common affection in children than has been hitherto supposed, and that for some reason or another it does not occur in epidemics as faucial diphtheria usually does; or
2. That, there are two varieties of membranous laryngitis, the one diphtheria of the larynx, primary or secondary, and the other non-diphtheritic. It is this view which I think best accords with the clinical facts; it is the view also which has been held by many thoughtful clinical observers up till quite recent times, and it is one which there now seems to be good bacteriological evidence to support.

9. ŒDEMA OF THE LARYNX (ŒDEMA GLOTTIDIS).

There are two forms of œdema of the larynx, the one inflammatory and the other of the nature of dropsy and non-inflammatory. They are conveniently distinguished by the terms œdematous laryngitis and hydrops laryngis.

The old term œdema glottidis is inaccurate, for many of the cases are not œdema, and it is not the glottis that is affected but the parts surrounding it.

I. Hydrops Laryngis.—The non-inflammatory form is rare. It occurs as a rule in the last stages of general dropsy, most frequently in the course of acute or chronic Bright's disease, although a few cases are recorded in the course of the dropsy of heart disease, emphysema and cirrhosis of the lung.

Peltesohn¹ in 3887 autopsies found œdema of the larynx 210 times—149 males, 40 females, 21 children. The ages of the males varied from 18 to 60, of the females from 21 to 54. In the children 13 cases were less than 5 years old. In 44 cases there was local disease in the larynx, syphilis, tubercle, perichondritis, etc. In 164 cases it occurred in the course of general dropsy.

Hoffmann,² in 6062 autopsies, found that œdema of the larynx was the cause of death in 33 cases—in 10 as the result of local disease, in 23 of general disease.

The former figures represent the frequency of the condition in general (=5·4%), and the latter its frequency as the cause of death (=0·5%), and shows its great rarity.

Hydrops Laryngis, when bilateral or general and rapid in its development, produces sudden and severe symptoms, but a local œdema, especially of the arytenoid folds, is often met with, *post-mortem*, in Bright's disease, which, having been unattended with symptoms during life, would be easily missed unless specially looked for. Fauvel states that œdema of the larynx may be an early or even the first symptom of Bright's disease, but I do not know upon what facts this statement rests, and I have never seen anything to justify it.

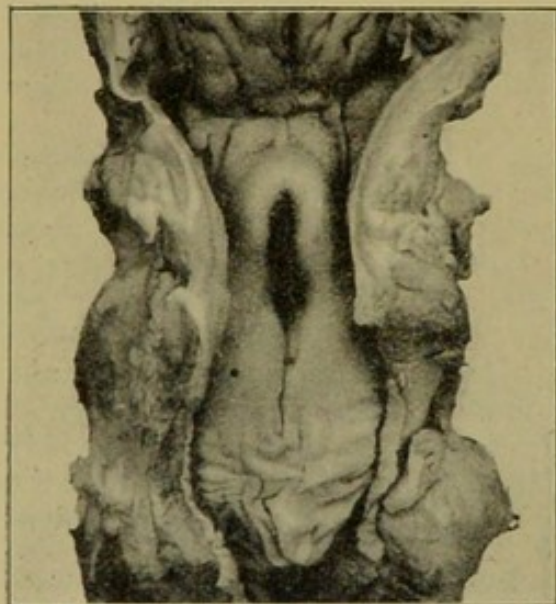


Fig. 18.

œdema of the glottis. From a case of syphilitic disease.

course of goitre, of new growths and lymphatic enlargements in the neck, of aortic aneurysm, and mediastinal tumour.

In a case of chronic parenchymatous nephritis with great general dropsy, which was under my own observation, general hydrops laryngis developed suddenly with very severe symptoms. Tracheotomy was performed with immediate relief, but the patient died shortly afterwards of general asthenia.

Angeio-Neurotic œdema (œdematous urticaria).—Under this name a curious condition is described in which swellings occur in various parts of the body, chiefly in connection with the skin. They develop quite suddenly and reach a considerable size. I have seen the lips swollen to such a size that the mouth could not be opened, and the cheek and eye so swollen that the features of the affected side were quite obliterated, and I have seen the whole scalp affected so that the hat rested on a sort of shelf an inch wide all round the head. Swellings such as this may appear in one hour and disappear as rapidly; they sometimes come on without any obvious cause, but in other cases may be produced at will by certain articles of food or by drugs. Thus I have seen a boy whose lips became enormously swollen an hour after drinking milk.

¹ *Berl. kl. Wochensc.*, 1889, p. 959.

² Quoted by Bosworth, *l.c.*

The present interest of these cases lies in the fact that such swelling may sometimes affect the larynx. Osler¹ refers to two fatal cases of this kind in the same family. Nothing so serious as this has ever come under my own observation, but I have seen one or two instances in which attacks of apparent asthma might be explained by this condition.

II. Œdematous Laryngitis.—The inflammatory form occurs both as a primary and as a secondary affection. The primary or idiopathic affection is the rarest of all. It is an inflammation of the greatest severity and has been well named phlegmonous laryngitis, for it is attended with rapid and massive brawny swelling, and short as its duration is, the tissues may be found suppurating or even almost gangrenous. It is a septic infection and invariably fatal.

With this rare exception œdematous laryngitis is always secondary to some inflammation either in the larynx itself or in its immediate neighbourhood. It may arise in any form of inflammation, but with the exception of the acute catarrhal laryngitis of children and of that which occurs occasionally in some of the specific fevers, *e.g.*, scarlet fever, measles, small-pox, typhoid, and erysipelas, it is only met with in the more intense forms of inflammation. Thus it may follow violent mechanical irritation produced by the inhalation of scalding steam or hot air, of irritating vapour like bromine or strong ammonia, or of corrosive liquids such as mineral acids or caustic alkalis.

In another very important group of cases the swelling is due to the irritation set up by a foreign body impacted in the larynx, especially by such as are of irregular shape and have jagged edges, like a fishbone, so that they produce more irritation than obstruction.

Apart from these mechanical irritations the most common cause of œdematous laryngitis is found in suppurative processes within the larynx itself. Of these the most frequent is perichondritis, to whatever cause it may be due, tubercle, syphilis, or cancer, etc.

The inflammatory processes external to the larynx to which the laryngitis may be secondary, are seated in the pharynx or in the tissues of the neck. If the inflammation spread from the neck to the larynx, as it may in the course of angina ludovici or with suppurating wounds or erysipelas, it is phlegmonous in character and almost invariably fatal.

With affections of the pharynx it is relatively rarer and not so fatal. Thus it may follow tonsillitis, parotitis, retropharyngeal abscess, or other pharyngeal suppuration. It may also follow faucial diphtheria, but hardly ever unless there be extensive sloughing.

Pathology.—The essential pathology of œdema of the larynx is simple, but the condition of the larynx varies greatly according to the cause, extent, duration, and intensity, from a slight local dropsical œdema to a general brawny phlegmonous infiltration.

The parts most affected are, of course, those which are loosest in texture, *viz.*, the arytenoid epiglottidean folds, the epiglottis, and the false cords, while the true cords themselves and the parts beneath them are but rarely involved. In some cases the swelling may be sufficient to almost entirely close the glottis, but the amount of swelling visible after death is often far less than was known to have existed during life.

Below the vocal cords (*i.e.*, infraglottic), œdema of any kind is uncommon, and below the larynx very rare indeed.

Acute œdema of the trachea and even of the bronchi has been met with now and then as the results of burns, but most of the cases described

¹ *Amer. Jour. of Med. Sc.*, 1888, vol. xcv. p. 362. Strübing, *Zeitsch. f. kl. Med.*, 1885, ix. 381.

in these regions are really chronic inflammatory infiltrations, and will be referred to later.

It is stated that iodide of potassium may produce œdema of the glottis, and that this is generally infraglottic. This must be very rare considering the frequency with which the drug is administered, and most probably depends upon the lesion of the larynx for which the iodide of potassium has been given, *e.g.*, syphilitic ulceration.

SYMPTOMS.—The symptoms are those of laryngeal obstruction, complicated with those due to the disease to which the œdema is secondary. They are, as a rule, of sudden onset; they may be so severe as to lead to suffocation in a few minutes, but such extremely acute attacks are rare. On the other hand, considerable partial œdema may exist without any urgent symptoms at all for some time; such cases require most anxious watching, for they may become critical at any moment; the tracheotomy instruments should be at hand, and the patient never left.

DIAGNOSIS.—In children this has to be made from other causes of sudden laryngeal obstruction, especially from the impaction of a foreign body, and from laryngismus stridulus.

In adults similar symptoms may arise in the course of aneurysm of the aorta and mediastinal tumour, and that without any cause being found *post-mortem* to explain them, and they are then usually referred to laryngeal spasm.

TREATMENT.—If the symptoms are not very urgent the ordinary remedies for laryngitis or for any other affection present may be employed. Thus cold may be applied externally by an ice-bag or Leiters tubes; ice may be given to suck; or three or four leeches applied over the larynx externally.

A subcutaneous injection of pilocarpin (grain $\frac{1}{8}$) has been recommended, but it is an uncertain and risky drug for this condition.

These measures failing or without waiting to try other treatment, if the symptoms are urgent, nothing remains but operative interference. Scarification has been recommended after painting the parts with cocaine (20 per cent.), but it is uncertain in its effects, and cannot be used in urgent cases. Intubation might seem indicated in preference to tracheotomy, but the swelling makes it very difficult or impossible to introduce the tube. Tracheotomy is the simplest and most satisfactory operation in all urgent cases, and should not be delayed.

10. NERVOUS AFFECTIONS OF THE LARYNX PRODUCING RESPIRATORY OBSTRUCTION.

Nervous affections of the larynx are of importance in respect of respiration in two ways, according as, on the one hand, they produce respiratory obstruction or, on the other, permit the passage of food into the air-tubes.

The latter group of affections has been already sufficiently dealt with when the subject of foreign bodies in the air-tubes was under consideration.

The neuro-muscular affections which produce respiratory obstruction are two only, *viz.*, adductor spasm and abductor paralysis, both alike leading to closure of the glottis.

ADDUCTOR SPASM—SPASM OF THE LARYNX.—Spasm might affect any one of the muscles of the larynx or any group of them, but practically we have to reckon only with adductor spasm; and for this there appear to be two obvious and simple reasons. In the first place, the adductors are the stronger set of muscles by far, there being five adductors and only one

abductor; and, in the second, they are constantly associated together in action for a variety of important purposes, viz., phonation, coughing, and closing the glottis to exclude foreign bodies or to fix the thorax for muscular effort.

Laryngeal spasm is almost always bilateral, and due, as Marshall Hall long ago asserted, in the great majority of cases, to reflex stimulation of the corresponding centres in the cord. These centres might be excited to action (*a*) by peripheral irritation of many sensory nerves, especially, of course, the pneumogastric, and (*b*) by descending stimuli from the higher centres, while (*c*) the reflex excitability would be greatly increased by any exaltation of irritability in the centre itself, or by any diminution in cerebral control. Semon and Horsley refer the attack to excessive irritability of the cortical adductor centres, consequent on defective nutrition. This theory is but another way of associating it with epilepsy.

Irritation of the superior laryngeal nerve is, as would be expected, the most likely to produce the spasm, and accordingly in a large number of cases the seat of irritation is found within the larynx itself, or perhaps in the trachea. Thus in children the attacks are often preceded by slight catarrh, and, in the adult, are not infrequently associated with gross disease of the larynx.

After the superior laryngeal nerve, the most important seat of irritation is in the trunk of the pneumogastric itself, especially in relation with its recurrent branch. The seat of mischief is then commonly in the mediastinum, and in this way adductor spasm may complicate thoracic aneurysm and mediastinal growth. In other cases the peripheral stimulation has been referred to the œsophageal or pharyngeal branches, to the cardiac, or even to the gastric branches, and thus adductor spasm has been described in association with foreign bodies in the pharynx and œsophagus, with enlarged tonsils, adenoids, and elongated uvula, with heart disease and with gastro-intestinal irritation.

The stimulation may reach the centres through other nerves, *e.g.*, through the fifth as in teething, through the spinal nerves as in diarrhœa, worms, etc., or even through the cutaneous nerve.

In the multitude of sources from which the irritation may come, laryngeal spasm presents a striking resemblance to epilepsy, as it does also in the almost necessary assumption of a peculiarly unstable condition of the nervous centres.

Direct irritation of one recurrent laryngeal nerve usually produces unilateral contraction of the corresponding muscles. Occasionally, however, it leads to bilateral contraction, *i.e.*, to adductor spasm. Of this, two explanations are given: (1) that the bilateral contraction is really a reflex spasm excited through coincident stimulation of centripetal fibres distributed to the trachea; or (2) that it is due to a powerful contraction of the arytenoideus muscle, the result of direct stimulation of the recurrent laryngeal nerve.

The attack of laryngeal spasm consists in a sudden paroxysm of dyspnœa of great severity but of short duration. The dyspnœa is chiefly inspiratory, inspiration being prolonged, laboured and attended by the characteristic crowing noise; while expiration is much less impeded, though there is, as a rule, some expiratory stridor as well.

Often, after a few respirations of this type, the breathing may cease for a few seconds altogether, the muscles of the chest and diaphragm becoming fixed and rigid. After a minute or at the most two, the spasm passes off as suddenly as it began, the relaxation of the spasm being referred to the accumulation of carbonic acid, while the growing deficiency of oxygen at the same time stimulates the

respiratory centre to violent action. If the spasm be not quickly relieved, the ordinary asphyxia convulsions set in, and the patient may die in, as it were, a fit. The paroxysm rarely lasts more than one or two minutes, but it may be repeated at short intervals, and thus the attack be apparently prolonged.

The paroxysms are liable to recur, but at quite irregular intervals, and are often excited by very trifling causes.

Laryngeal spasm occurs in both children and adults, but, in the former, it has an importance all its own and is described by a special name. It will be most convenient to deal with the commoner affection as it is met with in children first, and then to consider the conditions under which it is seen in adults.

SPASM OF THE LARYNX IN THE CHILD—LARYNGISMUS STRIDULUS.—The child, who has probably gone to bed in its usual health, wakes up suddenly in the night struggling for breath and making the characteristic crowing sound with inspiration. After a few such inspirations, the paroxysm subsides, the respirations become natural, and the child, after, it may be, crying a little from the fright, falls quietly asleep again. Such a slight paroxysm as this may constitute the whole attack, or it may be repeated several times in the same night.

In a severe paroxysm, after a few crowing inspirations the breathing suddenly stops altogether for a few seconds or a minute, the chest and diaphragm passing apparently into the same condition of spasm as the glottis. The child then becomes rapidly cyanosed, but almost immediately the cyanosis is succeeded by an ashy pallor, the eyes turn upwards, the head is thrown back, the back arched, carpopedal contractions occur, the child becomes unconscious, and passes urine and fæces involuntarily. Usually in this stage the spasm passes off, a few whistling respirations are taken, the colour returns, and the child comes to itself; but, if the paroxysm continues, the child may die in convulsions, as in asphyxia of any other origin.

Fortunately, very severe paroxysms are uncommon, and however alarming the attack may appear, a fatal result is rare except in infants; still it has happened that a young child subject to these attacks has been found dead in bed, presumably of an attack of unusual severity.

The attacks may come on by day as well as night, but Steffen doubts whether they are really so much more common at night, as is usually stated. General convulsions occur in about one-third of the cases, and tetany, or carpopedal contraction, is still more frequent, and generally persists between the attacks.

The younger the child the more severe, as a rule, the attack, as well as the more dangerous, and it is among infants almost exclusively that a fatal result occurs. As the baby grows older, the attacks diminish in severity as well as in frequency, and ultimately pass off altogether.

The **pathology** of the affection is absolutely negative. Of the morbid changes described, most may be attributed to rickets, which, according to Steffen, is present in nine cases out of ten, while the rest of the morbid changes are probably rightly regarded as the result of the affection itself. Thus to rickets may be referred the large liver, large spleen, tumid abdomen, intestinal catarrh, anæmia, craniotabes, and perhaps much of the bronchitis and laryngitis described. When the attacks are very severe, or often repeated, lung changes may arise, of which emphysema is the chief, but occasionally collapse or broncho-pneumonia develop; the brain and meninges also may show evidence of congestion and even hæmorrhage, just as after epileptic attacks; but all these conditions are the result and not the cause of the affection; or if they have any

part in the production of an attack, it is only by acting as a source of peripheral irritation upon a nervous system peculiarly susceptible.

Catarrhal affections of the larynx or lungs in the same way may excite a spasm, but cannot cause it. The same may be also said of the swelling of the thymus, or of the bronchial and tracheal glands, to which the attacks were formerly attributed.

Ætiology.—Laryngismus stridulus is by no means a common affection, though probably hardly so rare as hospital statistics seem to show.

It is most frequent between the ages of four months and three years, and is rare before or after this period, although cases are recorded in infants a few days or weeks old, and in children at the ages of eight or even twelve years.

Thus out of Morell Mackenzie's 31 cases, 21 occurred between the 5th and 9th months. Of 48 cases recorded by Gee, 1 was met with at 6 months, 19 between the 6th and 12th month, 16 between the 12th and 18th month, and 12 between the 18th month and three years. Out of West's¹ 37 cases, 31 occurred between the 6th and the 24th month. Steiner's larger statistics prove the same thing, for out of 226 cases 174 were under one year of age.

As regards sex, males are attacked more frequently than females in the proportion of two to one. The same preponderance of males is seen in some other nervous diseases, such as tetany and tubercular meningitis, and seems to point to a greater vulnerability of the nerve centres in male children.

The attacks are often excited by catarrh, and accordingly are commonest in the earlier months of the year, a fact which Gee attributes, in part at any rate, to the confinement within doors during the winter having reduced the general tone of the body and relatively increased the excitability of the nervous system in weakly children. In excitable children an attack may be brought on by violent crying or by other excitement, just as it sometimes is in hysterical adults. Romberg has also seen the condition arise on the sudden disappearance of a rash, and subside with its reappearance. Whether the affection is hereditary in the strict sense of the term is open to much doubt. It, however, frequently occurs in more than one member of the same family.

Thus Gerhardt records a family of nine, in which every child was attacked; Reid, one of thirteen in which only one escaped.

Its relation to rickets probably explains these curious facts.

Diagnosis.—Laryngismus stridulus is unlikely to be confused with any other affection, except perhaps the impaction of a foreign body in the larynx, and then the symptoms, though they may develop as rapidly, do not subside so quickly.

Acute catarrhal or membranous laryngitis, œdema of the glottis, abductor paralysis, or even whooping cough have all been mentioned as possibly giving rise to difficulties of diagnosis, but they are all alike distinguished from laryngismus stridulus by not developing and subsiding in the same abrupt way.

Very young infants occasionally give a crow on inspiration. This is of no significance and stands in no relation to laryngismus stridulus.

Prognosis.—The prognosis is, on the whole, good, and somewhat better, it is said, in girls than in boys. Unless the paroxysm be very severe or often repeated, or unless the child be very young or in a weak state of health or some complication arise, recovery is the rule. When death occurs it is almost invariably in very young infants. As the child grows older the attacks diminish in frequency and in severity, and finally cease.

¹ *Diseases of Children.*

Laryngismus stridulus is often associated with rickets, and then the more marked the rickets the worse the prognosis, for the ribs being soft, the recession of the lower parts of the thorax is considerable, and thus collapse of the lower parts of the lungs is more likely to occur. The want of resistance of the chest wall also explains why the attacks are more serious in infants.

Solis Cohen has observed the epiglottis drawn down by the aryteno-epiglottidean muscles and caught by the posterior wall of the pharynx, so as to lie like a lid over the glottis and cause fatal asphyxia.

Long-continued severe attacks, or slighter attacks often repeated, may lead to collapse of the lungs and gradual death in consequence, with or without bronchopneumonia.

Slight attacks may be frequently repeated, and Robertson¹ records a case in which spasm recurred every ten minutes, night and day, for a period of ten months. Eustace Smith records a similar case, which followed whooping-cough in a child of 20 months, though the paroxysms were less frequent (being only about two or three in the day).

Treatment.—The paroxysm itself is of such short duration as to give but little time for action; a hot sponge or cloth may be applied to the throat, or cold water douched over the head and neck. A little ammonia or smelling salts may be held to the nose. If the attack should be prolonged, a hot bath may be given and some chloroform inhalation administered with care. An emetic is often of great benefit, or vomiting may be excited by passing the finger into the fauces or tickling the throat with a feather; apomorphia also has been strongly recommended, but I think it is risky. If the breathing cease, artificial respiration should be resorted to at once, and may save life. Tracheotomy, however, is useless.

As the spasm is of such short duration the treatment mainly consists in combating, by appropriate means, the general defects of health upon which the paroxysms depend. Rickets should be dealt with in the usual way, *i.e.*, by good air, good food, cod-liver oil, and iron. The child should be sent as much as possible out into the fresh air, and may with advantage spend some time at the seaside. One of the best and safest remedies is the cold bath, given at a temperature of 65° F., even two or three times a day. Few cases resist this. If the weather be cold, the child may be allowed to sit in warm water and have the cold douche applied to the back. The general excitability of the nervous system may be allayed by the use of bromides, which may safely be given in 3 to 5 grain doses even to quite small children. Chloral in small doses at night is also found of service. A mixture containing 2 grains of chloral and 2 grains of bromide of ammonium may be given two or three times a day to a child one year old.

If the child be teething the swollen gums may be lanced; whatever other accidental symptoms may be present—such as dyspepsia, diarrhœa, worms—should be treated in the usual way.

As general antispasmodics, musk and castor were once favourite remedies, but have now gone out of fashion. In bad cases belladonna is often as beneficial as in whooping-cough, and is especially indicated when the spasm was originally started by that affection.

SPASM OF THE LARYNX IN THE ADULT.—Spasm of the larynx is a rare affection in the adult, and when it occurs the paroxysm is usually less severe than in children. It is most frequent in females about the time of puberty, and is usually associated with other signs of hysteria.

¹ Quoted by Eustace Smith, p. 283.

It is much more frequently symptomatic than in children, *i.e.*, it is more often a sign of some other grave affection.

Thus it may be associated with gross disease of the larynx, *e.g.*, ulceration or tumour.

More commonly still, it is due to some irritation in the course of the trunk of the pneumogastric nerve, especially its recurrent branch, and is thus met with in disease of the mediastinum, *e.g.*, thoracic aneurysm or tumour, in which affections it may be the cause of death.

It occurs also in the course of severe nerve-diseases; in tetanus and hydrophobia, it is a part of the disease, as it is also in an epileptic fit; it is met with occasionally, it is said, in chorea; in locomotor ataxy, it constitutes the so-called laryngeal crisis, and it has been described in the course of lead-poisoning.¹

Another rare but remarkable class of case is recorded, in which laryngeal spasm takes the place of the ordinary bronchial spasm of asthma.² In such cases the attacks may recur, like ordinary asthma, for many years. They are always severe and alarming, but are rarely fatal.

Lastly, laryngeal spasm may take the place of an attack of migraine (Liveing).

From what has preceded, it is clear that laryngeal spasm in the adult, though rare and usually less severe in itself, is a far graver affection, as indicating, in most cases, some serious and often irremediable primary disease.

The treatment of the paroxysm is much the same as in the child, except that stronger measures may be safely employed, and of these none is more useful than a subcutaneous injection of morphia. Chloral may be given in full doses, or cannabis indica administered at night.

I was once called to see a lady of 45 years of age who had taken a considerable quantity of tincture of belladonna with suicidal intent. On attempting to pass the stomach tube to wash the stomach out, a very severe attack of laryngeal spasm was excited, the patient became greatly cyanosed, and seemed in great danger of suffocation. The spasm, however, soon passed off, but it was thought too risky to attempt to pass the tube again. I was then informed that she had been subject all her life to attacks of the same kind whenever the throat was irritated in any way, or under conditions of excitement. She was not epileptic nor hysterical, but, on the contrary, was of a remarkably placid and phlegmatic temperament.

History and Synonyms.—The disease was described first by Richa in 1723 and by Verdries in 1726, and connected by them with a swelling of the thymus, but it was not till Millar's work, in 1769, that it became established as a well-recognised affection. Millar described it as asthma, and it was for some time called after him by the name *Asthma Millari*. The various names which the affection has borne may be grouped together so as to indicate the different theories that have been held as to its nature. The grouping of the synonyms in this way, of course, only approximately corresponds with the sequence of the theories in time.

Thus, for some time after Millar wrote, the affection bore the general name of asthma, to which various affixes were added to draw attention to different features in it. Hence the names *Asthma puerorum. s. infantile* (Boerhaave); *A. dentientium* (Pagenstecher); *A. thymicum* (Kopp); *A. thymo-cyanoticum* (Kussmaul).

Another group indicated the prominent symptoms without suggesting any theory. Of these were *Child-crowing* (Gooch); *Crowing inspiration* (Hood); *Apnoea infantum* (Rösch).

Other terms again indicate the seat of the affection, *e.g.*, *Angina stridulosa* (Bretonneau); *Spasm of the Glottis* (West); *Phreno-glottismus* (Bouchut); *Laryngismus stridulus* (Mason Good), the name which it now seems likely to bear for the future.

When croup became a recognised affection, terms were required to distinguish this affection from croup, and it was called *False or Pseudo-Croup* or *Spasmodic-croup*. The latter name is still sometimes applied to it. Of later years its relation to affections of the nervous system have been more prominently brought forward. It was thus styled *Cerebral Croup* (Pretty, John Clarke). Its resemblance to fits in children suggested the names *Internal Convulsions* (Barthez and Rilliet) or *Suffocative Convulsion* (Mackenzie). The more recent writers now appear to regard it as simply a form of epilepsy and due to the same causes.

¹ Steffen, *loc. cit.*

² Gowers, *Dis. of Nerv. Syst.*, ii. 266.

Theories.—Many of the theories indicated by these names are no longer tenable. The present usage of the term "asthma" now renders it unsuitable. The swelling of the thymus upon which stress was laid early is more often absent than present. The laryngoscope has shown that laryngitis, membranous or not, has nothing directly to do with the affection, so that almost the only question which remains for discussion is, whether laryngismus stridulus is really epileptic or not.

The facts established are—

1. That it is a neurosis, *i.e.*, that it has no known morbid anatomy;
2. That it is a spasm of central origin and generally reflex;
3. That it is frequent in children and rare in adults;
4. That it is necessary to assume, at any rate in children, some hyper-excitability of the nervous centres;
5. That it resembles the epileptic seizure in its sudden onset and short duration, in its liability to recur, and in its occurrence so often at night.

Strong as the resemblance to epilepsy often is, and epileptic in nature as some of the attacks may be, still, before the theory can be accepted that laryngismus stridulus is nothing but epilepsy, one or two difficulties must be removed. The difficulties are these: (1) that in undoubted convulsions or epilepsy laryngismus stridulus is rare; (2) that it is only in a few cases that actual convulsions occur, and they may then be due to the asphyxia; (3) that it is the exception and not the rule to find, in a child subject to convulsions, an attack of laryngismus taking the place of an ordinary fit and *vice versa*; (4) that children with laryngismus stridulus do not commonly become epileptic; and lastly, that epilepsy will hardly explain the cases of laryngeal spasm in the adult, and we should be then forced to assume that we have in adults a different affection to deal with.

All that we can say at present appears to be that laryngismus stridulus is, in many respects, closely related to epilepsy, but that it is not yet proved to be the same thing.

Eustace Smith, Wilks, and others have recorded instances in the same family in which one child suffered from laryngismus stridulus and another from general convulsions, and in the same child attacks of laryngismus stridulus alternated with general convulsions or was associated with them. As already stated, however, such an experience is the exception and not the rule.

11. CONGENITAL INFANTILE STRIDOR.

Infantile Laryngeal Spasm—Congenital Laryngeal Stridor— Respiratory Croaking in Babies.

Under these varying names is described a peculiar stridor, developing at or immediately after birth, persisting for the first year or two of life and then gradually disappearing.

The condition is not really rare, though little reference has been made to it in literature until recent years.

The noise is a kind of croak, usually attending inspiration only, but sometimes present with expiration also.

In the common form inspiration begins with a croaking noise; expiration is accompanied by a short croak if the inspiratory croak be loud, but at other times is noiseless.

The croaking varies in character, being at times deep and growling, at other times high-pitched, clear, and squeaking. It also varies in loudness at different

times, and there may be periods of complete intermission. It is often absent when the child is breathing quietly and also during sleep, but it reappears at once if the respiration be accelerated or deepened, and is easily brought on by exertion or excitement.

The cry and cough are clear and natural.

The obstruction that exists to the entrance of the air into the chest is shown by the inspiratory retraction of the lower ribs and of the intercostal spaces and soft parts of the chest. The amount of retraction varies directly with the amount of stridor, *i.e.*, with the noise made; it is rarely absent entirely, even at the times when the stridor is absent.

If the stridor be pronounced and long-continued, the shape of the chest may become altered, as with rickets.

There is rarely any amount of cyanosis unless the stridor be extreme, but attacks of an almost suffocative character have been described as coming on suddenly, especially after sleep.

The stridor is, as a rule, observed immediately after birth, but sometimes does not become pronounced enough to attract much attention for a few days. If not present during the first two weeks it rarely develops later.

Except for the stridor and for any effect that the inspiratory obstruction may directly produce upon the shape of the chest, the child is usually well nourished and in good health.

The stridor cannot be connected with any constitutional defect. The association with rickets, syphilis, or anæmia is accidental only, but these conditions would increase the tendency, which the obstruction naturally creates, to bronchitis or collapse of the lung.

In most cases the children appear to be little inconvenienced by the stridor, and continue in good health throughout.

The stridor observed at or soon after birth may continue to increase in loudness for the first two weeks, owing probably to the increased vigour of breathing as the child grows stronger. It may then continue more or less unchanged until the ninth or tenth month, and then gradually subside, until by the end of the second year it has entirely disappeared, or at the most is only occasionally present, being brought on by some excitement, exertion, or cold.

The prognosis is favourable in all ordinary cases. The affection gradually subsides without in any way prejudicing the health, until it disappears, as stated, by the end of the second year or earlier.

Where the obstruction is considerable, especially if the child be in bad health or the subject of syphilis or rickets, sundry complications may arise and thus carry the child off, but in the absence of such complications there is little or no risk to life.

DIAGNOSIS is easy, for the croaking is unlike any other form of stridor. It differs from laryngismus stridulus in that it is congenital; from laryngitis in the clearness of the voice and cry.

The symptoms resemble most closely those of abductor paralysis; thus there is the same inspiratory obstruction and stridor, while expiration is as a rule unimpeded, and the voice remains clear and unaffected; but it differs from abductor paralysis in that recovery is complete.

Most writers on the subject refer the symptoms to laryngeal spasm, and yet distinguish it from laryngismus stridulus. But such prolonged, recurrent, and in some cases constant spasm is practically unknown in that affection, and so renders some other explanation more probable. Thus it has been referred to an

undue laxity of the parts about the glottis, which are thus likely to get sucked in and over the glottis during inspiration.

The exact nature of the condition can only be determined by laryngoscopic examination or by *post-mortem* evidence.

Laryngoscopic examination in infants is a difficult matter, but by pressing down the tongue with the finger or a spatula, and pulling it forward, Dr. Sutherland¹ was able to examine the larynx in six cases. When a good view of the vocal cords could be obtained, they were seen to be normal and the approximation accurate. The following is a description of what was found, which is in exact accord with what was described by Dr. Lees.

"The epiglottis was sharply folded upon itself, and the two lateral folds lay in close approximation and sometimes in contact. The aryteno-epiglottic folds were approximated so that the upper aperture of the larynx was reduced to a long narrow slit. The folds bounding the aperture seemed quite flaccid, and flapped to and fro on respiration. The inspiratory column of air striking down on these folds drove them together, and, on expiration, they again separated. In one case in which there was a purring stridor the coarse vibrations of the folds could be distinctly seen."

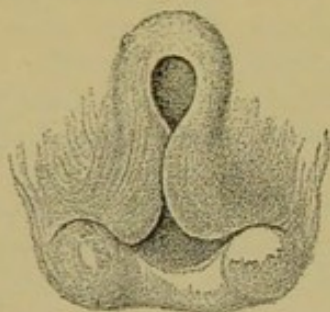


Fig. 19.

Diagram of the glottis as seen with the laryngoscope. The epiglottis is folded and leaves only the two spaces, shaded dark, through which air could enter the larynx.

Mr. Berry has recently had a case under his care in a female child of 13 months old.

The mother noticed the croak at birth. It had continued much the same, being, however, worse if the child had a cold. The child had been under several doctors, and a severe operation had been suggested, but the child appearing otherwise well, it was declined by the parents. The interest lies in the laryngoscopic examination, which was made by Mr. Berry, under chloroform. The epiglottis was then found to be lying over the glottis laterally folded, its sides being curled over so that the left overlapped the right, and instead of the normal opening of the glottis two small openings only were seen, a small anterior chink and a larger posterior opening. When the epiglottis was lifted with the finger the inspiratory croak ceased, and returned again as soon as the epiglottis was dropped.

Post-mortem evidence is hitherto scanty, for these children do not often die. The condition after death has been recorded now in three cases, by Lees,² Refslund,³ and Variot.⁴

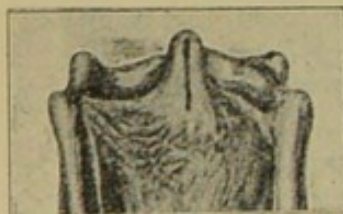


Fig. 20.

Lees' case of infantile stridor, child of one year; death from diphtheria.



Fig. 21.

Larynx of new-born child during experiment.

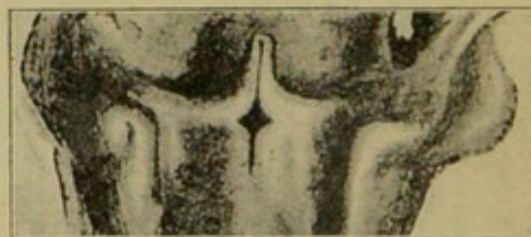


Fig. 22.

Refslund's case of infantile stridor, child of 2½ months. Death from pneumonia.

(Figures reproduced by permission of Drs. Thomson and Turner.)

In all cases the epiglottis was folded on itself in such a way as to leave but a narrow chink through which the air could pass into the larynx. Thomson and

¹ Sutherland and Lack, *Lancet*, 1897, Sept. 11.

² *Path. Soc. Trans.*, xxxiv. 19.

³ *Munch. Med. Woch.*, 1896, No. 48.

⁴ *Journal de Clin. et de Thérapie infant.*, 1896, June 18, and 1898, June 9.

Turner¹ have produced a closely similar condition in dead infants experimentally (fig. 21).

The causation of infantile stridor may now be considered as conclusively demonstrated.

Treatment.—Treatment is not required in most cases, for beyond the croaking, which seems to cause the child no inconvenience, there are rarely any symptoms, and in a few months the child is well.

Care, however, should be taken, especially in weakly children, that they do not get cold, which by fastening on the larynx or lungs would introduce complications and involve special risks.

In the rare cases in which the obstruction is severe, and suffocation seems to threaten, intubation would appear to be a more rational procedure than tracheotomy, but I do not know of any instance in which either the one or the other has been found necessary and performed.

12. ABDUCTOR PARALYSIS.

And other Conditions causing Median Position of the Vocal Cords.

If the movements of the vocal cords be observed, it is found that, on deep inspiration, they are strongly abducted so as to open the glottis to its widest extent, while, on phonation, they are as strongly adducted until they all but meet in the middle line. Both of these positions are due to the active contraction of the appropriate group of muscles.

If the larynx be examined when all the muscles are paralysed, all movements are absent, and the cords are found in a middle position between the two extremes. This, being the position in which the cords are found in the dead body, has been called the *cadaveric position*. It represents the position of equilibrium or rest.

If one of these groups of muscles be paralysed, the corresponding cord will not be able to move in that direction beyond the cadaveric position, but, if the paralysis continue, the antagonist muscles, being unopposed, will draw the cords in their own direction, producing what may be fairly called paralytic deformity, and it is even probable that these muscles may actually contract, as they do under similar conditions in the extremities. If the adductors are paralysed, the cord is found in the cadaveric position or a little outside it. Such a lesion is attended with loss of voice if it be bilateral, but with no symptoms at all if it be unilateral only.

The result is very different when the abductor is paralysed. The first effect of abductor paralysis would be that the cord would assume the cadaveric position, and that the respiratory dilatation would be lost. Very quickly, however, the corresponding cord is, by the unopposed action of the adductors, drawn to the middle line and fixed there. If this happened on one side only, there might be no symptoms at all, for phonation would not suffer, and dyspnoea if it arose at all would be experienced slightly only on exertion. If, however, the affec-

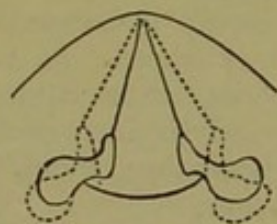


Fig. 23.

Diagram showing the position of the vocal cords on ordinary inspiration and on deep inspiration (dotted line). The former is also approximately the *cadaveric position*.

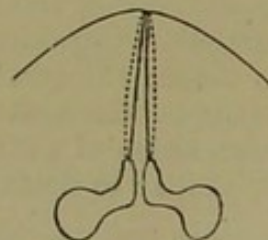


Fig. 24.

Diagram showing the position of the vocal cords in abductor paralysis, the continuous line on inspiration, the dotted line on expiration.

¹ *Brit. Med. Jour.*, Dec. 1, 1900.

tion were bilateral, severe respiratory difficulty would necessarily arise at once, for the vocal cords, being on both sides fixed in the middle line, nothing but a narrow chink would be left between them through which the air could pass, and the necessary result would be great dyspnœa.

The symptoms are peculiar and characteristic. The dyspnœa is extreme and almost entirely inspiratory. Inspiration is, indeed, attended with a long-drawn whistling or crowing sound, which may be often heard some distance off. Expiration, on the other hand, is comparatively free. If the cords be watched it is seen that the narrow chink between them is still further diminished on inspiration, as if the air forced them closer together, while on expiration they are separated somewhat, so that the air passes out without much difficulty. The voice is maintained, and, on phonation, the cords become tense, just as under normal conditions. Over the chest the vesicular murmur is drowned by the stridor. In the absence of complications there are no physical signs in the chest, no cough, no expectoration, and no fever. Cyanosis may be present, but its degree will depend upon the amount of obstruction. Owing to the absence of symptoms at first, the early stages have been but rarely observed. In the few cases recorded,¹ the sequence of events has been that already described, *i.e.*, the cords at first assumed the cadaveric position, but soon were drawn by the unopposed action of the adductors into the middle line. How long the interval usually is between these two stages is not known, but it may, apparently, be very short. In two cases a further stage still has been observed, where the cords, after having been some time in approximation, passed again, as the paralysis increased, into the cadaveric position. Contracture, *i.e.*, permanent shortening, though very probable, has not yet been actually demonstrated to occur.

The dyspnœa is liable to sudden exacerbations, the paroxysms being brought on by exertion, mental excitement, or, accidentally, by some secretion which cannot be readily expelled.

This peculiar association of symptoms, *viz.*, severe inspiratory dyspnœa, with complete retention of voice, is characteristic, and pathognomonic, not indeed of abductor paralysis only, but of that position of the cords to which adductor paralysis leads. This position may be the result of other lesions.

The conditions under which the cords may be brought into approximation to each other, and there fixed, may be arranged in two groups—the one neuromuscular, depending upon changes in the abductor muscles or the nerve-supply to them; the other mechanical and independent of either muscles or nerves—for example, the arytenoid cartilages may be fixed in close approximation by the contraction of a scar appropriately placed, or by changes in the joint or round it, similar to those which would produce a stiff joint elsewhere.

The possible causes to which the condition of the larynx under consideration may be due are then as follows :—

- I. Mechanical.
 - a. *cicatricial*.
 - b. *arthritic or peri-arthritic*, from
 - i. *extra-articular bands*,
 - ii. *intra-articular adhesions*,
 - iii. *dislocation, or other injury*,
 - iv. *disuse*;

¹ *Berl. klin. Woch.*, 1872, Nos. 20, 21; 1873, No. 7. (Feith), *do.*, 1874, No. 49.

- II. Neuro-muscular. *c. myopathic,*
d. neuropathic,
 i. *central, in cord or brain,*
 ii. *peripheral, in trunk or nerve endings.*

a. Cicatricial.—A case of this kind is recorded by Sidlo,¹ in which a scar was found extending across the posterior wall of the larynx, drawing the arytenoids together and fixing them in that position. Such a scar would be extremely difficult, if not impossible, to diagnose during life by the laryngoscope.

b. Arthritic and peri-arthritic.—The joints may be fixed either by adhesions within or by fibrous bands around.² Primary synovitis of the arytenoid joint is a possible lesion, but must be very rare; it has been described as due to rheumatism, gout, or even catarrh.³

In the majority of cases the changes in or around the joint are the result of inflammatory processes near it, especially if associated with perichondritis, but in most cases the perichondritis must be one that admits of cicatrization, and cases of this kind are hardly ever met with except in the course of syphilis and perhaps after typhoid fever.

Mere infiltration around, even when as extreme as it sometimes seems in syphilis or tubercle, is rarely sufficient to fix, though it may somewhat limit, the movement of the joint. Kidd⁴ records an instance which followed tubercular infiltration, and Watson Williams one that followed diphtheria.

Where the joint has been long fixed as the result of paralysis or disease in the neighbourhood, the stiffness which has ensued has been referred to disuse.⁵ The most interesting cases of this kind occur in young children after tracheotomy.

Lastly, a few cases have been attributed to injury, and Semon has recorded a case of dislocation in which it was present.⁶

c. Myopathic.—The myopathic form is very rare. A case was described in 1866 by Hughlings Jackson and Morell Mackenzie, and another by Riegel in 1872. This form has been attributed to cold, to over-exertion, to injury, to syphilitic affections of the muscle (probably gummatous), and to pressure. It is evident that the diagnosis of this form is attended with the greatest difficulties, and some of the myopathic cases may, as Gowers suggests, be, after all, of central origin. Even when the muscle is primarily at fault it may be still a question whether the lesion is one of the muscle fibres or of the nerve endings, as in a case of this kind attributed to lead poisoning.

d. Neuropathic.—It is about the neuropathic form that the interest centres on account of the difficulty of explaining its development satisfactorily.

In man, irritation of the Recurrent nerve produces closure of the glottis, that is adductor spasm, while complete paralysis of the nerve leaves the glottis in the cadaveric position. Many of the cases of abductor paralysis are found associated with pressure on the Recurrent laryngeal nerve. Now, although irritation of the Recurrent produces spasm of the glottis,⁷ the condition we are dealing with cannot be adductor spasm because of its gradual development and long persistence. The question then arises—How is it that partial paralysis of the Recurrent produces paralysis of the abductor only? Many suggestions have been offered in explanation.

1. That the abductors are supplied by the Recurrent only, while the adductors receive a supply as well from the Superior Laryngeal. This theory is not supported by anatomical observations (Exuls).

¹ *Wien med. Woch.*, 1875, No. 26.

² Semon, *St. Thomas' Hosp. Rep.*, 1883, p. 107.

³ Morell Mackenzie, *loc. cit.*

⁴ *Lancet*, 29th March 1890.

⁵ Solis Cohen, *Amer. Journ. of Med. Sc.*, 1883-84.

⁶ *M. T. and G.*, 1880.

⁷ Penzold, *D. Arch. f. klin. Med.*, xv. 604, records a case in which pressure over a goitre produced approximation of cords and dyspnoea, *i.e.*, adductor spasm.

2. That the abductors suffer most in paralysis, just as in a paralysed limb the abductors and extensors suffer more than the adductors and flexors. This is, however, but a doubtful analogy and no explanation (Rosenbach).

3. That the abductor fibres are external in the nerve, and therefore suffer first. This theory is also unsupported by facts.

4. That the tone of the abductor depends upon reflex action through the Recurrent, and that this reflex is the first to suffer. This is mere speculation (Zederbaum).

5. That as there are five adductors and only one abductor, the same amount of interference in the nerve supply might produce much loss of power in the single abductor and but little in the group of adductors (Müller. Bichât).

6. That there is a difference in the anatomical structure of the two sets of muscles, the abductors corresponding with the red muscles of Krause and being supplied with longer and more delicate nerves and fewer end plates, while the adductors are pale muscles and have shorter, thicker, and more abundant nerves, as well as more numerous end plates (Simanowski).

These statements are confirmed by two sets of observations.

1. The effect of ether, which has a differential effect upon the laryngeal muscles (Hooper).

2. The effects of freezing the recurrent laryngeal nerve, which show that the abductors die before the adductors (Fränkel and Gad).

It is probable that there is also similar differentiation in the nerve centres (Semon), seeing that in bulbar affections the abductor-nuclei succumb before the adductor.

Even here the difficulties do not end, for abductor paralysis is generally bilateral, whereas one recurrent only often appears to be involved.

In most cases of bilateral abductor paralysis we must assume, however, either a central lesion, or that both nerves are involved. However this may be, a few cases are recorded in which bilateral paralysis has occurred with a lesion affecting only one nerve (Whipham, Bäumlér, Johnson and Sommerbrodt).¹ This can only be explained, as Johnson suggests, as the result of a centripetal irritation acting on the nerve centre. Cases of abductor paralysis due to cerebral lesions are recorded by Mackenzie,² Penzoldt,³ and Semon⁴; but the evidence in some of them does not seem very strong, though the experiments of Semon and Horsley⁵ show that cortical stimulation near the speech centre will produce a bilateral adduction of the vocal cords.

Ætiology.—Of the neuromuscular forms the cause in most cases is quite obscure. Sometimes there is some obvious source of pressure on the recurrent nerve either in the mediastinum, as the result of thoracic aneurysm, enlarged glands, or new growth,⁶ or in the neck, as with goitre, enlarged glands, or malignant disease of the œsophagus or the thyroid. The paralysis is then usually unilateral, though, as stated, it may be sometimes bilateral. When unilateral it may be easily overlooked, for it would produce no symptoms, and Semon⁷ believes that it is a much more common affection than is usually supposed.

The bilateral affection has been met with after diphtheria, erysipelas, catarrhal laryngitis and lead-poisoning. Some of these may be nervous and some muscular in origin. It occurs also in the course of bulbar lesions, and in the later stages of locomotor ataxy. It is described also in hysteria, and it is possible that some of the cases described as long-continued spasm may really be hysterical abductor paralysis.

Abductor paralysis is nearly twice as frequent in males as in females. The great majority of recorded cases have occurred in adults, but a small and very interesting group of cases is met with in children after tracheotomy. In them, probably as the result of disuse, the abductors do not attempt to act on inspiration, and the glottis remains closed, so that the tube cannot be dispensed with.

In the case, recently under my observation, of an infant who had had tracheotomy performed for a fish-bone in the larynx, all that was necessary was the passage of a catheter through the glottis on one occasion for the respiration to become normal, and the tube to be dispensed with.

Duration and Course.—Except in hysterical cases and a very few others the condition is a permanent one, and recovery is out of the question.

¹ Gowers, *Dis. of Nerv. Syst.*, ii, 263.

² *Loc. cit.*

³ *Deut. Arch. f. klin. Med.*, xiii, 111.

⁴ *Clin. Soc. Trans.*, xv.

⁵ *Trans. Royal Soc.*, 1890.

⁶ Bäumlér records a unique case in which the cause of pressure was a large pericardial effusion.

⁷ *Berl. klin. Woch.*, 1883, Nos. 40-49.

Tracheotomy has to be performed sooner or later, and the tube worn for the rest of life. Life is not, however, necessarily shortened, and Gerhard¹ records a case in which the tube was worn for twenty-eight years.

Assagnée² describes a case in which the affection developed during pregnancy and required tracheotomy, but the patient made a complete recovery after delivery. Bosworth states that where abductor paralysis has continued for more than nine months, return of mobility is impossible, but where the conditions develop rapidly cure may occur. Mackenzie records a case in which a patient wore a tube for twenty-four years, and died of another disease.

The following is an interesting case of mixed paralysis and spasm of the muscles of the larynx associated with spasm of the diaphragm in a hysterical patient.

W., aged 38, had been out of health for the twelve months following an attack of influenza. She had chiefly suffered with palpitation of the heart, and latterly had some difficulty with her breathing. The only fact of importance in her previous history was that, at the age of 23, she had a contraction of the muscles of the neck and throat, so that she was unable to open her mouth. Some teeth were drawn, and she was fed for some weeks by means of a tube inserted through the opening. She was a well-developed, healthy-looking woman, with nothing especially to note about her, except that she was breathing in a laboured and hurried way. All her organs appeared to be healthy.

The respirations numbered 36 in the minute, but during sleep they were quiet and slower. The movements of the chest were peculiar; the diaphragm was apparently almost fixed in the inspiratory position, and did not move at all with inspiration; the movements of respiration were entirely conducted by the intercostal muscles, and were not unusually free; a little wheezing was audible over the chest, but nothing more; inspiration was noisy, but expiration easy. The noise was evidently laryngeal in origin.

Examination with the laryngoscope showed that the vocal cords, during inspiration, were in close approximation, leaving a very narrow chink between them. On expiration they separated somewhat, but not so far as to assume the cadaveric position; the voice was slightly hoarse, but there was no laryngitis.

The movements of the soft palate and fauces were extreme during expiration, the palate rising almost as high as possible during expiration, and sinking during inspiration. Although there was a good deal of obstruction on inspiration there was no excursion of the larynx.

The condition remained unchanged for the next ten days, and then, on examination, it was found that, although the anterior part of the vocal cords came into approximation as before, the arytenoids did not move. This suggested a loss of action in the arytenoidei posticus; but on phonation the arytenoids approximated normally.

The next day the movements of the diaphragm and of the palate were normal, and so were the movements of the larynx; that is to say, the approximation of the cords had passed off. But the movements of the cords were still uncertain; they sometimes receded normally as they should on inspiration; at other times they wavered, as if uncertain what to do, and at times remained in varying positions of partial abduction.

A week later all the symptoms returned again, except that the movements of the palate were not so exaggerated as at the time of admission.

Three days later the symptoms had improved, and a week later I took the following note. "The diaphragm is moving naturally and so is the palate; the vocal cords seem to be uncertain, moving constantly with ordinary respirations, so that in no two consecutive respirations do they occupy the same position. In the morning before my visit the cords had been closely approximated, but after the battery was applied this passed off, and now the cords remain always some distance from the middle line."

The interrupted current was frequently applied both to the abdomen and also to the throat, and each application caused improvement. In the course of some weeks all the symptoms passed off entirely, and the patient was discharged well.

The curious admixture in this case of paralysis of one set of muscles and spasm of the other is remarkable.

Diagnosis.—The symptoms are very striking, viz., great inspiratory dyspnoea with perfect retention of voice. Such a peculiar combination of laryngeal symptoms points at once to the cause. Other combinations of symptoms are similarly suggestive of other forms of laryngeal affection. Thus loss of voice as well as of the power of coughing with or without dyspnoea indicate grave organic disease of the larynx; loss of the power of coughing

¹ Riegel, *N. Syd. Soc.*, 1877, p. 309.

² *L'Union Medic.*, 1885, 31st March.

without loss of voice, paralysis of one vocal cord ; and loss of voice without loss of the power of coughing, unimportant adductor paralysis, probably hysterical.

Treatment.—The most important part of the treatment consists in tracheotomy. As long as the dyspnoea continues the patient's life is in constant danger ; a little mucus or a slight laryngitis suffices to produce the most urgent symptoms or even death, so that tracheotomy becomes absolutely necessary as a measure of precaution and should be performed as soon as possible after the diagnosis has been made. After tracheotomy, strychnia and electricity are often found useful, or the general remedies suggested by the condition or previous history of the patient may be employed. In hysterical cases tracheotomy will hardly become necessary ; besides general treatment the larynx should be galvanised, and the cases in children which follow tracheotomy may be sometimes cured rapidly by the passage of a catheter or probe, as in the case quoted.

History.—In the way of history there is but little to record. The first case was described by Gerhardt in the year 1862. In 1876 Ziemssen knew of only nine published cases, but Gordon Holmes in 1887 collected records of sixty. The chief advances made have been in the distinction between the neuromuscular and the mechanical group and the differentiation of the former into the central and peripheral forms. The first myopathic case was recorded by Mackenzie and Hughlings Jackson in 1866. For many of the recent advances we are indebted to Semon.

13. PERICHONDritis, NECROSIS, AND CARIES OF CARTILAGE.

In the neighbourhood of chronic inflammatory lesions the cartilages may calcify, and, if ulceration subsequently reach cartilages so altered, caries and gradual erosion may occur.

When the inflammation is more active, and especially if there be deep ulceration, the perichondrium is likely to become involved and suppuration to take place beneath it. It thus becomes completely separated from the cartilage, which necroses and lies as a dead mass in an abscess cavity, the walls of which are formed by the distended and thickened perichondrium.

The abscess sooner or later bursts either into the air-passages or into the surrounding tissues ; the former result is the rule, but in the latter case air may pass with the pus, and emphysema, which may extend widely, be produced. The emphysematous tissues are not unlikely to suppurate and a diffuse phlegmonous inflammation be the result.

When rupture has taken place a chronic discharging abscess will be left, which will continue as long as the dead cartilage or any portion of it remains. The necrosed cartilage is generally removed by gradual disintegration, but it is occasionally discharged into the air-tubes *en masse*. When so discharged it may be expectorated, but there is always the risk that it may become impacted and produce a dangerous or fatal paroxysm of dyspnoea, just as might occur with any other foreign body.

When the dead cartilage has been completely removed in either of these ways the possibility of healing will depend upon the original disease, with which the necrosis has been associated, and so it comes that there is practically little prospect of cure, except in cases of syphilitic origin ; and even then healing will be attended with so much cicatrization and contraction, that serious and permanent stenosis is likely to be produced.

It is stated¹ that necrosis of the cartilages of the larynx and the air-tubes, like necrosis of bone, may sometimes be dry, *i.e.*, attended with the production of little or no pus; if so it must be even still rarer than that very rare affection, dry necrosis of bone.²

The pathological processes are essentially the same in all parts of the air-passages, but they differ in gravity according to the part affected, and, as will be anticipated, their importance is greatest when the cartilages of the larynx are involved.

I. IN THE LARYNX.—In the great majority of cases the perichondritis is secondary, *i.e.*, is the result of ulceration near the cartilages affected, the ulceration being due to the common causes, viz., tubercle, syphilis, and carcinoma.

Bosworth, excluding tubercle and cancer, found in 33 cases of perichondritis the causes to be as follows:—

Idiopathic (no obvious cause)	9	Diphtheria	1
Syphilis	9	Traumatic	1
Typhoid Fever	11	Specific disease in neck	2

Ulceration is, however, not a necessary antecedent, for perichondritis may occur in the infiltration stage of these affections, before any ulceration has occurred.

The same may be said of the Specific Fevers. In some the perichondritis follows inflammation or ulceration in the larynx, secondary to destructive or suppurative inflammation in the pharynx, as in scarlet fever, small-pox and diphtheria.

In others pharyngeal lesions are absent, and the perichondritis is not connected with any obvious primary lesion in the larynx.

This occurs especially in typhoid fever, in the course of which necrosis of some of the cartilages of the larynx or of the epiglottis is not altogether rare.

Lüning³ collected 55 cases of perichondritis after typhoid fever and found the various cartilages affected in the following numbers:—

Cricoid	22	Cricoid and arytenoid	14
Arytenoid	9	Cricoid and thyroid	5
Thyroid	2	Cricoid, thyroid, and arytenoid	3

In other words, the cricoid alone or with others in 39; the arytenoid alone or with others in 26; the thyroid alone or with others in 10.

In typhoid fever this lesion is usually described as commencing as a perichondrial infiltration, which is held to be of the same nature, to run the same course, and to break down in the same way as the infiltration in the intestines. Bacteriological investigation has recently thrown doubt upon the identity of the two processes, for, in many cases at any rate, the typhoid bacillus has not been discovered, but some other bacillus, commonly a streptococcus.

It is possible that the perichondritis is not primary in the strict sense of the term, but is the result of previous ulceration near to the affected part. Laryngeal ulceration is a much commoner lesion in the course of typhoid fever than is usually supposed.

Thus Kanthack⁴ has shown that in fatal cases of typhoid fever ulcerations of some part of the larynx are found in no less than 26 per cent. Of all cases, *i.e.*, including the non-fatal cases, the frequency, as determined by laryngological examination, has been placed by Schrötter at 30 per cent.

In other cases of typhoid, perichondritis and necrosis have been observed to occur without marked infiltration. This form has been regarded as a kind of

¹ Schrötter, *Wien. med. Woch.*, 1888, Nos. 2 and 3.

³ *Arch. f. kl. Chir.*, xxx. p. 225.

² Holmishead, *Lancet*, 1888, i. 112.

⁴ *St. Barthol. Hosp. Rep.*, 1896.

decubitus, and referred to pressure of the laryngeal cartilages against the spine in feeble patients who have been lying long upon their backs in bed.

Similar necrosis (*decubitus-necrosis of Dittrich*) has been recorded in protracted cases of small-pox and in glanders (Eve). I have met with a similar condition myself in a case of chronic renal disease.

In all the foregoing forms the perichondritis is usually described as secondary.

Whether perichondritis is ever primary, *i.e.*, whether it ever arises as an idiopathic affection, independently of any of the causes mentioned, is a question which has led to much discussion. Some authorities, like Heinze, deny it altogether; Morell Mackenzie observed it in the course of catarrhal laryngitis; Ziemssen also records two cases; and a few have been recorded recently. The difficulty is chiefly one of diagnosis, for it is often impossible to be sure during life that all the causes mentioned have been positively excluded, and many a case which has been diagnosed as primary perichondritis proves subsequently to be secondary.

Inflammation need not necessarily proceed to suppuration, but may cause chronic changes resulting in cicatricial lesions; these almost invariably spread to the neighbouring joint, where adhesions are produced which may limit movement or even fix the joint entirely.

Where the arytenoid and crico-arytenoid joints are involved the cords often assume a position of close approximation. The cases thus come to resemble abductor-paralysis, from which it may be very difficult sometimes to distinguish them.

Perichondritis, as already stated, is usually found in connection with tubercle, syphilis, or cancer.

Of 44 cases analysed by Morell Mackenzie, tubercular disease was present in 19, cancer in 10, syphilis in 6, typhoid fever in 4, chronic laryngitis in 2, and 3 cases were thought to be primary. Retslag found tubercle in 10 cases out of 20, but of the rest no less than 8 were due to typhoid fever.

The frequency with which the different laryngeal cartilages are affected does not seem to have been exactly determined yet.

In Retslag's¹ 20 cases the cricoid was affected in 11, the thyroid in 3, the cricoid and epiglottis in 2, the cricoid and arytenoid in 1, and the thyroid and arytenoid in 1. My own observation would have led me to think with Schrötter, that the arytenoid was much more frequently attacked than these figures show. Of 33 cases collected by Bosworth, the cricoid was involved in 23, the thyroid in 3, the arytenoid in 4, the cricoid and thyroid in 1, and all the cartilages together in 2.

Symptoms.—The symptoms of perichondritis are not characteristic. We should expect that the inflammation would cause local pain and tenderness, and the swelling produce dyspnoea; these are the common symptoms, but they may sometimes be absent, and, when present, may be due to other causes, so that they are not conclusive. The laryngoscope provides the only means of positive diagnosis, and even this is not always adequate.

When perichondritis develops in the course of chronic laryngeal disease, its access is often indicated by the development of local pain and tenderness which had not been present previously, by fresh swelling in the seat of one of the cartilages, and, when the abscess has burst, by an increase in the amount of laryngeal discharge.

¹ *Wien. klin. Woch.*, 1888, p. 25.

Pronounced dyspnœa is not a necessary symptom, but an accident, and the result of various causes. It may be due:—

1. To the inflammatory swelling, *i.e.*, to the abscess itself and the œdema round it.

If so it will be relieved at once when the abscess bursts or is incised. The reason that the signs of abscess are not usually more marked is that the majority of the cases occur in the course of ulceration, so that the discharge takes place quite early through the base of the ulcer, and little tension is developed.

2. To immobility and fixation of one or both cords in the middle line.

This may be produced by the defective action of the abductors (*postici*) owing to their loss of insertion into the cartilages, the cricoid on the one hand and the arytenoid on the other, but it may be also the result of chronic inflammation in, or more commonly round, the crico-arytenoid joints, which have thus become fixed in a position of adduction.

3. Where the necrosis is extreme the larynx may so far lose its stiffness as to collapse. This is a rare occurrence¹; and it is certainly remarkable how extreme the destruction of cartilage may be without collapse occurring, the air-passages being kept patent by the brawny inflammatory induration which in such an advanced case surrounds the necrosed cartilages.

Thus in a case of laryngeal cancer which I have recorded,² the only cartilage left was a piece of the thyroid about the size of a florin, and yet the patient died of exhaustion and not of suffocation, and indeed suffered little from dyspnœa throughout.

4. To stenosis resulting from cicatricial contraction.

As stated, this is practically not met with except in syphilitic cases of long duration.

5. When the dyspnœa occurs in a paroxysm it may be due to some sudden inflammatory œdema, or to the detachment and impaction of the necrosed cartilage, the first being by far the commoner cause.

Diagnosis.—The diagnosis of the affection usually presents few difficulties, the swelling visible with the laryngoscope, the history of the case, and the symptoms present are usually sufficient. It is, however, by no means so easy to determine the cause. This difficulty is greatest in the case of cancer. Extreme infiltration of the cricoid, thyroid, and neighbouring tissues, with the occurrence of hæmorrhage from the larynx, is strongly in favour of cancer, but neither of these symptoms is conclusive.

Prognosis.—The prognosis is always gloomy, for though it is true that perichondritis rarely kills, except where tracheotomy has been too long deferred or pneumonia has resulted from the passage of some of the secretions into the larynx, still it is very unlikely to get well.

As long as any part of the necrosed cartilage remains in the larynx, the discharge will be a constant source of distress. Even if the dead cartilage be got rid of, the original disease has still to be reckoned with, and this in itself is in most cases incurable.

If not of itself fatal, perichondritis may greatly accelerate the course of the original disease and add to the misery of the patient.

Practically the only chance of healing is in the syphilitic cases, and even then serious stenosis will probably be left which will necessitate tracheotomy and the wearing of a cannula for the rest of life.

Treatment.—It will be necessary of course to treat the general condition of the patient as well as the local disease, if there be one, to which the

¹ Cf. Fränkel, *Contrib. f. Laryngl.*, iv. 103.

² *Trans. Path. Soc.*, 1887.

perichondritis is secondary; thus in syphilitic cases iodide of potassium should be given in full doses and some mercurial rubbed in externally over the larynx.

Locally the inflammation may be combated by the usual remedies.

For the pain and cough opium may be given internally, or opium and cocaine applied locally. The pain on swallowing, which may cause the patient to look forward to meal-times with dread, may be greatly relieved by painting the pharynx and neighbouring parts about the glottis with cocaine a short time before food is given.

The swelling may be relieved by scarification or the abscess laid open if necessary with the lancet.

If the dyspnoea be considerable, tracheotomy must not be too long delayed.

If stenosis develop, the stricture may be treated on the usual lines by means of cones, or graduated tubes with or without previous incision.

It has been proposed to deal with the necrosis locally, *i.e.*, to open the larynx and excise the dead cartilage. The operation has been successfully performed, but its ultimate success or usefulness in any case depends almost entirely upon the nature of the primary disease.

II. IN THE TRACHEA AND BRONCHI.—In the trachea perhaps the most frequent cause is the mechanical injury produced by the end of a badly-fitting tracheotomy tube, when it has been long worn: otherwise in the trachea and bronchi perichondritis and necrosis of cartilage are rare and almost invariably associated with the ulceration of tubercle, syphilis, or malignant disease.

The symptoms are indefinite; generally those of bronchitis or tracheitis, as the case may be; but if there were much swelling there might be much dyspnoea as the result of the stenosis produced by it.

In the trachea there would probably be some external swelling, which would be tender on pressure and might even fluctuate. If, as sometimes occurs, the abscess bursts, not internally but externally, into the tissues of the neck, diffuse subcutaneous emphysema will develop, and this may be followed by suppuration.

I do not know that the cartilages have ever been excised in the trachea, but, though a delicate operation, it might be practicable in appropriate cases, if the diagnosis had been correctly made and the seat of lesion accurately fixed.

In the bronchi nothing is possible except general treatment.

Necrosed cartilages both from the trachea and bronchi are sometimes exfoliated and coughed up, but this is a rare event.

In syphilitic cases a cure may sometimes result, but, as in the larynx, the risk of subsequent stenosis is considerable.

14. LARYNGITIS TUBERCULOSA—LARYNGEAL PHTHISIS.

Laryngitis is a very common affection in phthisis, and is, in most cases, associated with lesions which are undoubtedly tubercular, and, even where they are not characteristic, microscopical investigations prove that they are far more frequently tubercular than has been hitherto supposed.

Laryngeal tuberculosis is most frequently met with (1) as a peculiar infiltration and ulceration, but it may also occur in the form of (2) follicular ulceration,

- (3) superficial erosion or ulceration, (4) occasionally as a miliary granulation, and (5) as a papillary tumour.

The occurrence of acute miliary tuberculosis of the larynx has been denied, but cases have been recorded, both in acute general tuberculosis and in the course of chronic tuberculosis both of the larynx and of the lung.¹

Tubercles in the larynx do not differ in constitution from tubercles elsewhere, indeed, as Virchow long ago pointed out, the larynx is one of the best places for the study of them. Their only peculiarity in the larynx consists in the smallness of their number and size, and in the large amount of infiltration with which they are usually attended.

As a rule they are not visible to the naked eye, and even in cases of undoubted tubercular ulceration it is only rarely that they can be seen at the margin of the ulcer, but on microscopical examination they are common enough, and that often without any ulceration.

They are usually seated in the deeper layers of the mucosa, the basement membrane and the epithelium over them being intact. The swelling is due to a small-celled infiltration and is not a simple œdema.

As the affection goes on to ulceration the epithelium is shed and the basement membrane disappears; thus an ulcer is developed, the base of which is formed of the degenerating tubercular infiltration.

As the ulcer extends it may spread along the surface or into the deeper tissues.

The mucous glands and ducts are often preserved intact for some time, even in the midst of dense tubercular infiltration; but they are sometimes, on the other hand, the first structures to be involved, and that either at the mouth of the ducts or in the acini. This form constitutes the so-called *follicular ulcer*, which it is very difficult to distinguish, except by microscopical examination, from the glandular affection due to simple catarrh. The same difficulty arises in the case of the early epithelial changes described as *erosions* or *catarrhal ulcers*. The fact that the tubercles are so often deep-seated, and that the epithelium and basement membrane over them are intact, led Heinze to deny that the primary superficial lesions were really tubercular, but this opinion has proved to be incorrect.

The vessels, as elsewhere, are obliterated in the centre of a tubercle, the veins early but the capillaries later; the arteries, if not obliterated, are in a condition of periarteritis.

When the vessels are obliterated, necrotic changes may follow and lead to a rapid extension of the mischief.

The muscles may remain for long unaltered in the midst of dense infiltration, but if not involved in the ulcerative process they subsequently undergo granular degeneration and atrophy, either as the result of pressure or of interference with their blood supply.

The nerves, of course, are involved also, so that peripheral lesions, either of the fine branches or of the nerve-endings, may explain some of the paralyses observed in the course of the disease, which cannot be referred to gross disease of the nerve-trunks; but in many cases the motor defects are the result, not of any affection of the nerves, but of the muscles themselves, or else are due to inflammatory changes set up in the joints and fixing them. An interesting case of adhesive arthritis fixing the cords in the position of extreme adduction is recorded by Kidd.²

¹ Gouguenheim and Tissier, *Phthisie laryngée*, pp. 120 and 147. Fränkel, *Berl. klin. Woch.*, Nov. 1876.

² *Lancet*, Mar. 29, 1889.

The lymphatic glands outside the larynx are rarely enlarged as the consequence of intra-laryngeal tubercle, any more than they are in syphilitic or in malignant disease of the larynx.

If the cartilages are involved, acute perichondritis may be set up, and they necrose, but if not, they may, nevertheless, undergo other chronic degenerative changes. Thus they may become calcified or even, it is stated, ossified, and in the cartilages so altered caries may occur. As a rule the cartilages escape unless the ulceration extends directly to them, but in some rare cases they may be affected before the ulceration reaches them.

Chronic tubercular laryngitis may lead, like other forms of chronic laryngitis, to the formation of polypoid vegetations, composed of a soft connective tissue. Some of these are really tubercular and contain caseous nodules and bacilli. They are somewhat rare, and are difficult to diagnose from simple papillomata, but papillary growths on the posterior aspect of the larynx, *e.g.*, in the inter-arytenoid region, are usually tubercular, while those on the anterior part are usually not.

The seat of predilection of tubercular disease in the larynx is determined greatly by movement and exposure to infection. The favourite seats, in order of liability, are (1) the posterior part of the vocal cords, (2) the inter-arytenoid folds, (3) the laryngeal surface of the arytenoid cartilage, (4) far less frequently the epiglottis and ventricular bands, and lastly the sub-glottic region.

Lebert gives the following statistics for 80 cases of tubercular ulceration:—the vocal cords were affected alone in 33 per cent. and with other parts of the larynx in 39 per cent. more, the epiglottis alone in 13·5 per cent., and the sub-glottic region in 4·8 per cent. Infiltration existed alone without ulceration in 8·5 per cent.

The ulceration in the larynx is usually the result of infection from the lung, and in this connection it is interesting to know, as Kidd has pointed out, that it is on the posterior surface of the larynx that, in foggy weather, the fog-stained sputum is seen to lie. The superficial erosions or catarrhal ulcers, if not themselves of tubercular origin, are, no doubt, the predisposing causes of the infection.

The affection is generally unsymmetrical, *i.e.*, one side is affected either alone, or to a greater extent than the other; but where both sides are involved it is usually the symmetrical or corresponding parts that are the seats of disease.

Although all forms of tubercular lesion may be met with in the same larynx, still each may exist alone.

Swelling is often the most prominent feature in tubercular laryngitis, though it varies greatly in extent and position in different cases. It is not an œdema, but an infiltration. It differs from œdema in its pale reddish yellow colour and in its density, the section being firm and yielding but little serum.

It may be widespread and involve all the parts about the glottis, but, in spite of this, rarely produces actual stenosis.

When limited to the arytenoid region the swelling has a characteristic pyriform, conical, sugar-loaf shape. In the inter-arytenoid space it may be sufficient to interfere with the approximation of the cords. The false cords are often so much swollen as to overhang the true and hide them from view and to obliterate the ventricles of Morgagni.

The epiglottis when infiltrated assumes a cylindrical turban-like shape. It is often no longer vertical, but projects backwards and is capable of but limited movement. The true cords exhibit little actual swelling, and though the microscope shows that they are infiltrated, definite tubercles are not common in them.

Ulceration of the larynx is rare except in phthisis.

Heinze states that not more than 5·2 per cent. are due to other causes, that is to say, that of 100 cases of laryngeal ulceration due to all causes, in 94·8 the patients are the subjects of pulmonary phthisis.

The usual distribution of the lesion is shown by the following analysis of fifty cases made by Heinze :—

True vocal cords	40	{ Both	27
		{ One	10
		{ Anterior commissure	3
Arytenoids	23		
Epiglottis	26		

The *arytenoid region* is a favourite seat, and the cartilages and the crico-arytenoid joints are then likely to suffer. In the inter-arytenoid space, ulceration is common, but it is often very difficult to see with the laryngoscope on account of the swelling.

The *vocal cords* are still more frequently affected. In the early stage they are congested, have lost their polish, present linear or superficial erosions, and the edge is often finely serrated.

The first sign of ulceration is often visible at the junction of the posterior and middle third. When ulceration is advanced the edges of the cords are ragged and the tags may look like polypi, the margins are thickened and irregular, and their attachment to the vocal processes of the arytenoids may become broken through.

The *epiglottis* is less frequently ulcerated and hardly ever alone. It usually becomes involved by the extension of ulceration from the parts adjacent, especially the arytenoid region. The ulceration is usually near the base on the laryngeal surface, but it is occasionally on the edge. The lingual surface is never affected alone.

The ulcers are shallow, superficial, follicular and often confluent. Such ulcers are rare in other parts of the larynx except over the cricoid, but in the trachea they are common.

It is difficult during life to say whether the follicular or glandular ulcers are simple or tubercular, but practically in a case of phthisis nearly all such cases prove to be tubercular, and the same remark applies to erosions and superficial ulcers.

Polypoid tubercular vegetations are rare and are often difficult to distinguish by the laryngoscope from the tags of infiltrated tissue left by ulceration.

The earliest case of the kind was described by Andral, but Mandl¹ was the first to draw the distinction between tubercular polyps and other forms of laryngeal tuberculosis. He maintained that such a polyp in the larynx might be the first manifestation of tubercle and might even precede any lesion in the lung.

Rare as these tubercular tumours are, a sufficient number has been recorded for the following account to be given of them.

Their favourite seats are the base of the epiglottis, the inter-arytenoid space and the sub-glottic region, but they may also occur in almost any part²; on the true cords,³ on the false cords,⁴ and on the wall of the ventricles.⁵

¹ *Mal. d. Lar.*, 1872, p. 678, pl. vii. fig. 2. J. Mackenzie, *Arch. de Med.*, viii. 109.

² Gouguenheim and Tissier, *l.c.* Avellis—three cases recorded, with literature, *Deutsch. med. Woch.*, 1891, 6th August.

³ Kidd, *Med. T. and G.*, 1884, 6th August. *St. Barthol. Hosp. Rep.*, 1885.

⁴ Hering, *Curabilité de la Phthisie laryngée*, 1887, p. 42.

⁵ Schnitzler, *Wien med. Woch.*, April 1883. Hennig, *Berl. klin. Woch.*, 1888, 564. A good microscopical description is given by Nasse, *Deutsch. med. Woch.*, 1887, p. 307.

They are usually solitary, but may be multiple, and even by their number cause obstruction to breathing; they vary much in size, for though usually small, they are sometimes as large as a hazel nut, occasionally sessile, but usually more or less pedunculated; of a pale rose or yellowish red colour; of slight consistency, especially towards their insertion, so that they are not infrequently broken off and coughed up; their structure is that of young connective tissue, but their base is often caseous and contains bacilli.

Gouguenheim records a case in which dyspnoea was caused by a cauliflower growth extending from the base of the epiglottis to the anterior insertion of the vocal cords. Tracheotomy became necessary, after which the growths were removed and found to contain bacilli. The growth recurred and was removed on several occasions, and after a time the tracheotomy tube was dispensed with. No physical signs had developed in the chest up to the time of recording.

In the sub-glottic region tubercular lesions are much less frequent and important. There is as a rule but little infiltration, but if the cricoid be involved there may be so much swelling as to cause considerable stenosis. The ulcers are superficial and follicular, like those in the trachea, to which in its lesions this region presents more resemblance than it does to the larynx.

The frequency of tubercular laryngitis in phthisis is very difficult to determine except by *post-mortem* examination; for, in the first place, tubercular lesions may exist in the larynx without any symptoms; secondly, lesions may exist and be recognised in the larynx which can only be proved to be tubercular by microscopical examination; and lastly, the disease in the larynx may so mask the physical signs in the chest as to cause well-marked disease in the lungs to be missed.

Heinze examined 50 consecutive fatal cases of phthisis, with the result of finding only 3 in which tubercle did not exist either in the larynx or in the trachea or in both.

In the larynx alone in 23. In both the larynx and trachea in 24. In the trachea alone in 2.

In the larynx the ulcers were evidently tubercular in 39 cases and not evidently tubercular in 8.

In the trachea the ulcers were tubercular in 18 and not tubercular in 8, but in all these 8 there was tubercular ulceration also in the larynx.

Expressed as percentages—

Tubercular ulcer was present in the larynx alone in 40·8 per cent.

Tubercular ulcer was present in the larynx and trachea in 22·4 per cent.

Tubercular ulcer of the larynx was associated with non-tubercular ulcer of the trachea in 16·3 per cent.

Tubercular ulcer of the trachea was associated with non-tubercular ulcer of the larynx in 10·2 per cent.

Ulceration (non-tubercular) of larynx without ulceration of trachea was found in 4·0 per cent.

There was no case of ulceration in both trachea and larynx which was non-tubercular in both alike, and also no case of non-tubercular ulceration of trachea without tubercular ulceration of the larynx.

The figures given by different observers vary greatly, but the differences depend chiefly upon whether they are derived from observations made by the laryngoscope during life or by *post-mortem* examination.

Thus, out of 100 living patients, Morell Mackenzie found tubercular laryngitis in 33, and Heinze, out of a much larger number, in 30·6 per cent. ($\frac{276}{900}$).

Ormerod,¹ out of 100 out-patients suffering from phthisis of the lungs, found the larynx healthy in 25 only. The arytenoid was affected alone in 16, and with other parts in 14 more.

It may be stated in general that of phthisical patients about 30 per cent. have some form of tubercular ulceration in the larynx, but not more than about 12 per cent. have it in an active form at the time of death.

Sex.—Like pulmonary tuberculosis, tubercular laryngitis is most frequent in males (2·7 to 1, Morell Mackenzie; 4·5 to 1, Heinze), and between the ages of 20 to 30, but it is said to be fatal at a later age in males (40 to 50) than in females (30 to 40).

Occupation seems to be without influence.

Age.—The following table shows the approximate age distribution :—

Mackenzie (500 cases) .	0 to 10 10-20	20-30	30-40	40-50 50-60	60-70	70-80
	35	194	162	109		
Bosworth (376 cases) .	5 23			67 27		
	28	130	112	94	9	3
Percentage of (876 cases)	7·2	37	31·5	23·2	1	0·3

Tubercular laryngitis is extremely rare in children.

Heinze refers to only 9 cases out of 400 under the age of 14, and of these 3 were under 2 years of age, 2 between 2 and 5, and the remaining between 10 and 15. Morell Mackenzie met with it in 4 cases between 5 and 10 years, and in 4 between 10 and 15. Rheiner records a case at 4 years of age, but the youngest on record was met with in a baby of 11 months (Heinze).¹

History.—Laennec in 1819 was the first to insist upon the tubercular nature of the laryngeal lesion in phthisis, although the affection had been described by Pettit in 1790, and subsequently by Portal and Sauvée. After Laennec's time opinion was divided as to its tubercular nature, high authorities ranging themselves on either side.

The most important recent contributions are two, the one by Heinze² in 1879, and the other by Gouguenheim and Tissier³ in 1889.

LARYNGEAL PHTHISIS AND PRIMARY LARYNGEAL TUBERCULOSIS.—Tuberculosis of the lung and larynx are usually found associated together, but not invariably, for the lung affection may run its course to the end without the larynx being involved.

The question then arises, Is that true also of the larynx? May tubercular laryngitis run its course and the patient die of it without the lungs becoming affected; in other words, is there such a thing as **Laryngeal Phthisis**? In the strict sense of the term there is not. *Post-mortem* evidence is entirely against it, and cases recorded without *post-mortem* examination are worthless on account of the difficulty in advanced laryngeal disease of determining with certainty the condition of the lungs.

These difficulties of diagnosis are well illustrated by the following case :—A young man of 30 was a patient of mine at the Chest Hospital with advanced tubercular disease of the larynx. The patient had completely lost his voice and could only speak in a hoarse whisper; the respiration was stridorous and noisy, and could be heard so loudly all over the chest as to deprive auscultation of any value. There was no dulness to be found on percussion nor any defect of shape or movement. I examined the patient repeatedly without being able to detect signs of pulmonary disease, and I began to wonder if I had got a case of true laryngeal phthisis to deal with. Several of my colleagues also examined the patient with me and failed as I did to obtain any direct evidence of pulmonary disease.

The patient died shortly afterwards of asthenia, and on the autopsy which I made myself I found a large cavity at the right apex 2 or 3 inches in diameter, surrounded with thick walls in which caseous masses were abundant. The cavity was, without doubt, tubercular, of date long antecedent to the laryngeal mischief.

If, then, there be no such thing as laryngeal phthisis in the strict sense of the term, the question arises whether there be such a thing as primary laryngeal tuberculosis, *i.e.*, whether the larynx may be affected before the lung.

¹ *Jahrb. f. Kinderheilk*, 1891, vol. xxxii. Rheindorff was only able to collect 20 recorded cases in the year 1891.

² *Die Kehlkopf. Schwindsucht*, Leipzig, 1879.

³ *Phthisie laryngée*, 1889.

This was formerly denied by many high authorities, but the existence of such cases must now be regarded as conclusively established although their rarity is open to no question.

Orth¹ records a case in which the lungs were at the time of death still quite free. In Fränkel's case the laryngeal mischief had been watched for five years, and the mischief in the lungs was evidently, on *post-mortem* examination, of comparatively recent date.

An interesting case is recorded by Demme² in a child of 4½ years, who died of tubercular meningitis, in whom the first sign of tubercle appeared in the larynx, and the thoracic and abdominal organs were found quite healthy at the autopsy.

In the overwhelming majority of cases the lungs are the prime seat of the disease and the larynx only becomes involved secondarily.

Before the infective nature of phthisis was established the affection of the larynx was explained as the result of the irritation and attrition of the sputum as it was projected by coughing through the larynx, but it is clear that something more than simple attrition was necessary, for no such lesions were found, with anything like the same frequency, in chronic bronchitis, and this something more was infection. It was accordingly assumed that the tubercular infection was at first superficial, and it is true that all the superficial or so-called catarrhal lesions mentioned have been frequently found to contain bacilli and to be, therefore, tubercular in nature at the time of examination; still these lesions may have been simple to commence with, and have become infected subsequently. On the other hand the demonstration of deep-seated tubercles beneath intact epithelium and basement membrane shows that the surface cannot be the only source of infection.

There must, therefore, be two sources of infection in the larynx, the one deep-seated and connected probably with the blood-vessels, the other superficial and preceded by some lesion of the epithelium, often of a simple nature, such as the catarrhal erosion or ulcer.

Symptoms.—The symptoms are those of laryngitis, but they vary much according to the seat and extent of the lesion, and even with considerable mischief there may be no symptoms at all.

The relative frequency of the different symptoms is thus stated by Mackenzie—hoarseness 30 per cent., dysphagia 30 per cent., soreness in the throat 12·4 per cent., cough 85 per cent., dyspnoea 83 per cent. (but both of these latter symptoms may depend upon the co-existent lung lesion). Marked laryngeal dyspnoea occurs only in 2·2 per cent., and is of sufficient severity to call for tracheotomy in only 0·6 per cent. (Kidd, 0·4 per cent.).

Weakness of the voice is an early symptom in phthisis, that is to say, the voice gets readily tired and husky, and speaking is attended with a feeling of fatigue or even of pain.

Aphonia is generally due to imperfect movement of the cords, and if not functional may be due to infiltration or degeneration of the muscles or to an affection of their nerves or blood-vessels.

Hoarseness or loss of voice depends, as a rule, directly upon the lesions in the larynx, which diminish the elasticity of the cords or impair their free movement.

Cough is a constant symptom, but is often to be associated with the condition of the lungs rather than with that of the larynx. It may be very slight, but is sometimes severe and paroxysmal, and may be almost constant day and night, so as to break the rest and be the cause of great distress.

Pain or difficulty in swallowing is not rare, and is met with commonly where the arytenoid region or the epiglottis is involved, but it may be due to ulceration at the back of the tongue, in the pharynx, or in the œsophagus.

¹ *Lehrb. d. Spec. Pathol. Anat.*, p. 319. *Deutsch. Med. Woch.*, 1886, 490.

² *Virchow. Jahresber.*, 1883, ii. 642.

On the other hand, the epiglottis may be considerably affected without dysphagia or any other symptom except that the patient has to swallow with extra care; it may even be entirely destroyed without the food passing into the larynx.

Salivation has been described, but is probably due rather to the difficulty in swallowing than to an actual increase in the secretion.

Pain and tenderness on pressure over the larynx are not often experienced, unless there be perichondritis or some inflammation round the larynx.

Laryngeal dyspnœa is rare, and it is remarkable how large the amount of swelling may be without obstruction to the entrance of air. Laryngeal stenosis, which is so common in syphilitic and malignant disease, is rare in tubercular affections.

Hæmorrhage from a tubercular larynx has been described, but it must be extremely rare.¹ It is hardly to be diagnosed except by actual observation of the bleeding spot with the laryngoscope, but it is stated by Albers that the blood is brought up without coughing, and that the sputum is not stained for some hours afterwards as in ordinary hæmoptysis.

Prognosis.—Patients rarely die of laryngeal tuberculosis, except in those rare cases in which stenosis has been produced and tracheotomy has not been performed in time; but, though it does not often kill, it shortens life, either by the symptoms it produces, *e.g.*, the cough and the dysphagia, or by some complication to which it may give rise, *e.g.*, perichondritis.

Mackenzie stated that in phthisical patients the average duration of life with tubercular laryngitis was under two years, and without it not less than three years, so that tubercular laryngitis reduced the prospect of life in a phthisical patient by at least one year.

The general prognosis depends more upon the condition of the lungs than of the larynx, and is especially bad when the pulmonary mischief is rapidly advancing.

Of 100 cases observed by Mackenzie 79 terminated in from 6 months to 2½ years after the throat symptoms became pronounced, 9 died within less than 6 months, and 12 only lived from 2½ to 4 years.

The prospects of cure are very slight. Spontaneous cicatrization has been recorded by Virchow, Bouveret, and others.

Fagge² refers to 3 cases in which the laryngeal ulcer appeared to heal completely, and Mackenzie met with 4 cases of recovery where the lung condition remained stationary. Ziemssen³ and Heinze⁴ also record cases, but Ziemssen⁵ states that though the ulcer may heal in one part it usually at the same time spreads in another.

Charters Symonds⁶ also records one or two instances of spontaneous recovery though followed by subsequent relapse.

Solis Cohen⁷ records a remarkable case of stricture of the larynx, resulting from the cicatrization of tubercular ulceration in which he operated by incision of the cicatricial tissue with great benefit. The portions removed contained tubercle bacilli. When the patient died, about a year later, the scar of the operation had, to a great extent, cicatrized.

Diagnosis.—The diagnosis in an ordinary case presents no difficulties; the pallor of the velum and epiglottis, together with the character of the swelling and ulceration, leave no room for doubt.

In the very early stage the diagnosis must be made from simple catarrhal laryngitis, both acute and chronic; where swelling exists alone, from œdema

¹ Gouguenheim and Tissier, *l.c.*, p. 199.

² *Pract. of Med.*, 812.

³ Cf. also Hering, *Deutsch. med. Woch.*, 1887, p. 160.

⁴ Luc, *L'Union Médic.*, 1888, p. 254.

⁵ Bouveret, *Lyon Méd.*, 1882, p. 126.

⁶ *B. M. J.*, September 13, 1890.

⁷ *Internat. Jour. of Med. Sc.*, 1888, p. 517.

of the glottis, gumma or new growth; where ulceration is present, from cancer and syphilis; and where tubercular vegetations occur, from simple papillomata.

Under ordinary circumstances the diagnosis will probably lie between tubercle and syphilis.

In tubercle the chief seat of lesion is the arytenoid region, inter-arytenoid space, and the vocal cords. The epiglottis is rarely involved, and then usually at the base and on the laryngeal surface. The ulcers have irregular tumid edges and bases which are often caseous, and the swelling around is pale pink rather than red; they spread slowly and show no tendency to heal. Polypoid growths in the inter-arytenoid region are almost pathognomonic.

In syphilis, on the other hand, the ulcers are more sharply cut and usually larger, have a grayish base covered with pus, spread rapidly, tend to heal with much cicatrization, and may often be cicatrizing in one part while spreading at another. The swelling around is redder and more inflammatory in appearance. The arytenoid region is but rarely affected; the epiglottis is frequently affected, but then on the lingual surface, and the ulcers may be often traced by direct continuity from the pharynx.

Syphilitic ulcers may become tubercular and *vice versa*, or the two affections may exist side by side.

The diagnosis between the two diseases is usually not difficult, if the general as well as the local conditions and the previous history be taken into account. Besides the common forms of ulceration mentioned, it must not be forgotten that ulceration is occasionally met with in other diseases, for example, in typhoid, in pneumonia, in pyæmia, in erysipelas, and in Bright's disease; but with the exception of typhoid they are all of greater pathological interest than clinical importance, and rarely cause any difficulty in diagnosis.

Treatment.—The *general treatment* is the same as that of tubercle elsewhere. The *local treatment* is chiefly symptomatic, and although all the remedies employed in simple laryngitis may be used, dependence is chiefly placed upon direct applications by the brush or in the solid form to the parts diseased.

The cough, pain, and local irritation may be relieved by sedative drugs:

Morphine, either as an insufflation with starch powder (1 to 8), or dissolved in glycerine (*gr.* 1 to the *drachm.*), and applied with the brush;

Cocaine (10 to 20 per cent. solution) is most serviceable as a local anæsthetic, either to prepare the way for the use of other applications, or to allay pain and irritation. Where there is dysphagia, its application before a meal may enable the patient to take food in comparative comfort.

Menthol, as a 20 per cent. solution in oil, as well as *caffeine* and *salicylic acid* in equal parts, have been also used, but they are far inferior to either of the other sedatives.

Astringents are of little use, but those most recommended are *T. Ferri Perchloridi* (31 to 31), *chloride of zinc* (*gr.* 15 to 31), as well as alum and acetate of lead.

With the view of modifying the disease, *iodoform* has been much used, either in fine powder for insufflation or dissolved in glycerine. Iodoform does not cure, but cleanses the surface and promotes healthy granulation. A useful powder is composed of *Iodoform*, *Boracic Acid*, *aa gr. i*, *Morph. Acet.*, *gr. ½*.

Iodine dissolved in iodide of potassium and glycerine is said to be especially useful in checking vegetations.

Iodol dissolved in ether or alcohol and boracic acid in glycerine has been advocated, but is now little used.

Of the caustics, those most employed are *nitrate of silver*, either in the solid form or in strong solution; *solid chromic acid*, an irritating application, and one of which it is difficult to localise the action; *creasote*, in glycerine, 1 part in 30. These are now all to a great extent given up in favour of *lactic acid*. The use of lactic acid was introduced by Krause,¹ who recommended a strength of from 20 to 80 per cent. It has been applied undiluted with benefit. It is rarely necessary to use a stronger solution than 40 to 50 per cent., or to apply it more often than two or three times a week (Semon).

Many cases of cicatrization and recovery under its use have been recorded, though in most cases the disease subsequently recurs. Kidd records two cases in which there had been no return of the disease five years after treatment.

To reduce the swelling *scarification* has been advocated, but is now abandoned.

Attempts have been also made to deal with the disease locally by *scraping* or by the *galvano-cautery*, but without any permanent success.

Where polypi exist they may be removed, but if tubercular they frequently recur.

If food tends to pass into the larynx, liquids can be sometimes better swallowed when thickened with arrowroot, etc., or the patient may be fed with a tube. The passage of a tube often gives pain, and in some cases the patients prefer the ordinary difficulty of taking food to the use of the tube, but their objections will probably be overcome by the previous application of cocaine. The difficulty in swallowing may sometimes be relieved by the patient lying on the face, as Wolfenden suggested, and sucking the food up through a tube.

If extreme dyspnoea develop, *tracheotomy* must be performed, but it is a desperate remedy, and though it may give relief for the time it generally marks the beginning of the end.

Tubercle of the Trachea and Larger Bronchi.

Tubercular affections of the trachea and larger bronchi are common but of slight importance, for they produce no definite symptoms, and are not amenable to special treatment.

Infiltration is slight and hardly ever sufficient to produce any obstruction.

Ulceration is superficial and follicular. It chiefly occurs in the posterior membranous part, where it tends to spread in the long axis of the tube; if in the intercartilaginous parts it spreads transversely.

The cartilages may be involved and sometimes necrose, and, if perforation occur externally into the tissues of the neck, emphysema may be produced, but such an event is extremely rare.

The trachea is the best place to study the follicular form of tubercular ulceration. In this form the mischief usually commences in the mouths of the mucous glands. The ulcers are at first superficial, circular, funnel-shaped, with a narrow yellow margin showing up in sharp contrast against the surrounding hyperæmia, in the midst of which is seen the mouth of the duct.

If the ulcer extend along the gland it may reach the cartilage, but it more commonly spreads superficially, and by fusion with neighbouring ulcers assumes a racemose or irregular shape.

In the small bronchi the ulceration may often be traced in direct continuity from a cavity for some distance along the tube. Of treatment we can hardly speak, for except in some parts of the trachea, where they can be seen with the laryngoscope, the lesion is undiagnosable, and the only local treatment possible is by intratracheal injections, *q.v.*

¹ *Deutsch. med. Woch.*, 1886. Hering, *do.*, 1887, p. 127.

15. SYPHILIS OF THE AIR-TUBES.

Syphilitic affections occur in the air-tubes with a frequency which decreases rapidly with the distance from the pharynx; thus they are three times more common in the pharynx than in the larynx, while in the trachea and bronchi they are rare.

Morell Mackenzie found in 1145 cases the pharynx affected in 834, the larynx in 308, and the trachea and bronchi only in 3.

Lewin gives the frequency of laryngeal affections of any kind in the course of syphilis as 3 per cent., and of this 3 per cent. only about 1 case in 10 has more than trifling lesions.

The Larynx.—Syphilitic affections of the larynx are most common between the ages of twenty and thirty, that is, at the time when syphilis is most prevalent, but except in the form of catarrh they rarely develop before the third month after infection. They may arise at any subsequent period even after twenty or thirty years, and also at an advanced time of life; for example, even at sixty or seventy years of age; and in such cases they may be the only manifestation of syphilis present at the time. On the whole they are more likely to arise in the later periods of the disease, and indeed the liability to them seems to increase with the duration of syphilitic symptoms. They are stated to be commoner with the cutaneous than with the visceral forms of syphilis. They have been met with also in congenital syphilis in children, and even in infants.

Syphilis of the larynx.—The lesions met with in laryngeal syphilis are (1) catarrh, (2) condyloma, (3) gumma, (4) ulceration, (5) cicatrization.

Catarrh is common but presents nothing characteristic. It may be a very early complication, or occur at any later period. It is generally very slight, and often so transient that the patient does not apply for treatment. It is most likely to occur, like other forms of catarrhal laryngitis, in cold damp weather, and among persons who have to use their voice much.

Condyloma, or mucous patch, though very rare, has been described by competent observers. It may occur within a few weeks of infection or even as late as twelve months after. Its most frequent seat is on the upper surface of the vocal cords, and after that on the epiglottis, arytenoids, and ventricular bands. The symptoms produced depend upon its seat; the interest of this lesion is connected chiefly with its diagnosis.

Gummata occur in two forms, viz., as tumours and as an infiltration.

Gummatous infiltration is most frequent and may be very extensive, sometimes involving all the parts about the glottis (*the total infiltration of Dittrich*), and producing severe stenosis.

Gummatous tumours vary much in size. They may be small, like a shot or pea, and are then often multiple; but they occasionally reach a large size, as in the case of Norton's,¹ where a patient died of suffocation, and a gummatous tumour of the size of a pigeon's egg was found in the right arytenoid fold; a similar case is recorded by Moure²—the tumour was of large size and necessitated tracheotomy; it ultimately disappeared under treatment.

Gummata in either form often ulcerate and break down, but under treatment they may subside and rapidly resolve.

Ulceration is, after catarrh, the commonest syphilitic lesion in the larynx, it may occur in any part, even below the true cords, but it is commonest in the parts adjacent to the pharynx, i.e., in the folds connected with the epiglottis or on the posterior walls of the larynx. It often attacks both false and true cords,

¹ *Path. Soc. Trans.*, 1874.

² *Rev. mens. de Laryngol.*, 1884, p. 297.

and though it may arise independently and primarily in the larynx, still it is more frequently to be traced from some contiguous ulceration in the pharynx.

It has two great characteristics, viz., destructiveness and cicatrization. A syphilitic ulcer may commence as a limited superficial lesion, and, rapidly spreading, may even reach considerable size without producing marked symptoms. When the ulceration is due, as it so often is, to the breaking down of gummata, the ulcers formed are deep and penetrating, so that they are very likely to set up perichondritis with consequent necrosis of cartilage. This is a serious lesion, and may of itself be the cause of death, but the prognosis is better in syphilis than in tubercle, for if in syphilis the cartilages exfoliate and be got rid of, the parts may rapidly heal, but the resulting cicatrization has dangers of its own and may end in one of the most intractable forms of stricture.

The **diagnosis** has in most cases to be made between syphilitic ulceration and that due to tubercle. The special points of distinction have been dealt with when treating of tubercle. The diagnosis will, as a rule, be assisted by the existence of characteristic ulceration elsewhere, as, for instance, in the pharynx, or by the evidence of cicatrization in some adjacent part of the larynx.

It is asserted that the general health is better and longer preserved in syphilis than in tubercle of the larynx, but even if this statement were correct, it would be of little use in diagnosis. The diagnosis from malignant disease may at times present difficulties, and will then depend upon general considerations.

Cicatrization.—The healing of syphilitic ulcers is often associated with a massive production of connective tissue, in which remarkable contraction takes place. The effect upon the larynx will depend upon the seat of the scar. Thus in the arytenoid folds cicatrization may produce great deformity with few symptoms, but round the glottis it will lead to stricture and symptoms of great severity.

As in other forms of chronic laryngitis, so in syphilis, papillary out-growths may form and require removal.

A striking peculiarity of syphilitic ulceration is its tendency to contract adhesions as it heals with other ulcerated surfaces near it. Thus the epiglottis may become attached to one or other arytenoid fold and be fixed in a more or less horizontal position. This may occur without any symptoms of stenosis and without the passage of food into the larynx. When ulcerated surfaces on opposite sides of the larynx cohere, a remarkable result is produced, for a web may be thus formed stretching transversely across the larynx, and appearing almost completely to close it. This is a rare lesion (*cf.* Cicatricial Stricture).

Laryngeal Syphilis in Children.—Laryngeal lesions have been met with in congenital syphilis, and in very young children. Thus Parrot¹ records a case in which a round ulcer existed between the vocal cords in the anterior part of the larynx, in an infant of two months, and Fraenkel² another in which syphilitic ulceration was followed by necrosis of cartilage, and caused the child's death at the age of three months.

Barlow thinks from the frequency of laryngeal symptoms in syphilitic infants, that congenital syphilis of the larynx is commoner than *post-mortem* or other published records show.

Sturge³ records a case in a child of 3½ years of age, who lost his voice for a time when 12 months old, but regained it for a time, losing it again six months later, the loss of voice and dyspnoea continued then till his death two years later, which was quite sudden.

Extensive syphilitic ulceration was found, involving both vocal cords, and extending above and below them. Above, the ulceration was of older date, and associated with much cicatrization.

¹ *Prog. Med.*, 1878, No. 34.

² *Wien med. Woch.*, 1868, 69 and 70.

³ *Path. Trans.*, xxxi. 394.

Below, it extended into the upper part of the trachea. Between the true and false cords was a web extending across the anterior part of the larynx, which was thought to be congenital.

Semon records two very remarkable cases in brothers, who died at the ages of 5½ and 3½ years respectively, within three weeks of one another, of acute œdema of the larynx.

Both were victims of congenital syphilis, and had suffered from hoarseness and stridulous breathing from the age of a year or so. Both presented much the same lesions. In the elder there was considerable hyperplasia of the whole larynx, especially of the anterior wall, involving the epiglottic, aryteno-epiglottic and inter-arytenoid folds. In the younger the changes were not so extensive. In both numerous shallow ulcers existed around the hyperplastic tissue.

This hyperplastic condition, which is very rare in adults, is almost unknown in children.

Symptoms.—The symptoms of laryngeal syphilis are not specially characteristic. The voice is impaired more or less and may be completely and permanently lost; cough is frequent; pain is usually absent unless there be perichondritis; the expectoration of mucopurulent sputum is common in the ulcerative stage, and may be copious in some rare cases; hæmoptysis may occur, and even be fatal, as in a case recorded by Türk;¹ lastly, when cicatrization has occurred the symptoms of stenosis may arise.

The prognosis depends chiefly upon the lesions. Thus catarrh and condylomata usually get quite well, and so do also gummata if actively treated. The dangers of gummata arise from the stenosis which may be produced by the inflammatory swelling which accompanies them, or from the ulceration and the perichondritis it may set up. When cicatrization occurs, the prognosis depends upon the amount of stenosis produced.

Treatment.—The ordinary general antisyphilitic treatment must be actively and steadily pursued. Of local remedies the direct application of *ether*, *calomel* or *iodoform* in powder, of weak solutions of *perchloride of mercury* or of *iodine in iodide of potassium* (one part each) with *glycerine* (five parts) are among the most useful.

Stenosis may require tracheotomy, after which the stricture may be dilated, with or without previous incision (*cf.* Simple Stricture).

Syphilis of the Trachea and Bronchi.—Syphilis may possibly lead to catarrh of the trachea and bronchi as it does of the larynx, but of this we know nothing definite; nor is more known of lesions corresponding with condyloma gummata, *i.e.*, syphilitic tumours are also very rare. Two good instances have been recorded, one by Lucas,² the other by Semon.³ In both cases the tumour was detected by the laryngoscope, diagnosed, and watched till it completely disappeared under treatment. Other cases, probably of this kind, are also described by Carrington and Charlewood Turner.

In Carrington's case⁴ a localised ulcer in the trachea had perforated the œsophagus and caused death by the passage of food into the lungs.

In Turner's case⁵ death was due to hæmoptysis. An ulcer was found in the trachea just above the right bronchus which had perforated into the superior vena cava, while a second ulcer of similar character was present in the trachea an inch and a half higher up. Both lesions were regarded as gummata.

As a rule syphilis of the trachea and bronchi takes the form of a diffuse superficial infiltration which rapidly breaks down and leads to extensive ulceration. The swelling is rarely sufficient to produce much stenosis of itself, and this is true also of the ulceration which follows, so that the symptoms may be those only of subacute or chronic tracheitis or bronchitis with more or less profuse purulent and occasionally blood-stained expectoration. The ulceration may extend throughout the whole trachea and even into the main bronchi, but it is

¹ *Klinik*, 413.

² *Brit. Med. Jour.*, 1887, 1378.

³ *St. Thos. Hosp. Rep.*, xiii.

⁴ *Path. Soc. Trans.*, xxxvi. 98.

⁵ *Ibid.*, xxxvi. 117.

hardly ever uniformly distributed; it usually occurs in irregular patches or lines, and its favourite place is close to the bifurcation.

The bronchi are rarely affected except by extension from the trachea, though a few cases of independent syphilitic ulceration in them have been recorded.¹

When the upper part of the trachea is affected it is nearly always associated with laryngeal syphilis. The ulceration, wherever it occurs, is extremely likely to set up perichondritis, and if an abscess form, it opens commonly into the tubes, though it may burst into the tissues round them, setting up diffuse suppuration or emphysema, or into neighbouring organs—such as the œsophagus, pulmonary artery, vena cava, or even aorta.²

In Kelly's remarkable case (*Path. Soc. Tr.*, xxiii. 45) a syphilitic ulcer of the right bronchus opened into the pulmonary artery, causing fatal hæmoptysis. A similar case is recorded by Watson.³

If the rings necrose they may be exfoliated and expectorated. When they have been got rid of in this way, syphilitic perichondritis has a fair chance of healing, as in the larynx.

Actual syphilitic ulceration of the trachea and bronchi is, however, rarely seen, and our knowledge of its frequency and distribution is derived chiefly from the scars left behind it. Such scars are common enough and often of considerable extent and are not infrequently found when their existence had not been suspected.

Syphilitic cicatrices in the trachea and bronchi as elsewhere undergo great contraction. When the scars are small and in the long axis of the tubes little effect may be produced. Secondary changes are often set up in the cartilages; thus as the scar contracts they may be brought close together and even made to overlap, and they not infrequently atrophy or calcify. If the scar be transverse, an annular stricture will be the result and a serious or fatal stenosis follow.

The frequency of scars and the paucity of records of ulceration prove, I think, that on the one hand syphilis of the trachea and bronchi is not altogether so rare as commonly stated, that, on the other hand, it often gives rise to no definite symptoms and usually heals.

Treatment.—The chief difficulty in the way of treatment of syphilis of the trachea and bronchi lies in its diagnosis. When once recognised the general treatment is clear; the lesions are rarely within reach of local remedies; for stenosis when it occurs there is little if anything to be done, for it is in most cases due to cicatricial tissue and is beyond the reach of remedies either medical or surgical.

16. CICATRICAL STRICTURE OF THE AIR-TUBES.

With few exceptions this is the result of past syphilitic ulceration, especially if, as so frequently happens, the ulceration has been associated with necrosis and exfoliation of cartilage.

The healing of syphilitic ulcers is characterised by three great peculiarities—(1) the massive production of new connective tissue which often takes place; (2) the remarkable power of contraction which the scars exhibit; and (3) the strange

¹ Wilks, *Guy's Hosp. Rep.*, 1863.

² Solis Cohen, *J. of Lar. and Rhinol.*, 1888, 398.

³ *New York Jour. of Med.*, 1873, i. 57; cf. Spec. Roy. Coll. Surg. Mus. 3500b.

tendency that contiguous ulcerated surfaces have to form adhesions to each other.

These peculiarities explain why syphilis is so common a cause of stenosis in the air-passages.

I. OF THE LARYNX.—Syphilitic scars are common in the region of the epiglottis and in the aryteno-epiglottidean folds, and as they contract may lead to great distortion of the parts, even so as to produce an apparent narrowing of the entrance into the larynx; but these deformities rarely produce actual obstruction, unless the parts about the glottis are also involved. When this occurs the entrance into the larynx may be reduced to a narrow vertical chink. This form of syphilitic stenosis is rare.

In the commoner form the scar tissue develops round the glottis, usually at the level of the true cords, which are themselves involved and take part in the stricture, but occasionally both above and below them. The orifice of the glottis may then be reduced to a small round hole, sometimes not larger than will admit a quill-pen.

Simple stricture of the larynx has been observed as the consequence of severe burns, diphtheria, typhoid fever, glanders, and measles.¹

Symptoms.—The symptoms are those of chronic laryngitis with those of laryngeal obstruction superadded. They are often extreme and liable to sudden and severe exacerbations, so that without warning urgent dyspnoea may arise, and if not relieved be rapidly fatal.

For this reason when stenosis is advanced tracheotomy is usually performed early as a precautionary measure, and as a preliminary to the actual treatment of the stricture itself.

Treatment.—When the immediate symptoms have been relieved by tracheotomy, the stricture may be dilated with or without previous incision by the passage of bougies or by the use of graduated cones, as Schroetter advocated. In many cases the stricture has been stretched and relief given by such means, but it is rare for the patient to be able to dispense with the tube, so that in most cases it has to be worn for the rest of life.

Webs.—The most remarkable, but at the same time the rarest form of syphilitic stenosis, is that in which a web is formed by the cohesion of ulcerated surfaces on opposite sides of the larynx. In the majority of cases, as would be expected, the cohesion takes place between the true cords, but in others between the false cords, and in a few below the true cords in the subglottic region.

The web develops from before backwards, that is to say, it is the anterior portions of the cord which are first involved. The aperture through which respiration takes place is posterior and usually sickle-shaped.

This cohesion may develop with very great rapidity. Thus, Rossbach observed the cords to become adherent to the extent of two-thirds in a period of eight days, and Sommerbrodt² to the extent of one-third in fourteen days.

Webs in the larynx are almost invariably due to syphilis, but a few are of traumatic origin and follow wounds, especially those below the cords (Rossbach).³

Congenital webs.—In a few instances the web is congenital and due to defective development. In the foetus the larynx is at first closed and the glottis is formed by a split which forms in the middle line posteriorly and spreads

¹ Demme, *Wörzb. med. Zetsch.*, ii. and iii. G. H. Mackenzie, *Edin. Med. Jour.*, xxix. 317.

² *Berl. klin. Woch.*, 1878, 175.

³ Langenbeck, *Archiv.*, ix. 491.

forwards;¹ so that, if this division were arrested, it would leave a web such as is met with in these cases.

The congenital web² is a thin membrane, usually extending between the vocal cords from the anterior part for a greater or lesser distance backwards, and may involve as much as two-thirds of the glottis, ending in a concave, rounded edge posteriorly. A second web of similar kind has been observed above this between the false vocal cords or just below them.

The **symptoms** produced by such webs are, of course, those of stenosis, but often not to the degree which might be anticipated.

The only **treatment** practicable consists in division by the knife or cautery with subsequent dilatation;

this treatment has been practised successfully. In one case the membrane has been resected in its entirety (Elsberg).

Bruns³ records a case, dilated and cured in 18 months, and gives references to thirteen cases collected from literature.

2. OF THE TRACHEA AND BRONCHI.—Although syphilitic cicatrization is much rarer in the trachea and bronchi than in the larynx, still it seems more frequently to exist without symptoms. The scars which produce no symptoms are usually in the long axis of the tube, and, though they may produce much deformity, do not lead to stenosis. They are often small, but may be very numerous and widespread.

When the cicatrix is transverse, as it must be after exfoliation of cartilage, stenosis is the necessary result, and the contraction is aggravated, as in the larynx, by the massive formation of new connective tissue.

In both trachea and bronchi the scar tissue may occupy the whole transverse section of the tube, involving all the coats and even the parts outside, but occasionally the chief seat of cicatrization is internal to the cartilages.

In the trachea the favourite seat of syphilitic stenosis is just above the bifurcation, and it then usually involves also one bronchus or both, especially the left. If the stenosis exist in the trachea alone, its favourite seat is in the middle portion. In the upper part it is much rarer, and is then almost invariably associated with cicatrization in the larynx.

It is possible for one or both bronchi to be stenosed while the trachea remains free, but this is very unusual. Still rarer is it to meet with cicatricial stenosis of the small bronchi, though a case of this kind is recorded by Lancereaux.

Rolleston⁴ records a case of stenosis of both bronchi at their origin due to cicatricial tissue, which had arisen in connection with an enlarged bronchial gland. There was no evidence of syphilis or tubercle. It occurred in a female aged 50, who had suffered from cough for four years, and was admitted with stridor.

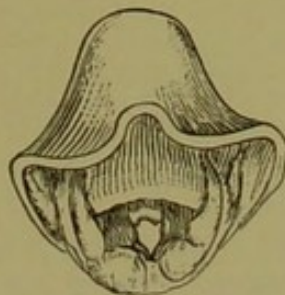


Fig. 25.

Congenital web. In this there were two—the one between the aryteno-epiglottic folds, the other below this, between the vocal cords. (Heymann, *Handb. d. Laryngol.*, p. 527.)

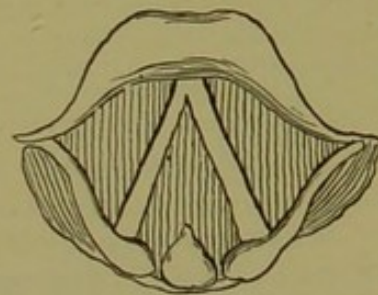


Fig. 26.

Syphilitic web between the vocal cords (acquired). (Heymann, *Handb. d. Laryngol.*, p. 527.)

¹ Seifert, *Sitz. d. Phys. Med. Ges. zu Würzburg*, 1888, No. 2.

² Heymann, *Handb. d. Laryngol. and Rhinosc.*, vol. i. p. 526.

³ *Arch. f. Laryngol.*, 1893, vol. i. pt. 1.

⁴ *Path. Soc. Trans.*, 1894, p. 23.

A very complete case of the common kind is described by Sydney Jones.¹ A patient 31 years of age expectorated 2½ years before death several complete rings of the trachea. When he died a dense cicatrix was found in the trachea 2 inches above the bifurcation, and from this part the rings had completely disappeared. Both bronchi were stenosed, the left most, which for the distance of an inch was so narrow as to admit with difficulty a No. 12 catheter.

A similar case is recorded by Gibb.² It is remarkable for the length of the stricture, which extended from the middle of the trachea to the bifurcation, and involved both bronchi. The case is interesting further as having been associated with a growth on the right side of the larynx in the region of the false cords, which may probably have been a syphilitic papilloma.

This case was described as one of simple fibrous stricture, but there is little doubt that it would have been called in the present day syphilitic.

If one bronchus only be affected the stricture may be so considerable that a fine probe will hardly pass through it.

Whether complete obliteration could ever occur in a main bronchus is open to doubt, but in the small bronchi it is of course possible, though very rare (Ratgen).³

There is, of course, nothing in the scar itself which is pathognomonic of syphilis, but the diagnosis is usually clear from the history of the case, the distribution of the lesions, and the evidence of syphilis elsewhere.

Thus, Goodhart⁴ records a case in which the whole trachea was puckered with scars and both bronchi stenosed. These lesions were associated with fibroid degeneration of the heart, aneurysm of the thoracic aorta, and with nummular aortitis.

Syphilitic stenosis of the trachea and bronchi may also occur in congenital syphilis.

Thus, in Parker's⁵ case, which is perhaps the most interesting on record, a child had tracheotomy performed at the age of 7 years for what was believed to be syphilitic laryngitis. He died at 15 with the symptoms of tracheal stenosis. The trachea for an inch and a half from the bifurcation was reduced to less than a quarter of an inch in diameter and the left main bronchus was almost completely occluded. The section showed that the greater part of the new tissue was internal to the cartilages.

Of the few non-syphilitic cases tracheal stenosis is described by Lublinski⁶ and Korti⁷ as following a tracheotomy wound.⁸

Anything corresponding with a web across the trachea is almost unknown, but Turck⁹ describes and figures one such case which occurred in a lad of 18 years of age who had suffered with dyspnoea since the age of 12, and in whom no evidence of syphilis could be obtained.

Syphilitic stenosis of the bronchi alone is extremely rare. According to the literature given by Rolleston and Ogle¹⁰ there are only 10 cases recorded, in 7 of which both bronchi were affected.

The stenosis is sometimes so extreme that it is difficult to understand how life can have been maintained.

In one case the right bronchus admitted only a fine probe, and the left a No. 8 catheter.

Of 9 cases, 5 were in men and 4 in women.

The average age was 36 years; the youngest occurred in a woman of 25, and the oldest in a woman of 75.

Diagnosis.—It is not difficult to diagnose tracheal or bronchial obstruction, but to determine whether it is due to syphilitic stenosis is often attended with insuperable difficulties. Still the diagnosis has been frequently successfully made from the history and general features of the case.

¹ *Path. Soc. Trans.*, x. p. 60.

² *Virch. Arch.*, xxxviii.

³ *Ibid.*, xxxvii. 19.

⁴ Langenbeck, *Archiv.*, 1881, 822.

⁵ *Krankh. d. Kehlkopfes*, p. 501.

⁶ *Ibid.*, xv. 90.

⁷ *Path. Soc. Trans.*, xxvi. 13.

⁸ *D. med. Woch.*, 1887, 554.

⁹ Roy. Coll. of Surg. Mus. Prep. 3512.

¹⁰ *Path. Soc. Trans.*, l.c.

Prognosis.—The prognosis is grave as regards life, for a sudden paroxysm of dyspnoea may at any moment occur and prove fatal, and, if the stricture continues to increase, life would soon become impossible.

Treatment.—Recovery is impossible, and the best that can be hoped is that the stricture should remain stationary. The affection is usually beyond all treatment, medical and surgical. Lublinski records a successful case of the dilatation of a tracheal stricture by catheterization after tracheotomy; but such treatment is rarely possible, and, considering the doubt that must in most cases attach to the diagnosis, forcible dilatation of this kind is attended with too great risk to be often attempted.

17. TUMOURS IN THE AIR-TUBES.

Tumours in the air-tubes derive their chief importance from the risk of respiratory obstruction which attends them. They are not uncommon in the larynx, but are extremely rare in the trachea or bronchi. They fall into two groups, according as they are of a benign or malignant nature.

I.—IN THE LARYNX.

I. NON-MALIGNANT.—Of the non-malignant tumours of the larynx, by far the commonest are papillomata and fibromata.

Papillomata are wart-like tumours arising usually from the true cords or between them anteriorly. Sometimes they are connected with the false cords or with the epiglottis, but they are hardly ever found in the arytenoid region. They vary much in size, from that of a mustard seed up to that of a walnut, but are usually not larger than a split pea. Though sometimes single they are more frequently multiple, and then perhaps symmetrically placed, or they may be very numerous and spread over a considerable surface. They may be of conical or bulbous shape, or of cauliflower growth. They are generally pink in colour and soft in texture. They have been sometimes detached in fragments or *en masse*, and expectorated.

Fibromata are rarely found except springing from the true cords. They are of altogether larger average size, varying from that of a pea to that of a hazel nut, or even a walnut. They may be round or pear-shaped, sessile or pedunculated, smooth or occasionally lobulated; they are usually whitish in colour and hard in texture, but are sometimes soft. When pedunculated they may hang down below the cords, and only appear above them on coughing.

These two tumours constitute by far the majority of benign growths in the larynx—papillomata, about 60 per cent., fibromata, about 20 per cent. They are often stated to be the result of long-standing laryngeal irritation, and to be met with chiefly in the subjects of chronic catarrh, and among those who have to use their voice much; but these statements Semon disputes with justice. Though commonest in males and in adult or middle life, they have, however, been found in quite young children, and some are undoubtedly congenital.

Lore¹ records three cases at the ages of M. 2½, F. 3½, and M. 6 years respectively, in two of which operation was successful, but in one the tumour recurred. Johnson² also records a case in a boy of 3 years. Other cases are referred to by Bosworth.

¹ *Jahresb. f. Kinderheilk.*, xv. 126. *Arch. of Laryngol.*, i. 58.

² *Dis. of Nose and Throat*, p. 719.

Besides these common forms of benign tumour, others of great rarity are described, such as *myxoma*, *angioma*, *lipoma*, *adenoma*, *ecchondroma*, and *cysts*.

Cysts are very rare. They may arise from the epiglottis, as in the cases recorded by Durham and by Moure,¹ but are sometimes found in other parts of the larynx, *e.g.*, in the subglottic region, as in the cases of Abercrombie,² or even arising from the vocal cords (Sommerbrodt).

Some of these cases are congenital, the child dying of suffocation when only a few days old (Abercrombie,³ 14 days; Edis,⁴ 37 hours). In Durham's and Moure's cases the children were both in the eighth year.

The cysts originate in the obstruction of the duct of a muciparous gland, and may reach a considerable size.

The **symptoms** of laryngeal tumours depend a great deal upon their situation and size.

When seated on the cords they are likely to affect the voice, which may be even lost.

Cough is an almost constant symptom as a consequence of the accompanying catarrh, or of the direct irritation of the tumour itself, which may even produce the feeling as of a foreign body being present in the throat.

Dyspnœa is not constant; if the tumour be small or so seated as not to reach the glottis there may be no dyspnœa at all. If there be dyspnœa it varies greatly at different times, and is often produced or aggravated by certain positions or movements, and sometimes by coughing, the tumour in these ways reaching the glottis and producing obstruction, which may sometimes be sufficient to lead to sudden suffocation.

Diagnosis.—Although the existence of tumour may sometimes be suspected from the symptoms, a certain diagnosis can only be made by means of the laryngoscope.

Eversion of the sacculus laryngis has been described as simulating tumour, but there are but few cases recorded.⁵

Prognosis.—Small tumours may exist for years with no special symptoms. The prognosis is in all cases uncertain, for severe dyspnœa may arise at any time and be fatal. The risks are greater in children than in adults on account of their greater liability to laryngitis and spasm, and because they are not as good subjects for tracheotomy if the necessity for it arise.

Treatment.—Spontaneous resolution has been described, but very rarely occurs.

If the tumour be small, direct applications are sometimes employed in the form of caustics or escharotics, but they rarely do any good, and are attended by much risk from the irritation and inflammation they are liable to set up.

Nothing therefore remains but removal by surgical means. External operations, *i.e.*, after thyrotomy, are practically abandoned now in favour of endolaryngeal operations, *i.e.*, through the mouth, the tumour being removed either by cutting or crushing forceps, by the guillotine, actual cautery, or the knife.

With a cyst it is usually enough to incise and cauterise its interior, after which it rarely refills. Papillomata are liable to recur after removal, but this seldom happens with fibromata.

¹ *Med. Chir. Tr.*, xlvii. *Gaz. des hôpitaux*, 1879, 125.

² *Path. Soc. Trans.*, xxxii. 34.

⁴ *Tr. Obstetr. Soc.*, xviii. 2.

³ *Virch. Jahrb.*, 1880, ii. 137.

⁵ Moxon, *Path. Soc. Trans.*, 1868.

2. IN THE COURSE OF SPECIFIC DISEASE.—1. *Tubercular tumours.*—Tuberculosis of the larynx is sometimes attended by the formation of a tumour, which may precede any other manifestation of tubercle in the larynx, or even any detectible disease in the lung. Rare as these cases are, still a fair number have been now recorded, and, in many cases, their tubercular nature has been conclusively proved by the demonstration in them of the tubercle bacillus.

Besides these tumours, which are frequently attached to the vocal cords, vegetations or papilloma-like growths are not uncommon in the arytenoid region. This is a part of the larynx in which papillomata are never seen, so that vegetations in this position are almost pathognomonic; still it is often very difficult to determine with the laryngoscope that the vegetations are not the infiltrated edges of tubercular ulcer (*cf.* Tubercle).

Closely allied to tubercle are *lupus* and *leprosy*, which, however, rarely give rise to anything that can be fairly called a tumour; the swelling is rather a diffuse, irregular infiltration, similar to that which occurs in the common forms of tubercle and syphilis.

2. *Syphilitic tumours* of the larynx are of two kinds, viz. (1) gumma and (2) papilloma, *i.e.*, a simple tumour, the result of the chronic catarrh (*cf.* Syphilis).

3. MALIGNANT.—The malignant tumours of the larynx are two, viz., sarcoma and carcinoma.

Sarcoma.—Sarcoma is very rare.¹ It is usually of the spindle-celled variety, and grows on or near the vocal cords. In the early stage it is not to be distinguished from a simple tumour, but its rapid growth quickly discloses its nature.

Carcinoma.—Carcinoma, though rare in relation to cancer elsewhere in the body, is not so rare in relation to other laryngeal growths. It occurs nearly four times more frequently in men than in women, and as a rule after the age of 40, though cases are recorded before 20. The larynx may, of course, become secondarily involved by extension from some primary growth in the vicinity, *e.g.*, in the pharynx, œsophagus, or neck, but cancer is more important when it arises as a primary growth within the larynx itself.

Primary cancer commonly starts in the false cords or ventricles or from the true cords.

Semon's figures are :—Vocal cords,	15
Ventricular bands,	3
Ventricle of Morgagni,	2
Not to be made out with certainty,	35
	<hr/>
	55

It may form a localised tumour, but being usually of the epitheliomatous type, it more frequently presents itself as a superficial infiltration. Ulceration takes place early, and gives rise to a fetid discharge, which is often blood-stained.

The ulceration slowly spreads until, it may be, the whole interior of the larynx is destroyed. Sooner or later the cartilages become involved, and perichondritis is set up with its customary consequences.

If the cartilages necrose, parts of them may exfoliate and be expectorated, but they are more commonly destroyed by a process of gradual erosion or molecular disintegration.

I recorded in the *Path. Soc. Trans.* for 1887 a case in which practically the whole thyroid cartilage had disappeared.

The growth may, for a long time, be entirely confined within the larynx, so that there is no external swelling nor even any glandular enlargement, unless perichondritis develop. The suppuration that then follows generally discharges

¹ Butlin, *Malignant Diseases of the Larynx*, 1883.

itself through the larynx, and not externally, unless an incision has been made or a communication formed with the tracheotomy wound. It may also perforate the œsophagus, and discharge itself in this way.

Secondary growths in other parts of the body, unless it be in the lungs, are rare, and the cancerous cachexia may be absent even to the end.

This absence of cachexia, as well as of glandular enlargement, is due to the peculiar arrangement of the lymphatics of the larynx, which are isolated, have no anastomosis with the neighbouring lymphatics, and terminate in two small glands, one beneath the greater corner of the hyoid bone and the other at the side of the trachea.

The Symptoms are by no means characteristic, being for some time those only of chronic laryngitis with cough and more or less hoarseness according to the situation of the growth.

Dyspnœa is frequently due to the cancerous infiltration, and is then a late symptom, though it may arise at any time as the result of inflammatory swelling or of perichondritis.

Pain is a common symptom, and being, in the absence of perichondritis, rare in other laryngeal affections, it becomes of some diagnostic importance. In the early stage, besides being felt in the larynx it is often described as darting up to the ear.

As soon as ulceration sets in, an abundant discharge becomes established, which is often fetid and blood-stained, while in some cases the hæmorrhage has been profuse and even fatal.¹

When perichondritis occurs the dyspnœa may become severe and necessitate tracheotomy.

Lymphatic enlargement is generally absent at first, that is to say, for the first six or even twelve months, and may be absent throughout.

Cachexia is often long absent, and may not be at any time extreme or more than could be explained by the constant fret of the various harassing symptoms.

When the epiglottic or arytenoid regions become involved, pain and difficulty in swallowing arise, and food, especially liquids, pass into the larynx, so that patients dread the taking of food and require to be fed with an œsophageal tube.

As the result of the passage of irritating discharges or of portions of food down the air-tubes, bronchitis pneumonia or gangrene of the lung may be produced and lead to death. The condition of the patient towards the end of life is miserable to the last degree, and death is often welcomed as a happy release.

Diagnosis.—In the tumour stage the diagnosis has to be made from other simple tumours or from gumma. But it may be generally assumed that a swelling in or near one of the ventricular bands in a patient after middle life, in whom there is no history of syphilis or chronic laryngitis, is in all probability cancerous. In the ulcerative stage the diagnosis has to be made from other forms of laryngeal ulceration, especially tubercular and syphilitic. Tubercular disease occurs as a rule earlier in life and is accompanied with evidence of tubercle elsewhere.

In the case of syphilis the difficulties are greater, but marked and continued improvement under antisyphilitic treatment, and especially the evidence of cicatrization in some part, is conclusive.

Semon, however, states that he has seen temporary improvement take place in cancer under the use of iodide of potassium.

When perichondritis is present the symptoms of that affection may so predominate that it may be difficult to determine the cause to which it is due.

¹ Gottstein, *Krankh. d. Kehlkopfes*, 1888, p. 164.

The Prognosis is of course as bad as it can be. Recovery is impossible if the disease cannot be removed, and the only question to determine is the probable duration of life.

The disease rarely lasts so long as two or three years from the commencement of laryngeal symptoms, the average duration being not more than twelve to eighteen months, and though cases are recorded of much longer duration, it is hard to believe that the disease was really cancerous all the time.

The longest well-authenticated case, according to Semon, was one of Fauvel's, which lasted 6½ years, and in a case of his own in which the duration was 4½ years.

The end may come in various ways. Sudden suffocation, sometimes due among other causes to œdema of the glottis, is a risk to which the patient is always liable as long as tracheotomy has not been performed. When this danger has been obviated by tracheotomy, the patient may die of gradual exhaustion, but more frequently death is produced by bronchitis, pneumonia, or gangrene of the lung, set up by the passage of food and secretions into the lungs.

A point of some interest is the dissemination in the lung of the new growths, consequent, no doubt, upon the aspiration of portions of the growth into the lung and their growth within the smaller air-tubes (*cf.* New Growths in the Lung).

The Treatment is general and chiefly palliative, directed to support the strength and to relieve distressing symptoms as they arise. Among these remedies may be included tracheotomy, which is sure to become necessary sooner or later; but necessary as this operation is to prolong life, it often proves to be an additional source of discomfort from the irritation which the tube produces, and from the unhealthy sloughing and suppuration which take place in and around the wound. Remedial treatment consists in operations for the removal of the disease. Of these there are two, viz., erosion after thyrotomy and extirpation of the larynx.

Erosion is, of course, only practicable when the disease is limited in extent, and until lately the results have not been satisfactory, but the recent experience of Butlin¹ and Semon² is remarkable.

Of 16 cases of Semon's, recovery took place in 9, *i.e.*, 56 per cent., and in some of them the duration of life was considerable, 7½ years in one and 6 years in another, both these cases dying of other diseases. In 4 other cases the patients were alive and well 4 years, 3 years, 2 years, and 1½ years after operation.

Extirpation of the larynx is a fearful operation to contemplate, and has only been successful in something less than 10 per cent. of recorded cases.

Recovery took place in 13 cases out of 138,³ the patients being known to be alive some time after the operation. Three cases lived more than 16 months, 2 more than 2 years, 5 more than 3 years, 2 more than 5 years, while in Harne's case the patient was well 8 years later.

The operation was first performed by Patrick Watson in 1866, and again by Billroth in 1875, in both instances for cancer.

II.—IN THE TRACHEA.

Tumours of the air-tubes below the larynx are extremely rare, but they are of the same kinds as those described in the larynx.

I. NON-MALIGNANT TUMOURS.—Simple *papillomata* or *polypi* have been described by various authorities. They appear to be generally smooth papillary or occasionally cauliflower growths, sometimes sessile, sometimes pedunculated.

¹ *Operative Surgery of Malignant Disease.*

² *Radical Operation for Malign. Dis. of Larynx*, 1894. Article in Clifford Allbutt's *System of Med.*, iv. 839.

³ Bosworth, *l.c.*, 757.

They have been several times diagnosed by the laryngoscope, and in one case even removed by operation through the mouth (Schroetter).¹

In Fifield's² remarkable case a fleshy polyp attached to the trachea hung down over the right bronchus, which it occasionally completely occluded. The patient was a young woman of seventeen, who suffered greatly from asthma, as it was thought, and died of exhaustion.

In Türck's³ case a fibroid tumour was found in the anterior wall of the trachea in a patient who died of phthisis. Gibb⁴ describes a cyst or abscess in the anterior wall which burst spontaneously and healed.

Perhaps the most important tumours of this kind are those described as *post-tracheotomic vegetations*. Fleshy vegetations are common enough, both internal and external, while the tube is being worn, and besides being the cause of pain, irritation, and bleeding, may, when numerous within the trachea, also produce so much obstruction as to prevent the tube being dispensed with. Post-tracheotomic vegetations, however, occur also after the wound has cicatrized. They are found especially in children, and usually in males, and not before the 15th day or after the 2nd month from the healing of the wound.⁵ They cannot of course be diagnosed till the symptoms of respiratory obstruction arise, but they have then been correctly diagnosed and successfully operated upon.

Ecchondromata have been described, but are extremely rare. They are connected with the cartilages. Cohen⁶ records a case in which there were several such tumours found in the trachea of a patient who died of phthisis; they had produced no symptoms (New Growths of the Lung).

Gummata, as already described under syphilis, may form distinct tumours, and have been diagnosed during life and successfully treated.

I do not know of any case of tubercular tumour of the trachea or bronchi similar to those which have been met with in the larynx.

2. MALIGNANT TUMOURS.—When the trachea is the seat of malignant mischief it is, as a rule, involved only secondarily by the direct extension to it of a primary growth in the neighbourhood, as for instance from the œsophagus, thyroid or glands of the neck or mediastinum, but there are a few cases in which the growth clearly originated within the trachea itself.

Of *sarcoma*, two cases are recorded by Schroetter⁷ and another by Meyer-Huni and Kaufmann.⁸ In the last the trachea was opened and the tumour removed. The patient was a man of twenty-seven years of age and made a good recovery, but it is hard to believe that this was really a malignant tumour at all. *Primary cancer* is very rare indeed.

The first and for a long time the only case on record was that of Langhans,⁹ where the tumour involved the anterior wall of the trachea and extended a little distance down both bronchi. Two cases have been recorded by Mackenzie.¹⁰ In the first, a man of 40, the growth occupied the same situation, and like the preceding was not ulcerated. In the second case, a woman of 57, a cancerous ulcer occupied the middle third of the trachea on its posterior surface. A similar case is recorded by Virchow and Gerhardt¹¹ in a woman of 38, and another by Morra. The most recent case came under Handford's¹² observation.

Some recent cases, with a review, will be found described by Oestreich, *Ztscht. klin. Med.*, xxviii. 383; Beschorner, *Samml. klin. Vortr.*, 1893, 73.

¹ *Wien med. Jahrb.*, xv., Sept. 1, 1868, p. 64.

² *Boston Surg. and Med. J.*, 1861, Nov. 14.

³ *Loc. cit.*, p. 502.

⁴ Petel, "*Des polypes de la Trachée*," Paris, 1879. The most complete monograph on this subject.

⁵ *Dis. of Throat*, 2nd ed., p. 511.

⁶ *Corresp. bl. f. Schweiz. Artzl.*, 1880, No. 9.

⁷ *Loc. cit.*

⁸ *Berl. klin. Woch.*, 1887, 933.

⁹ *Larynx*, p. 392.

¹⁰ *Laryngol. Mittheil.*, 1875, 102.

¹¹ *Virch. Arch.*, liii. 470.

¹² *Path. Soc. Trans.*, vol. xl.

Secondary metastatic growths appear to be unusual, although in Virchow's case some nodules existed in the lungs, and in one of Mackenzie's the neighbouring glands were infiltrated.

In all the cases the growth seems to have originated in the mucous and sub-mucous coats, and in several was entirely confined to them. When the neighbouring tissues are infiltrated the difficulty consists in determining that the growth did not arise primarily in the peritracheal or peribronchial lymphatic tissue or in the adjacent glands.

The disease can only very rarely be recognised during life, and when diagnosed admits of no remedial treatment.

III.—IN THE BRONCHI.

Primary tumours of the bronchi are extremely rare, and are of two kinds only—**enchondroma** and **carcinoma**. These are both dealt with later in connection with tumours of the lung.

18. OBSTRUCTION TO THE AIR-TUBES FROM WITHOUT.

Compression is the term used to denote obstruction to the air-tubes produced by pressure from without.

The cases fall naturally into two groups, according as the part of the air-tubes involved lies in the thorax or in the neck. In the former group are found the most frequent as well as the most serious forms of the affection, for the air-tubes within the thorax, being placed between two rigid structures, viz., the spine behind and the sternum in front, are easily compressed by even small tumours or swellings near to them; while in the neck, owing to the feeble resistance offered by the skin and other tissues, the air-tubes may escape compression even by tumours or swellings of considerable dimensions.

(A.)—COMPRESSION IN THE NECK.

The larynx with its stiff cartilages is difficult to compress, and unless some actual lesion be produced, such as fracture or dislocation, pressure from without is hardly capable of causing obstruction.

In the trachea, on the other hand, the cartilages are less resistant, and what is more important, the rings are incomplete, so that they readily yield to lateral pressure, while in the membranous portion, placed as it is posteriorly and separated only by the œsophagus from the spine, any pressure from behind will produce its full effect, so that slight lesions here may lead to severe obstruction.

The causes of compression in the neck are affections of the thyroid gland, tumours, new-growths, enlarged glands, inflammation or hæmorrhage in the tissues of the neck, disease of the cervical spine, and certain affections of the œsophagus.

I. AFFECTIONS OF THE THYROID GLAND.—Embracing almost completely the trachea as it does, almost any affection of the thyroid may lead to pressure, but the commonest and the most important of all is the ordinary bronchocele.

Bronchocele.—It is by no means necessarily the largest bronchoceles that produce the most pressure; more depends upon the direction of growth than upon the size, for if the goitre develop forwards, as it often does, little discom-

fort may be produced by it, except that due to its weight, while a single small cyst developing inwards, especially if situated towards the posterior part of the trachea, may produce severe symptoms; and still more if, as sometimes happens, the growth makes its way between the trachea and the œsophagus so as to press upon the membranous portion. When the enlargement of the gland is uniform, the trachea is gripped between the lobes and pinched laterally, but it often

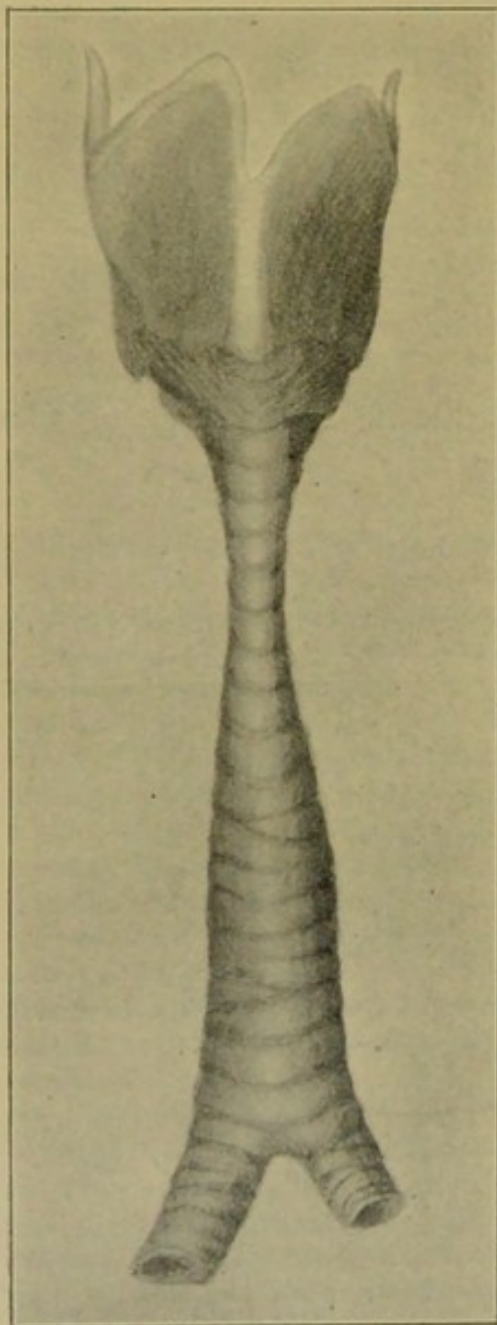


Fig. 27.

Bilateral compression or pinching of the trachea, produced by a goitre (Berry).

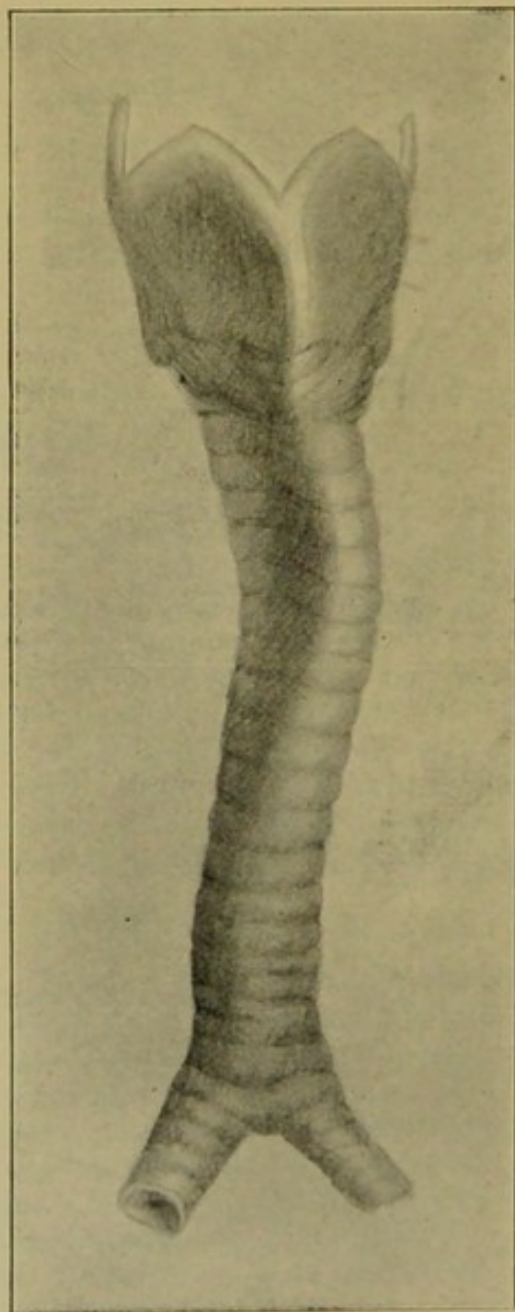


Fig. 28.

Displacement and lateral compression of the trachea, produced by unilateral goitre (Berry).

happens that a goitre is free of all pressure symptoms until it extends downwards so as to get behind the clavicle or sternum, and it is in these cases that the most severe dyspnœa is met with.

The shape which the compressed trachea takes in goitre depends upon the direction of pressure; thus in a uniform enlargement of the gland the sides are pinched together and the trachea is flattened laterally; if the enlargement be

unilateral the trachea may be bulged inwards on that side, and in extreme cases even pushed out of the middle line of the neck, sometimes to such an extent as to lead to a sharp bending or kinking at the level of the cricoid, which may still further increase the obstruction. Beneath the sternum the pressure may be from the front and the trachea be then flattened in the antero-posterior direction.

The cartilages of the trachea pressed upon have been stated to undergo atrophic changes, which diminish the resistance of the trachea to such an extent that it cannot stand upright when removed from the body, but bends at the seat of pressure. It is to these changes that many of the cases of sudden death in goitre have been referred.¹

By other authors the dyspnœa has been referred to muscular action, the muscles over the enlarged thyroid being irritated and kept in a state of tension, thus pressing the gland back upon the trachea. Acting on this theory the muscles have been divided, but, as a rule, without relief to the dyspnœa. Although the muscles have in a few cases been found hypertrophied, in most they are atrophied and spread platysma-like over the goitre, a fact which disposes of the theory, at any rate for all but exceptional cases.

When the goitre passes behind the sternum or clavicle, the mechanism of the dyspnœa is clear. When it does not, the dyspnœa is due to compression of the trachea either laterally, where the enlargement is uniform, or locally, where the enlargement is partial.

The dyspnœa is liable to exacerbations as the result of catarrh of the mucous membrane in the region of the obstruction or to the accumulation of secretion there. Rapid aggravation of the dyspnœa is generally due to some sudden increase in the compression consequent on hæmorrhage or inflammation in the cysts.

Bristowe² records a case in which a patient was suddenly seized with severe dyspnœa. A small thyroid cyst which was found on examination was quickly punctured, and with the escape of its contents the dyspnœa was immediately relieved.

Compression due to other affections of the thyroid are rare. Most of them occur in the course of malignant disease. This, while producing pressure, also very early ulcerates through into the trachea.

In a case of this kind recently seen by me, the thyroid formed a hard mass of no large size. The patient came under treatment on account of repeated hæmoptysis, in an attack of which he ultimately died.

Apart from bronchocele, other affections of the thyroid, such as inflammation, abscess and hæmorrhage, are extremely rare as causes of obstruction.³ A few cases of hydatids in the thyroid have been described, and a very remarkable case of accessory thyroid⁴ recorded which extended from the chin to the sternum, and was the cause of serious obstruction.

Exophthalmic Goitre is commonly associated with dyspnœa, but unless the gland has passed beneath the sternum the explanation is to be generally sought in other causes than pressure, and will be found rather in the nervous or vascular conditions present. Still in the few cases where the dyspnœa is due directly to the goitre it is brought about in the same way as in bronchocele by the gripping of the trachea by the enlarged gland.

Aërial Goitre, Luftkropf—Aërocele.—In connection with goitre reference may be made to the rare but remarkable affection which has been named *aërial goitre* or *aërial bronchocele* (Luftkropf), and which has been also called *Tracheocele*, *Pneumatocele*, *Hernia of the Trachea* and *Aërocele*.

¹ Edw. Rose, *Arch. f. klin. Chir.*, vol. xxii.

² *St. Thos. Hosp. Rep.*, iii.

³ Wilks and Moxon, *Path. Anat.*, 2nd ed., p. 290. Gurlt, *Cystengeschw. des Halses*, 1855.

⁴ Schnitzler, *Wiener Klinik*, 1877.

This consists of a tumour, varying usually with respiration, increasing with expiration and sometimes almost completely disappearing on inspiration, often tympanitic when distended, but at other times not resonant to percussion. It is found in the thyroid region, and is sometimes bilateral, sometimes median, but most frequently unilateral. Its walls resemble mucous membrane, and the contents are mucous or mucopurulent. These tumours are sometimes congenital, but more often develop as the result of violent coughing, vomiting, or straining.

The symptoms are unimportant, for dyspnoea is rare, but it may be severe, as in a case recorded by Eldridge,¹ in whose paper the literature of the subject is given.

The congenital cases are rarely cured, but the acquired may disappear spontaneously. Mechanical pressure has been employed. Extirpation is a risky proceeding, and the remedy may prove worse than the disease.

As to the exact pathological nature of these cases, much difference of opinion exists. Many varieties of **air-containing tumours** in the neck have been described.

Some appear to have their origin in *abscesses* which have communicated with the pharynx, œsophagus or trachea. What follows, however, in these cases is usually cervical emphysema, and hardly ever anything that could be fairly described as an air-containing tumour.

Others, especially those of congenital origin, are referred to *persistent bronchial fistulæ*, the external orifice of which has become closed while the other part remains patent.

There is, I believe, no case of this kind recorded which can be considered conclusive.

True *aërial goitre*, i.e., an air-containing tumour of the thyroid, could only arise when a thyroid cyst has ruptured into the trachea. It is a most unlikely condition theoretically, and as a matter of fact it has never been conclusively demonstrated. This being so, it is odd that Aërial Goitre or Luftkropf should be the name by which the condition is commonly known.

It has been stated, but I do not know upon what grounds, that a *persistent thymus* may be forced out of the thorax by violent expiration and present as a tumour not unlike goitre in the neck.

Two varieties remain, viz., tracheal and œsophageal pouches. Both are well recognised, though rare, pathological conditions, and are no doubt the true explanation of most of the cases recorded.

Tracheal pouches (cystic tracheëctasis) were described and figured by Virchow. They arise from the posterior wall of the trachea, usually about the level of the manubrium sterni. As they increase in size they extend upwards and laterally, and may present above the clavicle and reach below and behind the thyroid gland, thus closely resembling a goitre.

It is stated that the communication with the trachea may become closed so that a cyst is left, lined with mucous membrane and containing mucus.

Tracheëctasis is in most instances a pathological curiosity—the pouches are small, and could not have been obvious during life.

Œsophageal pouches of small size are not altogether rare, but it must be rare indeed for so large a swelling to be produced which could in any way suggest a goitre. Such cases, however, undoubtedly exist, of which the following is an interesting example. It was under the care of Mr. Berry, to whom I am indebted for the opportunity of seeing it and for permission to refer to it.

¹ *Amer. Jour. of Med. Sc.*, July 1879. Cf. also Petit, *Rev. de Chirurgie*, 1890.

The patient was a woman 23 years of age, who complained of difficulty in swallowing and dyspnœa.

Her symptoms dated from the age of 18, when she began to have a feeling of tightness in the throat after swallowing, which was relieved by vomiting. These attacks used to come on about every two months at first, but gradually became more frequent, until she has recently found it impossible to take any food without vomiting. Lately the dyspnœa was brought on at once by lying down flat, while stooping forward brought on an attack of coughing.

When quiet the neck looked a little full, but otherwise normal; when coughing a large symmetrical tumour came up from the thorax into the neck, looking like a bronchocele. It reached as high as the hyoid bone and as far back as the posterior border of the sterno-mastoids. The trachea could be felt in front of it in the middle line, and the swelling was soft and resonant.

It was evidently an air-containing cyst, and seemed to me like what had been described as "Aërial Goitre" or "Luft-kropf."

On operation it was discovered to be an œsophageal dilatation, and part of it was removed. For a time the patient did well, but then began to vomit persistently. When no food could be retained gastrotomy was performed as a last resort, but the patient succumbed.

The *post-mortem* examination was incomplete; but the œsophagus was found dilated in its whole length, and there was no sphincter at the œsophageal opening into the stomach.

It seems as if the condition is to be referred to a congenital absence of the sphincter, so that the air which the stomach contained was forced by coughing, vomiting, or straining into the œsophagus, distending it, and in time leading to the formation of the remarkable tumour in the neck described.

2. EMPHYSEMA of the cervical tissues may be attended with severe dyspnœa. It is easily recognised by the crepitation beneath the skin, and by the resonance on percussion. The dyspnœa may be considerable, but this it is not easy to assign to its proper cause, for though in part caused by pressure round the air-tubes, it may be also due to mediastinal emphysema, to interstitial emphysema of the lung itself, or even to pneumothorax.

3. OF TUMOURS in the neck little need be said. Cancer, sarcoma, lymph-adenoma, cysts, enlarged and inflamed glands may all cause pressure, but whether they do or not depends entirely upon their position and direction of growth.

I have seen two cases in which a small abscess formed in the deep tissues just above the sternum in the middle line. It rapidly caused pressure on the trachea, and threatened to perforate it. In the situation they occupied it required some skill and courage to open them, but with the evacuation of a small amount only of pus, all the symptoms disappeared and recovery was rapid.

4. DIFFUSE PHLEGMONOUS INFLAMMATION of the neck sometimes kills by dyspnœa. This may be the result, on the one hand, of acute inflammation of the parts about the glottis or, on the other, of the collection of pus in such a position as to produce pressure on the air-tubes. The same explanation holds also of the inflammation round a foreign body like a bullet lodged in the neck.

5. NEW-GROWTH AND FOREIGN BODY are the only two affections of the œsophagus that are likely to lead to compression. A new-growth may push the trachea before it and cause compression, but more commonly it attacks the walls, and quickly ulcerating through them, forms a fistulous communication between the two tubes, with the result that food passes into the air-tubes, and excites pneumonia, of which the patient dies.

6. DISEASES OF THE CERVICAL SPINE.—New-growths in this position are rare, and still more rarely cause compression. Caries may be attended with suppuration and lead to compression as in some cases of retro-pharyngeal abscess; but the pus as a rule collects at the sides or posteriorly, and not towards the front, thus avoiding the trachea.

(B.)—COMPRESSION WITHIN THE THORAX.

Within the thorax the two commonest causes by far are aneurysm and new-growth.

1. ANEURYSM.—It is obvious that the aneurysms which are most likely to press upon the air-tubes are those of the arch of the aorta, but even among them there will be differences. In the first part of the arch an aneurysm tends to develop forwards and outwards, and though it compresses the upper part of the lung it avoids the large air-tubes. In the second part the aneurysm develops upwards and backwards and will press upon the trachea almost at once, and being in the narrowest part of the thorax it will produce its full effect; thus well-marked pressure symptoms have been caused by an aneurysm not larger than a filbert or a walnut, and have been the earliest or indeed the only signs of mischief until profuse or fatal hæmoptysis proved the nature of the lesion. In the same way the rapid development of a small secondary pouch in this position from a larger aneurysmal sac often explains the sudden aggravation of the dyspnœa. The same remarks apply to aneurysm of the innominate or right carotid developing in this position. In the third part of the arch aneurysms develop downwards and towards the left, and they thus not infrequently compress the left bronchus. Wherever the aneurysm exercises pressure it may burst, and where it is very small profuse hæmoptysis may be the first grave symptom.

In the descending part of the arch the direction of development is away from the root of the lung, and though the lower lobe of the left lung may be compressed, the air-tubes usually escape.

2. MEDIASTINAL TUMOUR.—Every mediastinal tumour is capable of exercising pressure upon the air-tubes, but it is especially the malignant forms that are important in this respect, for by their continuous growth they are certain to press upon the air-tubes sooner or later. How soon, however, will depend upon the place of development and the direction of growth. Dyspnœa in the course of mediastinal tumour is, however, not of itself sufficient to indicate pressure on the air-tubes, for it may be due to secondary deposits in the lung or to the direct spreading to the lung of the growth, to the compression of vessels, both arteries and veins, to pressure on nerves and consequent laryngeal affections, to compression sequelæ in the lung such as bronchitis or pneumonia, or lastly to pleuritic effusion.

In whatever part of the mediastinum the growth may have originally started, it ultimately reaches the trachea or main bronchi at the root of the lung and compresses them. Of the bronchi it may involve one or both, but, if both, rarely both equally.

The diagnosis of mediastinal tumour presents in many cases but little difficulty. The signs all point to some solid mass in the mediastinum. They are (1) dulness beneath the sternum or in the interscapular space, with increased vocal vibrations, vocal resonance and breath sounds over the dull area; (2) compression of veins shown by dilatation of the superficial veins over the chest or of the large veins in the neck or arm, with, not uncommonly, œdema of the corresponding parts, *i.e.*, of the head, neck or arm; (3) compression of arteries indicated by a difference in the carotid or radial pulses; (4) pressure on nerves as seen in various paralytic or spasmodic affections of the larynx; (5) and lastly by pressure on the air-tubes leading to stridor where the trachea is involved alone or to obstruction to the entry of air into the lung where the main bronchus is involved.

All these signs may be equally given by an aneurysm, but as a rule with aneurysm there is not as great an increase in the voice and breath sounds over the dull area as with solid tumour, and there are the more special and characteristic symptoms of aneurysm, viz., visible and palpable pulsation, with probable secondary cardiac changes. Easy as the diagnosis between new-growth and aneurysm is in most cases, in others there are no distinctive signs which enable us to get beyond the fact that there is some mediastinal obstruction, and the differential diagnosis has then to be made by general considerations, viz., by the age and history of the patient. Thus if the patient be a man of middle age, with a history of hard muscular work, of drinking, and of syphilis, and who has not lost flesh, the diagnosis of aneurysm is fairly certain; if, on the other hand, the patient be young or old, and have lost flesh, the probability will be equally strong in favour of growth.

Still, take what care we will, the diagnosis may be incorrect, for even such characteristic signs as pulsation and murmurs may mislead, as the two cases following show.

1. A man of 70 had dyspnoea and stridor and dullness beneath the sternum, with increased voice and breath sounds. He had no pulsation or definite signs of aneurysm and had lost flesh rapidly. The diagnosis of new-growth seemed clear. He died suddenly of hæmoptysis, and on the autopsy a very large thin-walled aneurysm of the whole arch of the aorta was found. It was a case of non-pulsating aneurysm similar to those described by Mr. Morrant Baker in the popliteal and other parts.

2. On the other hand a young man of 25 came under observation with the signs of mediastinal mischief. He was well nourished and had not lost flesh then. There was distinct pulsation under the sternum, and the diagnosis of aneurysm of the aorta was made. He was placed upon Tuffnell's diet, and after a few weeks the pulsation abruptly disappeared beneath the sternum, and at the same time thrombosis occurred in the femoral vein. The conclusion seemed obvious that clotting had occurred in both places, and the diagnosis of aneurysm seemed to be confirmed. Before long it became clear that the diagnosis was wrong, and when the patient died some months later a large sarcoma was found in the mediastinum involving the pericardium and roots of the lungs.

In the common form of mediastinal tumour the growth starts in the glands or other tissues of the mediastinum and only involves the bronchial glands late or secondarily. There is, however, another form which may fairly be classed with mediastinal growths, though it is sometimes placed among tumours of the lung.

Primary Tumour of the Root of the Lung.—In this form the primary growth is round the bronchus at the root of the lung. This is a very remarkable form of affection, and one to which the attention has not been given which it seems to me to deserve. It is of all kinds the most difficult to diagnose, and may even be easily overlooked on the *post-mortem* table, for the growth is often extremely limited and the glands in the mediastinum are not involved.

The growth seems to me to originate in the peribronchial tissue, and I believe it to be always cancerous, with a large amount of fibrous tissue in it, which undergoes contraction and thus compresses the bronchus early. It does not invade the inner walls of the tube and ulcerate, but spreads along the tubes in the peribronchial tissue for a distance of two or three inches into the lung. On section it appears white, and looks like fibrous peribronchial thickening, and its real nature might be, and is, easily overlooked. The lung beyond shows the usual results of obstruction, and in the airless infiltrated portions, cavities are often found which have been described as softened secondary nodules, but are really patches of subacute pneumonic inflammation which have undergone necrotic degeneration.

When the main bronchus is affected the whole lung passes into the condition described. Dulness is found over the whole side, the movements are restricted or absent, and as the vocal vibrations, vocal resonance and breath sounds are

also absent, the diagnosis from pleuritic effusion will be very difficult. When the secondary changes in the lung lead to a rise of temperature, as they not infrequently do, the case may closely resemble phthisis. In one case empyema was diagnosed, and a needle inserted which brought away some puriform fluid. The nature of the case was subsequently recognised and diagnosis of cancer at the root of the lung correctly made. The *post-mortem* showed that the fluid obtained by the needle came from one of the necrotic cavities in the lung.

I have recorded three cases of this kind in the 35th volume of the *Pathological Transactions*, and another is published in the 39th volume by Dr. Newton Pitt, which is remarkable in two respects, first, that the growth originated in a bronchial gland, and secondly, that there were two secondary nodules in the opposite lung. In the cases described, and in others I have seen, secondary growths were absent (*cf.* chapter on New-Growths of the Lung).

3. ENLARGED BRONCHIAL GLANDS.—The bronchial glands are situated round the bifurcation of the trachea and roots of the lungs. They are ten to twelve in number, the largest being placed opposite the bifurcation of the trachea, the smallest round the bronchi and their primary divisions for some little distance within the substance of the lungs.

They are of great importance, first because the lymphatics of the lungs run to them, and they are thus subject to enlargement, both acute and chronic, as the result of acute or chronic affections of the lungs; and secondly, because of the important anatomical relation in which they stand to the air-tubes, so that slight enlargement may give rise to marked symptoms of pressure.

In adults the glands are almost always much pigmented from the deposit in them of the carbon and other foreign particles introduced into the lungs from the air. This pigmentation is often associated with some enlargement, chiefly of a fibroid nature, but rarely enough to produce symptoms. In all acute inflammations of the lung—for instance, in pneumonia, gangrene, or abscess, as well as in acute bronchitis—the glands are, as would be expected, acutely enlarged, but, again, rarely produce evident symptoms.

In chronic bronchitis there are always chronic changes in the glands, which sometimes lead to enlargement, but at others to contraction. In the acute exacerbations of chronic bronchitis the glands undergo fresh enlargement, unless they are prevented by fibroid or other chronic changes, and the pressure caused by them may produce the stridor-like sounds which are often heard in such cases and are usually regarded as a form of rhonchus.

Thus I had under my care a case of chronic bronchitis in which the dyspnoea and stridor were such marked symptoms that I was inclined to think that there must be stenosis of the trachea, probably of a syphilitic nature, with bronchitis as its result, but as the bronchitis subsided the stridor disappeared and with it much of the dyspnoea, a result to be fairly attributed to the relief of pressure as the inflammatory enlargement of the glands subsided.

The bronchial glands may of course be the seat of new-growth, but almost invariably secondarily to new-growth in some other part of the mediastinum. A case of primary sarcoma of a bronchial gland at the bifurcation is recorded by Dr. Newton Pitt.¹ It was discovered by accident in a patient who died of fractured base, and was the only growth in the body.

The chief cause of chronic enlargement of the bronchial glands, however, is tubercular disease, and this alone requires to be considered in any detail.

Chronic Tubercular Glands.—In phthisis the bronchial glands are always more or less affected, sometimes enlarged or sometimes shrunken and cretaceous, but they do not usually add to or specially modify the symptoms; at

¹ *Path. Soc. Trans.*, xxxix. 54.

any rate, it is not possible to assign to them their proper share in the symptoms produced. It is possible that the bronchial glands, like the glands in the neck, may be tubercular and caseate without phthisis in the lung or any evident tubercular disease elsewhere to which they might be secondary. This is probably not uncommon, in children at any rate, and may give rise in them to spasmodic breathing and paroxysmal cough, or even to attacks which are asthmatic in type, as in cases recorded by Eustace Smith¹ and by Gee.² Indeed, the former writer states that in asthma in children the possibility of enlarged bronchial glands should always be borne in mind. Dulness beneath the sternum has been described, but this cannot be caused by any enlargement of the bronchial glands, for they are too deeply seated. It can only be present when the lymphatic glands in the anterior mediastinum are affected.

To one point in the diagnosis of this condition Eustace Smith draws especial attention, viz., to the production of a venous hum beneath the sternum when the head is thrown far back.

That a murmur may be produced frequently in this way in children, in whom the glands are presumably in the condition described, there can be no doubt, and Eustace Smith claims to have connected it conclusively with enlarged bronchial glands, and he states further that such murmurs are not present in simple flat chests, or with an enlarged thymus. The mechanism of production of the murmur he believes to be this: when the head is thrown back the larynx and the glands with it are tilted forward, and thus the glands, if they are enlarged, press upon the innominate vein. The larynx and parts about it, however, must be free to move, for if not the necessary pressure cannot be produced.

A similar condition of the gland may occur in the adult. It is much less frequent than in the child, but the following puzzling case I believe to have been one of this kind.

A lad of 16 was under my care for very peculiar attacks of dyspnœa, brought on by swallowing solids. Liquid food could be taken without discomfort, but a lump of any kind brought on a severe attack which lasted a few minutes, and did not completely pass off sometimes for an hour or more. There were no physical signs in the chest, and the case seemed to be best explained by the assumption that there were enlarged bronchial glands so placed that the passage of a solid body down the œsophagus pushed them forward and caused pressure on the trachea. The patient was treated on that assumption. He remained under observation for some time, the symptoms slowly subsiding and ultimately entirely disappearing. He has continued since then in perfect health for some years.

Results.—As a rule acute enlargement subsides as rapidly as it comes on when the source of irritation is removed. If the irritation be chronic the glands may continue enlarged and produce pressure; more frequently they become fibroid, pigmented, shrunken, or cretaceous, and cause no further trouble.

With chronic tubercular glands, however, other important changes may occur, viz., softening in them or suppuration in or round them. Such changes, owing to the position which the glands occupy, are of serious consequence. The pressure alone may give rise to severe symptoms, and even cause death, as in a case recorded by Makins,³ in which tracheotomy was performed, but without relief. Barlow⁴ has described a similar case in which great thinning of the compressed bronchus had taken place, but without actual rupture. It most often happens that the gland ruptures and discharges its contents into the parts near: if into the mediastinum, with the production of a mediastinal abscess; if into the pleura, with the production of an empyema. Fortunately, these more serious results are not so frequent as the rupture into the trachea or bronchus. When this has occurred, the degenerating gland may in some rare cases slowly disintegrate and

¹ *Dis. of Children*, 1889.

³ *Path. Soc. Trans.*, xxxv.

² *St. Barthol. Hosp. Rep.*, vol. xiii. 165.

⁴ *Ibid.*, xxx.

be expectorated, the place ultimately healing completely. Not infrequently the discharges set up pneumonia, which is likely to gangrene or slough, and cause rapid death with septic symptoms. Gee¹ has recorded three interesting cases of this kind, and they are by no means rare. Where less acute inflammation is set up, the lung may pass into a condition of interstitial pneumonia or slaty induration. It is interesting to note that in some of these cases the caseous substance has set up a secondary tuberculosis in the lung. Sometimes the gland is discharged bodily into the trachea. It then becomes a foreign body and will act as such. It may be coughed out at once, but there is the risk of its impaction in the larynx or in the trachea, with immediate suffocation, as occurred in cases recorded by Edwardes² and by Kidd.³ In the process of extrusion the gland may project into the trachea and lead to great obstruction, as in Gulliver's⁴ case, where a caseous gland lay loose in a small abscess cavity, but projected into the trachea so far as to almost touch the opposite wall.

The child, aged 4, had been under treatment for bronchitis, but had improved so far as to be able to leave the hospital. A week later it was brought back with urgent dyspnoea, and died in a few hours, when the condition described was found *post-mortem*. The first attack was probably due to the discharge of the fluid contents into the air-tubes, and the final one to the protrusion of the gland itself into the trachea.

Sometimes a double perforation occurs, into the air-tubes on the one side, and into the pleura or mediastinum on the other, producing in the one case pneumothorax, and in the other mediastinal emphysema.

Treatment.—It is chiefly in children, as being most subject to the chronic tubercular enlargement of the glands without phthisis, that the question of treatment requires to be considered. In them treatment consists in the use of the ordinary remedies given for the similar enlargements of glands in the neck, viz., cod liver oil, steel wine, good food, and sea air. It was under treatment of this kind, with the addition of some iodide of potassium, that the lad of 16 just spoken of recovered.

Little aid can be looked for from surgery. Tracheotomy, though it has been performed in several cases because of doubt as to the exact diagnosis, must fail to give relief; the tracheal opening is above the seat of obstruction, and the lesions in the mediastinum are in most cases beyond reach of the knife.

Fortunately it is only the rarer cases that give rise to very serious symptoms, and if enlargement of the bronchial glands is as common a disease as some authorities believe, it is clear that in a large number of cases complete recovery must take place.

A successful case of operation from the front, for mediastinal suppuration in connection with tubercular glands, is reported by Milton.⁵

4. MEDIASTINAL ABSCESS.—Suppuration in the mediastinum is a very rare disease. In the anterior mediastinum pressure upon the air-tubes is unlikely to be produced unless the abscess lie beneath the manubrium; in the middle mediastinum, an abscess may easily lead to pressure, and, as already stated, softening or suppurating bronchial glands are the most probable cause; in the posterior mediastinum the abscess will be most likely due to caries of the spine. Such cases cannot, however, be anything but rare, for either the disease would have to be in the upper dorsal or the lowest cervical vertebra, or if the lower vertebra be the seat of disease, the abscess must develop upwards, while, as a matter of fact, caries of the spine is commonest in the mid or lower dorsal region, and the abscess develops downwards, *i.e.*, away from the air-tubes.

¹ *St. Barth. Hosp. Rep.*, vol. xiii.

² *Path. Soc. Trans.*, xxxvi. 104.

³ *Med. Chir. Tr.*, vol. lvii. and vol. xxvi.

⁴ *Ibid.*, xl. 38.

⁵ *Lancet*, 1897, i.

One case only of the kind has come under my observation. A little boy, nine years of age, was under Mr. Willett's care for acute disease of the spine in the dorsal region, for which he had been placed upon a spinal support, and kept upon his back. A fortnight before I was asked to see him he had begun to suffer from dyspnoea of a paroxysmal type. He lay in much distress, breathing with difficulty, and slightly cyanosed. Beyond slight stridor with moderate inspiratory recession of the lower ribs and soft parts of the chest, there were no definite physical signs, the air entering equally into both lungs, and the percussion note being everywhere resonant. There was evidently obstruction to the main air-passage, and as there was but little respiratory excursion of the larynx, and no laryngeal affection, it was clear that the trachea was the seat of obstruction, and the cause enlarged or suppurating glands, or else an abscess due to the disease of the spine. A few days later the lad died of dyspnoea.

The *post-mortem* showed that there was extensive disease of the spine from the second to the tenth dorsal vertebra, the ninth being the most affected, and the ninth disc being displaced. An abscess lay in front of all these vertebrae, and extended upwards into the neck nearly as far as the cricoid cartilage, largest below, narrow in the middle, but widest in the upper dorsal region opposite the manubrium sterni, where the oesophagus was compressed and the trachea flattened out, so that just above the bifurcation the walls were almost in contact. At the same level on the front of the trachea were two or three small and hard caseous glands which no doubt helped to increase the pressure. The lungs were congested but not otherwise altered.

A similar case is recorded by Schnitzler,¹ but in connection with disease of the second and third cervical vertebrae.

5. MEDIASTINAL EMPHYSEMA.—Air may reach the mediastinum either from the vesicles of the lung when they have been ruptured by violent coughing or other expiratory effort (*interstitial emphysema*); from the rupture of one of the large air-tubes, near the root of the lung; from the neck, as sometimes happens after tracheotomy, or when the air has spread to the neck from the subcutaneous tissue, as after a fractured rib. Air in the mediastinum may, of course, exercise pressure on the air-tubes, especially when it reaches the posterior parts of the trachea, but it is not easy to assign the part which pressure plays in these cases, for the dyspnoea may be due besides to the interstitial emphysema of the lung where the lung has been ruptured, or even to pneumothorax, to which emphysema of the mediastinum not unfrequently leads.

6. HÆMORRHAGE INTO THE MEDIASTINUM is another possible cause of pressure, but being due to the rupture of a large vessel or of an aneurysm, the symptoms of pressure are entirely subordinate to others of a more serious character.

7. AFFECTIONS OF BONES.—Abscess in connection with disease of the spine has already been referred to, but besides this, tumours of the upper dorsal or lower cervical vertebrae, as well as tumours of the sternum clavicle and upper ribs or abscesses resulting from diseases of those bones, have to be mentioned as possible though unlikely causes of compression.

8. NEW-GROWTHS OR FOREIGN BODIES IN THE OESOPHAGUS may be the cause of compression within the thorax, just as they are in the neck, but instances of this are very rare.

9. PERICARDIAL EFFUSION.—Large pericardial effusions are always attended with dyspnoea, due, no doubt, chiefly to pressure on the root of the left lung, but in part also to compression of the lower lobe of that lung, which is sometimes found collapsed and almost airless. With large effusions patients generally find the semi-recumbent or half-erect position most comfortable for them; the pressure is to a great extent taken off the root of the lung, and at the same time the whole weight of the effusion is not thrown upon the diaphragm.

¹ *Wiener Klinik*, 1877.

10. DILATED LEFT AURICLE.—From the relation in which the left auricle stands to the left bronchus it is quite possible that when greatly distended it might exercise considerable pressure on it. There are, however, not many cases recorded in which this has been actually demonstrated after death. The first cases were published by King¹ in 1838, and the preparations are preserved in Guy's Hospital Museum. Friedreich² has recorded a case in which the diagnosis was correctly made during life, and a good specimen of the kind was exhibited by Dr. H. H. Taylor³ at the Pathological Society.

The patient, a lad of 16, was the subject of mitral stenosis. The left auricle was extremely dilated and contained 11 ounces of dark clot, and its walls were nearly a quarter of an inch thick. The left bronchus passed immediately behind it and was flattened from before backwards in its whole length.

19. BRONCHITIS.

Bronchitis is a catarrhal inflammation of the air-tubes.

History.—The term bronchitis does not appear in medical literature until the beginning of the present century, when it was introduced into this country by Badham, and into Germany by Peter Frank. Although the term was not used, Sydenham is usually credited with having been the first to recognise one clinical form of the disease under the name *peripneumonia notha*, but Badham gives reasons for believing that Sydenham did not so much discover the disease as crystallise, in his graphic description, the knowledge of his contemporaries and predecessors, and he gives references to prove that what Sydenham described as *peripneumonia notha* was recognised and described by the early writers of the Christian era, notably by Aretæus.

Sydenham had the merit at any rate of fixing attention upon the disease, and the name he gave it lived. It was not, however, until the end of the next century that its pathology was comprehended. Frank, writing in 1792, thus defines *peripneumonia notha*—"Peripneumonia notha fortior nobis bronchiorum catarrhus est, quo in pituitosis obesis senibus cachecticis laxisque hominibus, frigida et humida sub tempestate, ab accedente membrane mucosæ hos canales investientis irritatione, copiosior tenaxque pituita celeriori passu secreta bronchiorum fines opplendo, suffocationem sat cito minuitur, quin ob dolorem aut ob primariam inflammationem spiritus præcludatur."

After the appearance of Badham's short but classical treatise the subject was quickly worked out, and Laennec was able to give so complete an account of the disease that it has formed the basis of all subsequent descriptions, and his classification is that still generally adopted. Since his time our knowledge has been extended chiefly in the direction of the minute pathology and bacteriology of the disease and of the complications to which it leads.

ÆTIOLOGY.

I. PREDISPOSING CAUSES.—Among the predisposing causes none is more important or commoner than *catching cold*; by this is meant not the direct irritation of the bronchial mucous membrane by exposure to cold air, but the effect upon the body of a chill.

It has been shown⁴ that when rabbits, after having been for some time in a hot chamber, are suddenly exposed to cold, they shiver and rapidly lose heat until the internal temperature is some degrees below normal; later, the temperature rises, and if the animal be killed after two or three days, interstitial changes are found in the lungs, liver, and kidneys, and even in the heart and nerves. The direct action of cold upon the skin is capable of producing nutritive changes, which may end in inflammation, as is seen in chilblains, and it is supposed that similar effects may be produced upon the internal tissues by the chilled blood, the spot picked out being that of least resistance.

If on an animal's belly a hot poultice be laid for some minutes, and replaced then by ice, the mucous membrane of the trachea and larynx becomes very pale from the contraction of the

¹ *Guy's Hosp. Rep.*, Ser. I., iii. 175.

² Virchow, *Spec. Path. u. Ther.*, v. Abt. 2.

³ *Path. Soc. Trans.*, vol. xl.

⁴ Landois and Stirling, 481. Rosenthal, *Wärme Regulation b. d. warmblut. Thieren*, Erlangen, 1872.

vessels.¹ If the ice continue to be applied, the pallor is replaced by redness, and finally by lividity of the mucous membrane, the circulation is impeded and mucus is secreted in increased amount. If the poultice be again applied the vessels contract again and the mucous membrane becomes pale, to be followed again by similar changes when the poultice is replaced by the ice. These experiments show how sensitive the mucous membrane of the trachea is to reflex stimulation of other parts of the body by heat and cold, and demonstrate how easily a chill to some parts of the body can cause inflammation of the respiratory organs.

These experiments men and women perform upon themselves daily, and there is no commoner cause of chills and consequent bronchitis, in summer as well as winter, than exposure to cold and draughts, after violent exercise or when the skin is acting freely.

The geographical distribution of bronchitis coincides with that of catarrh. Bronchitis occurs all the world over, and though there is a gradual increase in frequency from the equator to the poles, still the increase is by no means regular, for bronchitis is more frequent in some tropical places with an average winter temperature of $+10^{\circ}$ C. than in colder places with one of -5° C.² It is not so much that the place is hot or cold as that it is subject to violent changes of temperature, and that especially the fall in temperature is accompanied by a cold and cutting wind.³ Yet there are many places where the changes are sudden and considerable, and cold winds prevail, and yet bronchitis is rare. In these places the air is very dry.

The regions that are freest from bronchitis are :—

1. Those with a high uniform temperature and low dew point, such as are met with especially in the tropical and subtropical zones, as, for example, in some parts of India, the Antilles, the southern parts of California, and in Egypt.

2. Those with an extremely dry atmosphere, for example, many parts of North America, especially the high western prairies of Illinois, Wisconsin, Missouri, Iowa, and in some parts of the eastern hemisphere.

The three conditions, therefore, which favour the prevalence of bronchitis are violent temperature changes, with a moist atmosphere and cold raw winds. These conditions prevail most at the break of the year, and bronchitis is therefore commonest all the world over at the respective spring and autumn.

Season.—What is true of different places is true also of the same place in different seasons and in different years.

In this country bronchitis is most prevalent in the late winter and early spring months.

Haller's statistics, which are taken from the returns of the general hospital in Vienna, are defective, from the fact that but few children are admitted as patients into that hospital, but they are worthy of record as extending over many years and dealing with very large numbers (nearly 15,000).

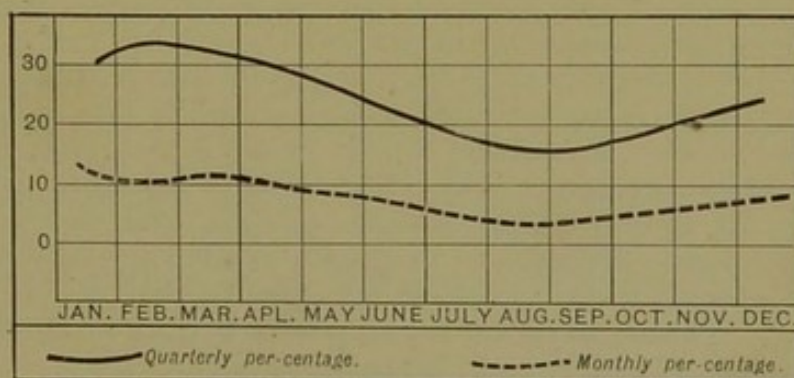


Fig. 29.

Curve constructed from Haller's observations, to show seasonal prevalence of bronchitis.

¹ Brunton, *Pharmacol. and Therap.*, 216.

² Hirsch, *Hist. Geogr. Pathologie*, Stuttgart, 1881.

³ Biermer, *Virch. Arch.*, vol. v.

The following statistics illustrate the effect of the time of year upon the prevalence of bronchitis in England, Zurich, Breslau, and Vienna.

	England. ¹	Zurich. ²	Breslau. ²	Vienna. ³
1st Quarter	36.793	43.5	28.6	33.2
2nd „	20.301	21.5	33.3	26.8
3rd „	18.327	17.5	9.5	18.1
4th „	32.570	17.5	28.6	21.9

The total number of patients admitted during this period was 223,328, this giving the rate of 6.7 per cent. for the relative frequency of bronchitis, compared with all other diseases.

Race in itself is without influence, except that the hardiest races suffer least. Natives of the tropics suffer more than immigrants from colder climes, and, when in temperate or cold regions, more than the inhabitants of those regions, and in these respects Europeans who have lived long in hot countries resemble the natives of those countries, so that they are very susceptible on their return home and need to take care of themselves until again acclimatized.

Feeble health.—A hardy and healthy constitution being the best protective against catarrh, all conditions which reduce the hardiness and healthiness of the body will increase the liability to catarrh; and further all local conditions by which the resistance of the bronchi is diminished will increase the tendency of the catarrh to settle in the bronchi.

Bronchitis is for these reasons common among weak and sickly persons, among those who lead effeminate enervating lives, or those whose health is reduced by disease, or old age, or among convalescents from illness.

Age.—Bronchitis is commonest and also most fatal at the extremes of life, *i.e.*, among little children and in old persons. Amongst children it is most frequent and also most fatal in the early months of life, up to the age of two years, after which the liability decreases rapidly with each year, but if the mortality can be taken as any test of frequency, very nearly one-half of all the bronchitis cases occur in children under 5 (*cf.* Table).

It is stated that, of very young children, suckling infants under six months of age suffer least, and from this is argued a less susceptibility to bronchitis, but the simpler explanation is more probable that little infants are better cared for and less exposed to cold. The excess of bronchitis under 5, though in part due to the feeble resistance which children offer to cold, may be often more fairly referred to the exposure to which children are subjected under the mistaken idea of hardening them. Children are often exposed as even strong adults would never be. This so-called hardening process is irrational and the cause of much disease, the weakly suffer and the strong do not need it.

But besides these causes, it is in the first few years of life that those diseases most prevail which are so liable to be associated with bronchitis, *viz.*, rickets, congenital syphilis, teething, and diarrhoea.

Sex.—It is stated on Lebert's authority that men are affected more often than women in the proportion of two to one. The mortality tables, to be referred to later, do not bear out this statement.

The figures taken from the Registrar-General's Table show that 27,445 men died as against 25,965 women, and out of 920 cases admitted into St. Bartholomew's hospital in the course of eleven years, 404 were males and 516 females.

The excess, if there be any, on the men's side may be explained by the greater exposure to which men are subject from their occupations, though this is to some extent compensated for by the greater hardiness it develops.

¹ B. W. Richardson, *St. Andr. Med. Assoc. Trans.*, 1869, ii. 231.

² Lebert, *Berl. klin. Woch.*, 1869, p. 53.

³ Haller, *Stat. allg. Krankenh. (Wien)*, 1869.

General diseases.—Certain general states of health seem to predispose to bronchitis, for instance, gout, Bright's disease, chronic alcoholism, or if they do not actually increase the liability by prolonging the duration of an attack, they diminish the chances of complete recovery.

Infectious diseases.—Of certain infectious fevers, bronchitis is so common an accompaniment as to seem to form an almost essential part of the disease; this is especially the case with measles and influenza. In typhoid fever again it is rarely entirely absent, and it may be so severe as to mask for a time the real nature of the disease. In scarlet fever, typhus, and small-pox it is not rare, and in the latter affection may be associated with an actual eruption of pustules in the large air-tubes. It is met with also occasionally in ague, and yields, like that affection, to full doses of quinine.

Murchison¹ states that though more frequent in typhus than in typhoid, bronchitis is in both fevers one of the commonest complications of severe cases, and a not-infrequent cause of death. It may in either develop very early, or at any period of the illness, and, having once developed, may persist for some time and so retard convalescence.

Apart from those fevers, in which there is a special tendency to bronchial catarrh, the frequency of bronchitis in fever may be attributed to several causes—(1) To direct irritation of the bronchial mucous membrane by some poison circulating in the blood. (2) To the increased respiratory interchange necessitated by the high temperature, which, while it increases the excretion of carbonic acid and water, increases also the demand for oxygen, this extra work leading to overwork, which manifests itself in bronchitis. (3) And lastly, where the fever is intense, to cardiac failure, to which cause may be referred many of the cases of so-called acute pulmonary congestion, of which perhaps the simplest and most striking instance is met with in hyperpyrexia.

Previous Attack of Bronchitis.—Among the local causes that predispose to bronchitis there is none more potent than a previous attack. The widespread tissue changes to which bronchitis leads explain the slowness of recovery and the risks there are of incomplete resolution. Each fresh attack leaves its traces behind, and by reducing the resistance predisposes to another.

Extension of Inflammation.—Bronchitis often develops also as a complication in the course of tubercular, syphilitic, or malignant disease of the air-passages, and is then due either to the direct spreading of inflammation from the diseased parts or to the passage over the mucous membrane of irritating discharges.

Again, inflammation may spread to the bronchi by direct extension either from the pharynx or larynx, as in croup and diphtheria, and after tracheotomy; or from the lung itself, as in pneumonia or gangrene; or from a tumour involving the air-passages, whether an aneurysm or new-growth.

Venous Congestion and Compensatory or Collateral Hyperæmia.—Another important group of predisposing causes is that connected with congestion of the bronchial mucous membrane.

Under the term "congestion" two different conditions are often included—the one, ordinary venous congestion, such as is commonly met with in the course of morbus cordis, the other, better described by the term collateral hyperæmia. In both cases the term "congestion" would be probably more appropriate than bronchitis; the physical signs are the same, but the cause different, being in the former case chiefly mechanical, in the latter physiological, but in neither necessarily inflammatory or produced by organisms.

¹ Wagner, *Arch. f. Heilk.*, 1872, p. 107.

Venous Congestion.—The bronchial veins open, it will be remembered, into the vena azygos and superior intercostal, and are thus connected with the right side of the heart. They also anastomose freely with the pulmonary veins.

When from any cause, as in mitral disease, there is obstruction to the circulation on the left side of the heart, the pulmonary veins become distended, as do also the branches which anastomose with the bronchial veins, and the result is congestion of the bronchi. If the obstruction continue the congestion spreads through the lungs to the pulmonary arteries, and so to the right side of the heart, and when this gives way to the large systemic veins, into which the bronchial veins open.

In advanced cases of morbus cordis the bronchial veins, therefore, become congested in two ways, first, from the pulmonary veins direct through the anastomotic branches, and, secondly, indirectly through the systemic veins. The result in either case is bronchitis; the morbid changes, however, resemble more closely those of chronic œdema than true inflammation.

A similar explanation applies to the bronchitis associated with emphysema. Though emphysema is commonly the result of bronchitis, still it also in turn leads to bronchitis, for the wasting of the alveoli and consequent destruction of vessels leads to obstruction to the circulation through the lungs, and, in the end, to dilatation of the right heart and systemic veins, and thus to congestion of the bronchi.

The congestion will be further aggravated by the paroxysms of coughing, and it is quite possible that violent coughing of itself is adequate to produce bronchitis. When during a paroxysm of cough the inspiration is long delayed or insufficiently performed, the circulation through the lungs is checked, the face becomes purple, the large veins swell, and some of the small ones not infrequently rupture, so that hæmorrhages occur into the skin, conjunctiva or from the nose. What is visible in the skin occurs also in the bronchi; the veins are distended and may rupture, both into the tissue and upon the surface, in the latter case giving rise to the streaky hæmoptysis so common in bronchitis. When the cough is chronic the veins of the skin are often permanently varicose upon the nose, cheek, and upon the chest, especially near the costal arch, and the bronchial veins also are often in a similar varicose condition. In whooping-cough also it is evident that the bronchitis which occurs is to some extent the mechanical result of the violent paroxysm of coughing.

Collateral Hyperæmia, on the other hand, is connected more with the pulmonary arteries than with the veins.

When any part of the lungs is rendered useless for the purposes of respiration, the blood which should have circulated through it is directed to the non-affected parts, and these do their best to make good the deficiency. If they succeed compensation is complete and no symptoms result. If, however, they fail, compensation will be incomplete, the parts of the lung which are extra-worked will become over-worked and break down; the break-down will declare itself by the signs of bronchitis, which under such circumstances is of very grave significance.

When the cause of the collateral hyperæmia is removable, as in pleuritic effusion, pneumothorax, or ascites, the congestion may disappear and recovery rapidly take place. In these cases the signs of bronchitis become the indication for immediate operation. When, however, the cause is irremovable, as in pneumonia, phthisis or tumour, they are very serious, and probably of fatal significance.

Local Irritation.—The last group of predisposing causes is that in which the irritation is applied directly to the surface of the bronchi. The irritant may be simply cold air, though this is rare by itself, unless the bronchial mucous membrane be peculiarly susceptible. The irritants are more commonly foreign substances existing in the air in gaseous, fluid, or solid form, and carried into the lungs by inspiration.

Bronchitis due to direct irritation of this kind is usually met with in connection with particular trades or occupations.

Of *gases* or *vapours* the most irritating are nitrous and nitric acid, sulphurous acid and bromine; but as a rule gases are much less irritating than dusts, and in some cases, as with chlorine and iodine, toleration may be established after a time and no further irritation result.

Few substances gain access to the lungs in the form of *liquids* except where they exist as spray, or where hot air, charged with products which are liquid at the ordinary temperatures, is cooled. Many of these substances are aromatic or tarry bodies, and produce no symptoms.

Fogs, which are so common in London and other large towns during the winter, belong to this group, and call for more than a passing notice.¹ The particles of moisture of which fog is composed become coated with a thin film of oily matter derived from the coal products in the air, to which its density, colour, and opacity are due. This coating renders evaporation difficult, so that a London fog is as thick in a warm room as out of doors, and may be carried into the country many miles away from town. Such fogs are of a most irritating nature to the eye as well as to the respiratory passages. The effect of fogs upon the prevalence and mortality of bronchitis is evident every year.

The following extract taken from the *British Medical Journal* of February 13, 1882, is of interest in this relation:—"In the week ending Dec. 20, 1873, following the memorable fog which was so fatal to the beasts in the cattle show, the death-rate in the metropolis rose to 37·5

"In the week ending Feb. 7, 1880, following a week of intense frost and dense fog, the rate rose to 46·7; and again last week, influenced by the dense fog of the preceding week, and a few days of low temperature, the exceptional death-rate of 35·3 was recorded. The mortality due to fog affects each group of ages, but is smaller in infants under one year of age and greatest in elderly persons. The deaths from diseases of the respiratory organs rose from 415, 543, and 647 in the three preceding weeks to 994 last week, of which 694 were due to bronchitis and 185 to pneumonia, exceeding the corrected average by no less than 427.

"The extraordinarily fatal character of the London fog may be inferred from the fact that during last week, when the metropolitan death-rate rose from 26·4 and 27·1 of the two previous weeks to 35·3, the rate in 27 provincial towns, having an aggregate population exceeding that of London, only rose from 23·4 and 23·1 to 25·2. Thus the London death-rate exceeded last week the average in the 27 provincial towns by no less than 10·1 per 1000, and as the mean temperature in the provincial towns scarcely differed at all from that in the metropolis, the excess in the death-rate of 10·1 per 1000 may be attributed to the pernicious effects of the London fog."

Dusts.—The *solids* exist in the form of dust. Coarse particles do not travel far into the respiratory passages. They produce coughing and are generally expelled at once. The finer particles make their way in time into the ultimate branches of the bronchi, and are found in great numbers round the alveoli and infundibula, whence they may be traced along the lymphatics to the glands at the root of the lungs.

It is only a portion of the dust inhaled which reaches the alveoli, even if the particles be fine; the rest is deposited on the walls of the tubes, and thus gives rise to bronchitis.

Hirt divides these dusts into vegetable, animal, mineral, and metallic, and of these the vegetable dusts are the most irritating, and the mineral and metallic dusts come in order next. But the latter groups are the most important, on account of their greater frequency.

When this irritation has been long continued, it is likely to be complicated with, or end in, phthisis. The liability to this is greater with some forms of dust than others—for instance, though bronchitis is more frequent with the vegetable dusts, phthisis is more frequent with the mineral and metallic, as the following table shows.

TABLE showing, out of a hundred sick persons in each group, the number in each affected with the disease specified.²

	Metallic.	Mineral.	Vegetable.	Animal.	Mixed.
Chronic Bronchitis,	14·8	11·0	19·0	13·6	18·4
Emphysema,	3·1	9·0	4·7	3·0	5·1
Pneumonia,	7·4	5·9	9·4	7·7	6·0
Phthisis,	28·0	25·2	13·3	20·8	22·6
Total,	53·1	51·1	46·4	45·1	52·1

No conclusion can, however, be drawn from this table as to the relative frequency of bronchitis and phthisis in these two groups, for the phthisis was in many cases preceded for a longer or shorter time by bronchitis, so that the two groups are not mutually exclusive.

¹ Frankland, "On Dry Fog," *Proc. of Roy. Soc.*, 1879, No. 192.

² Hirt, *loc. cit.* Merkel (Ziemssen, *Encycl.*, vol. v. p. 489.)

Of *vegetable dusts* the most irritating are those derived from the siliceous husks of cereals, or from the fluff of flax, hemp, or cotton, and consequently bronchitis is common among labourers in grain ships and grain warehouses, among flax, hemp, and lint dressers, etc.

The chief *metallic dust* is that of steel, and its effects are seen in the tendency to chest disease and in the shortened duration of life among knife-grinders, needle-pointers, metal-turners, etc., but the effect upon the lungs will much depend upon whether the grinding is done wet or dry, and how near the grinder sits to his work.

The *mineral dusts* are chiefly derived from the working of stone or pottery, and therefore masons, mill-stone makers and some kinds of potters suffer severely.

Into this group come also coal and charcoal, but of these charcoal is comparatively inert and coal is much less noxious than others.

The *animal dusts* arise from the working of feathers, wool, or bristles, and thus the trades of weaving, spinning, and brush-drawing are rendered dangerous to health.

As the various dust particles have been found in the lung tissue, terms have been used to indicate the nature of the dust to which the lung lesions are due, viz., anthracosis, for the coal and charcoal group; siderosis, for the steel group; and chalicosis, for the stone-dust group. These affections will be described more fully later.

Inheritance.—It is possible that a certain delicacy of the bronchial tissues, as of general constitution, may be transmitted, which may render the offspring of bronchitic parents more liable to bronchitis, but in the strict sense of the term inheritance cannot be shown to play any important part in the causation of bronchitis.

II. EXCITING CAUSES.—If we are to apply the conclusions of general pathology to bronchitis, we may conclude that the inflammation is excited by the presence of pathogenic organisms.

Bacteriology.—What these organisms are it is obviously very difficult to determine, for it is so easy for germs of all kinds to gain access to the air-tubes, and when once there to flourish readily.

The finer bronchi and vesicles are usually entirely free from organisms, but the trachea and main bronchi, even in health, have on their surface many organisms both pathogenic and non-pathogenic. Among the latter are the *pneumococcus*; *Friedlander's bacillus*; *Strept. pyogenes*; *Staph. pyogenes aureus*, and *Staph. p. albus*. As the pathogenic organisms are found in greater numbers in the bronchi than in the mouth, it follows that they must grow there freely.¹

In bronchitis all these organisms have been found in greater numbers than in health, and many others too, e.g., *Proteus*, *bacillus typhosis*, *bacillus coli communis*, as well as some *hyphomycetes*, e.g., *Aspergillus fumigatus*, *Oidium albicans*, etc.

The observations upon broncho-pneumonia may be also applied to bronchitis, and we may conclude that the two commonest organisms in bronchitis are those which have been found to be commonest in broncho-pneumonia, viz., the pneumococcus and streptococcus, either alone or together, or associated with others.

The organisms are most commonly introduced with the air inspired, either directly from without or indirectly from the throat or mouth. In this respect it is interesting to remember that nothing is commoner than for a catarrh to begin

¹ Bartels, *Utbl. f. Bakteriolog.*, xxiv. 12.

in the throat or nose and spread downward to the chest. The most striking and obvious instances of this are seen in diphtheria and scarlet fever; but the fact is not less important, though less obvious, in such diseases as measles and influenza, and there can be little doubt that a more careful toilette of the nose, mouth, or pharynx might do much to prevent bronchitis from developing at all.

Another source of infection is from within, as when irritating discharges are evacuated through the lung, *e.g.*, from an empyema or a tubercular or other infective cavity in the lung. Or again, the infection may be carried by the blood, and possibly this may be the explanation of bronchitis appearing in the course of some specific fevers.

In the case of the specific fevers the further question would arise in bronchitis as in broncho-pneumonia, whether the infective organisms were the same as those of the specific fever or not, *e.g.*, Eberth's bacillus in typhoid, Löffler's bacillus in diphtheria, the influenza bacillus in influenza, etc.

It seems probable that in most instances the infection is due to some of the common non-specific organisms, *e.g.*, the streptococcus.

If the bacterial view of inflammatory bronchitis be the true one, most of the so-called causes of bronchitis become, not exciting, but predisposing, causes, and must act in preparing the way for the attack of the organism by reducing the resistance of the bronchial tissues.

This reduction of the normal resistance of the bronchi may be brought about by general, or by local, causes; for instance, the resistance may be normally feeble, as at the extremes of life, *i.e.*, in the young or old, or be reduced by accidental causes, by ill-health, as in rickets or during convalescence, or by gout, or albuminuria, etc.

Of the local causes the commonest is chill, whether this acts directly upon the mucous membrane of the bronchi, or indirectly through the blood. In this connection it is interesting to remember Pasteur's observation that fowls which were normally immune to anthrax could be rendered susceptible to it by cold.

Another group of predisposing causes is formed by direct irritants, such as the vapours of iodine and bromine; solid particles, such as stone dust or metal filings; or irritating discharges passing over the surface.

Of all predisposing causes none is more potent than a previous attack of bronchitis, and the reason of this it is easy to comprehend, when there are borne in mind the profound changes produced in the bronchial tissues by an attack of bronchitis, and the long time which is required for complete recovery.

Congestion of the lungs.—If the germ theory be accepted for bronchitis, then it will be necessary to draw a sharper distinction than is usually done between the inflammatory and the other forms of bronchitis, so that the old distinction between active and passive congestion will have to be revived in some form.

The signs of congestion of the lung are those of bronchitis, and *acute congestion* of the lungs would then be equivalent to acute or inflammatory bronchitis; the *passive congestion* of the lungs, such as occurs in the course of morbus cordis, is probably of mechanical and not of bacterial origin.

Another form of congestion which also should probably be separated from both of these is that which is sometimes called *complementary congestion* or *compensatory hyperæmia*, the typical instance of which is seen in acute pneumonia. Thus the signs of congestion or bronchitis developing in the non-inflamed parts of the lung, on the same side or on both sides, are well known to be of very grave significance, as being evidence of the physiological break-down of an over-worked lung.

Although the views I have just expressed as to the pathogeny of bronchitis are, no doubt, correct theoretically, we are not in a position to express them in

the form of a complete classification; but this is not so very important, because the other factors in the causation of bronchitis are not thereby rendered of any less importance, though they are reduced from the rank of primary or exciting causes to that of predisposing or secondary causes.

At the same time to grasp fully the importance of the bacterial element in the causation of bronchitis will have a good influence on our method of treatment, and what is still more important, our measures of prophylaxis.

It would suggest the more persistent use of antiseptic inhalations and sprays as curative and not merely palliative measures, and in respect of prophylaxis would lead to the more careful treatment on antiseptic principles of local affections of the naso-pharynx and mouth, especially in patients whose general condition renders them especially susceptible to infection, as during an acute fever, or the convalescence from it.

FREQUENCY AND MORTALITY.—Bronchitis is an extremely common affection, but there are, I believe, no trustworthy statistics which show its frequency relatively to other diseases.

Hospital statistics are useless for this purpose, for the record of out-patients is usually too incomplete, and only the severe cases which are admitted as in-patients enter into the annual statistical reports, and lastly, in these reports there is no account taken of bronchitis which is secondary to other diseases, and it is only where bronchitis is the primary disease that the case appears at all under this heading. For these reasons the statements made differ widely.

The percentages vary from 2 to 25. Lebert gives 8 as the relative percentage for Breslau and Zurich, Haller 6·7 for Vienna, and Hirsch 21 for the northern parts of Europe, where bronchitis is most prevalent.

For the same reasons it is impossible to determine accurately the percentage mortality of bronchitis, for though the actual number of deaths is recorded the number of cases cannot be ascertained; the statements made are therefore only approximate guesses.

The mortality is said to be 2·8 for England; Lebert gives 2·4 for Zurich and 3·0 for Breslau.

The reports of the Registrar-General supply ample materials for the determination of the absolute mortality from bronchitis in relation to the mortality from other diseases.

TABLE giving the death-rate for every 1000 persons living. The years given are chosen so as to be well before the advent of influenza:—

	From all Causes.		From Bronchitis.	
	Male.	Female.	Male.	Female.
1883	20·67	18·4	2·3	2·1
1884	20·7	18·5	2·08	1·9
1885	20·05	18·03	2·3	2·14
1886	20·34	18·27	2·25	2·16
	<hr/>	<hr/>	<hr/>	<hr/>
	20·4	18·3	2·23	2·07

This gives a death-rate for bronchitis of 11 per cent. for males, and of 11·3 per cent. for females, *i.e.*, of every 100 persons dying about 11 die of bronchitis.

Taking, then, all ages together, bronchitis accounts for about 10 per cent. of all deaths, but the mortality varies greatly at different periods of life. This is more clearly seen in the next table, which is derived from that just given.

TABLE showing out of 1000 deaths from bronchitis the number that die at each period :—

Under 1 year	286	} 419	Under 25 years	3	} 457
„ 2 years	133		„ 35 „	18	
„ 3 „	37		„ 45 „	28	
„ 4 „	14		„ 55 „	60	
„ 5 „	8		„ 65 „	112	
„ 10 „	12		„ 75 „	156	
„ 15 „	2		„ 85 „	107	
„ 20 „	2		Over 85 „	22	

From this table it is seen that out of every 1000 persons dying of bronchitis, no less than 419 die before the age of 2 years, and 457 more after the age of 55. Without laying too much stress upon these numbers, which would have to be corrected for the number of persons living at each period, the table shows how fatal bronchitis is at the extremes of life.

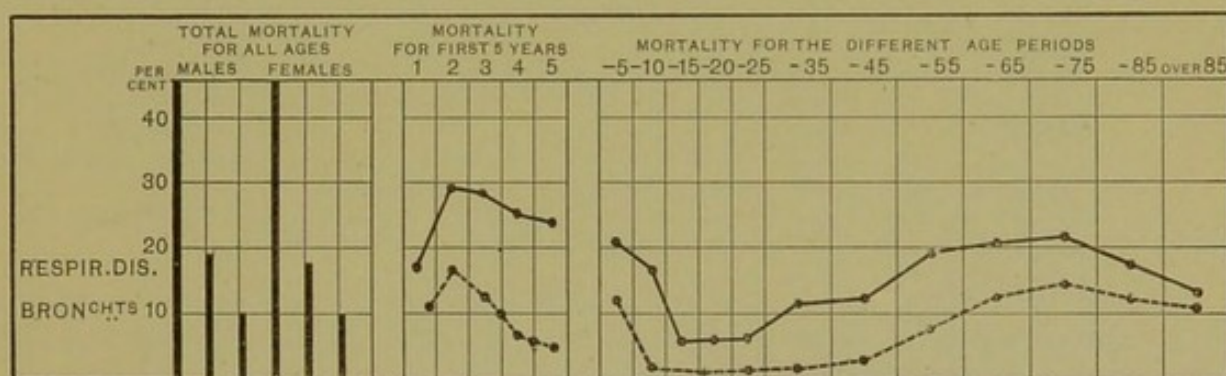


Fig. 30.

TABLE constructed from the Registrar-General's statistics for the years given. It shows the mortality (1) of all respiratory diseases (continuous line); (2) of bronchitis (dotted line), relative to the mortality from all causes (reckoned as 100 per cent.) at the different age periods and also for the first five years of life.

THE MORBID ANATOMY OF ACUTE BRONCHITIS.—The bacteriology of the disease has been already dealt with.

Even where bronchitis has been the cause of death, there is often singularly little to be seen, with the naked eye; the lungs may appear more or less congested, with irregular patches of emphysema or collapse scattered here and there, or if a section be made and the surface squeezed, drops of frothy mucus or mucopus may exude from the orifices of the divided tubes; the lower parts of the lungs behind are usually in a condition of hypostatic congestion, *i.e.*, are oedematous and sometimes almost airless, and, on section, more or less frothy watery fluid flows from them. The right side of the heart is greatly dilated, and the various organs show the usual results of venous congestion consequent on a failing heart.

If the bronchi be laid open, their surface is found covered with mucopurulent secretion, and when this is removed the mucous membrane beneath looks swollen and is altered in colour.

The secretion varies much in quantity; it may be scanty and viscid, or copious and frothy, watery, or more or less puriform. It constitutes the sputum, which will be fully described subsequently.

The mucous membrane generally is swollen, often velvety in appearance, and usually redder than normal. The redness may occur in spots (punctate), in patches of irregular size and distribution, or involve the whole surface uniformly; but it may be completely absent, so that the mucous membrane may look even paler than normal. The colour may be pink, bright red, dusky, or livid; and it is not easy to distinguish these inflammatory colour changes from those due to *post-mortem* staining. It is not possible from the appearances after death to draw any but the most general conclusion as to the condition during life, for owing to the large amount of elastic tissue which the lungs and bronchi contain, the signs of congestion are rapidly effaced after death. As a general rule, the smaller the tubes affected the more likely are naked eye changes to be observed.

The microscopical changes are, however, in severe cases widespread and profound.

They have been investigated minutely by various methods:—

1. By examination of the human lung in cases which have died with bronchitis in all its forms.¹

2. By means of experiment upon animals.

a. By the insufflation of irritating substances or fluids into the trachea and bronchial tubes, *e.g.*, chromic acid, or bichromate of potash (Socoleff²) or nitrate of silver (Cornil and Ranvier³).

b. By the subcutaneous injection of appropriate drugs, such as cantharidin (Cornil and Ranvier). The solution used was 4 to 6 grammes of a solution of cantharidin in acetic ether saturated at a temperature of 20° C., and equivalent to about 5 to 8 milligr. of cantharidin. This produced symptoms of intense poisoning, and in the animals employed, *viz.*, rabbits, was fatal in a few hours.

The results of all these investigations are practically the same, the differences being only those of degree and accounted for by the method employed.

Acute bronchitis, like other acute inflammations, commences with active hyperæmia, which is followed by nutritive changes in the tissues and by exudation.

The hyperæmia, or congestion, shows itself first in great distension of the small vessels, branches of the bronchial artery, which ramify in the inner fibrous layer of the mucous coat beneath the basement membrane. Some loops of these vessels may become so distended as to push the basement membrane before them and form villus-like prominences on the surface, to which the velvety appearance referred to is due; occasionally they burst and hæmorrhage occurs into the bronchus. As the inflammation extends, the deeper vessels in the muscularis and adventitia take part in the congestion, and some of the small vessels here may give way, producing the punctate hæmorrhages not infrequently found *post-mortem* in severe cases.

The hyperæmia is immediately followed by increased secretion from the mucous glands and from the surface of the tube. The secretion at this time consists almost entirely of mucus and contains very few cells. It is viscid and sticks to the surface.

The mucous membrane now rapidly swells, and marked histological changes are evident.

The basement membrane is œdematous, and often thrown into folds.

The epithelial layer is much thickened owing to a great increase in the number of transitional cells (α_1). These new cells being derived (according to Hamilton and Socoleff) from Debove's membrane, which is in a state of active proliferation, the ciliated columnar epithelium is gradually pushed farther and farther out, and is finally shed (*e*). Sometimes a few round or cubical ciliated cells are seen

¹ Hamilton, *Practitioner*, 1879.

² *Virch. Arch.*, lxxviii. 611.

³ *Manuel d'Histolog. Pathol.*, 1884.

for a time, but soon all trace of ciliated cells and columnar cells is lost, and the epithelial coat is made up of round cells only, which are either shed as such or after undergoing mucoid change (*c*).

The mucous glands (*n*, *n*₁) are in a state of great activity; they are swollen and often easily seen by the naked eye, the cells being enlarged and very granular, and the ducts filled with these cells in various stages of mucoid change, and mingled with them a few leucocytes. When the contents are pressed out

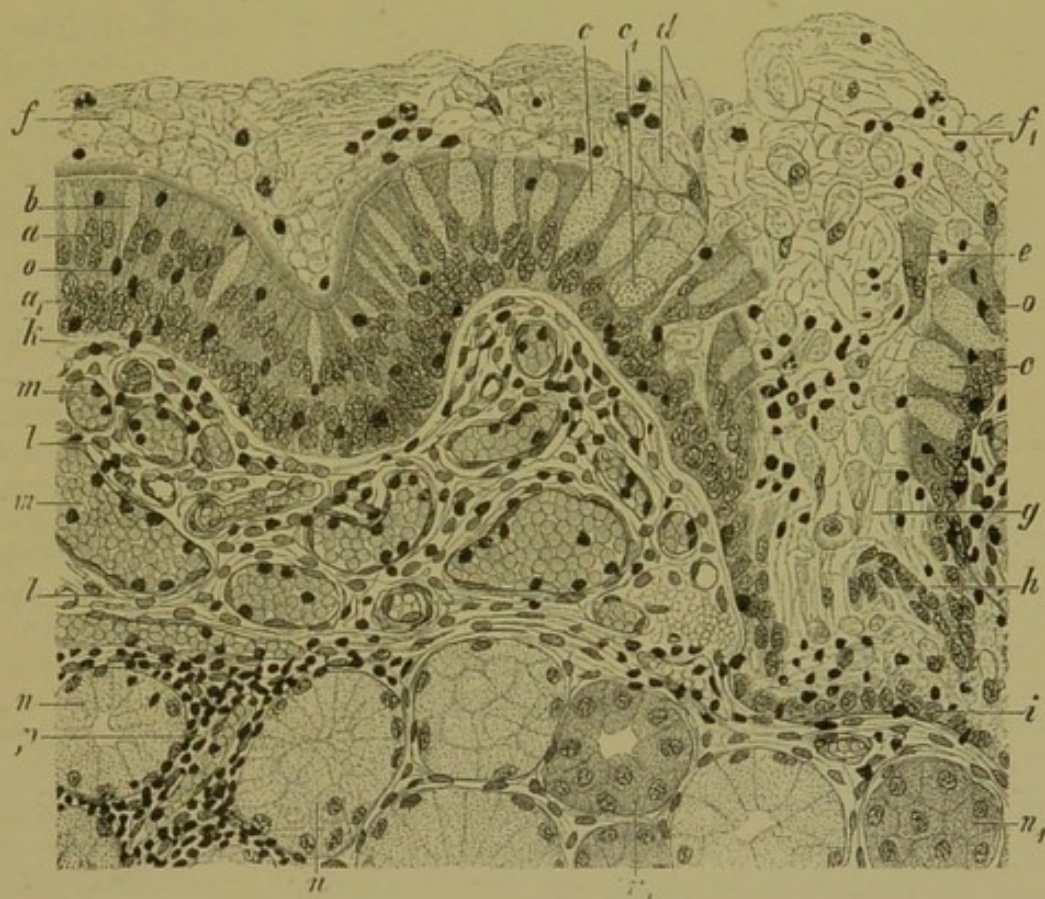


Fig. 31.

Acute bronchitis. *a*, ciliated epithelium; *a*₁, deep layers of cells; *b*, goblet cells; *c*, cells which have undergone extreme mucoid degeneration; *c*₁, nuclei affected in same way; *d*, such degenerate cells cast off; *e*, ciliated epithelium cast off; *f*, deposit on surface composed of drops of mucus (*f*'), stringy mucous and pus cells (*f*₁); *g*, duct of mucous gland filled with mucus and cells; *h*, epithelium of duct detached; *i*, epithelium of duct still attached; *k*, swollen hyaline basement-membrane; *l*, connective tissue of mucosa, partly infiltrated with cells; *m*, distended blood-vessels; *n*, mucous glands filled with mucus; *o*, wandering cells in epithelium; *p*, cellular infiltration of the connective tissue of the mucous gland. (Ziegler, *Pathol. Anat.*)

of the ducts their dilated orifices look like small erosions, and have been incorrectly described as catarrhal ulcers.

The secretion in the tubes is in this stage more abundant; it is formed partly of serous exudation from the vessels, and partly of mucus derived from the glands and epithelium. It is rich in mucus and small round cells.

Whether the small round cells are all derived from the epithelial layer, or in part also from the vessels by migration, is not agreed upon, but since red blood cells have been observed among the cells of the transitional layer, and that not in sufficient numbers to be explained by actual hæmorrhage, the possibility of migration in bronchitis cannot be denied, although Hamilton and Socoleff have failed actually to observe anything of the kind.

The lymphatics immediately beneath the basement membrane are packed with small cells, and as the inflammation extends the cellular infiltration is found in the intercellular spaces of the muscularis, and in the larger lymph-channels of the adventitia (*l*). It may be traced thence along the perivascular and peribronchial trunks to the root of the lung, where the glands are quite early in the affection swollen and hyperæmic. The nutrition and function of the muscular coat are in this way greatly impaired, a fact which accounts for the dilatation of the bronchi observed in some acute cases.

The changes just described are those met with in a *middle-sized bronchus*. They are the same in the *trachea*; with these differences, that the congestion is more evident, being especially marked between the cartilages and in the membranous portion, that the mucous glands reach a greater size, even that of a hemp-seed, and that the ducts and their orifices are more dilated.

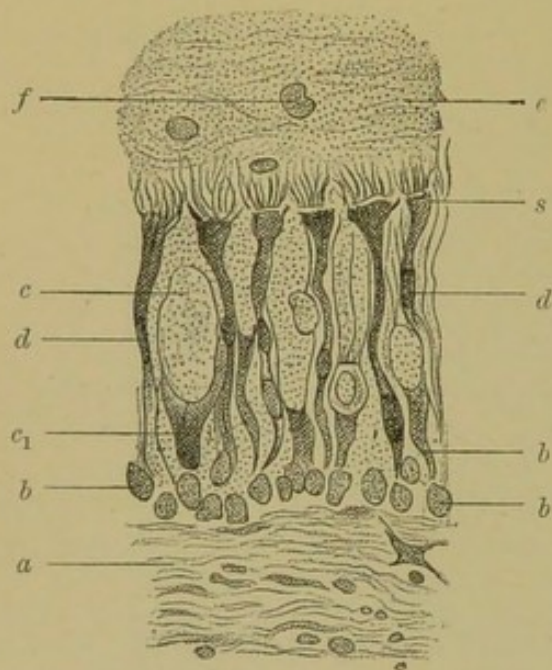


Fig. 32.

Section of epithelial layer of trachea in catarrhal inflammation (Cornil and Ranvier, *Man. d'Histol. Pathol.*). *a*, basement membrane; *b*, round cells in relation with it; *c*, goblet cells; *c*₁, their nucleus; *d*, narrow compressed cylindrical epithelial cells; *e*, mucus on surface free, and in globules, *f*.

In the *finer tubes*, *i.e.*, in tubes with a diameter less than one millimetre, the lumen is so small that it is readily plugged by the swelling and secretion, and thus rendered impervious to air. The result is that as the air behind the obstruction is absorbed, the lung in that part collapses, while the immediately surrounding parts dilate. In this way are produced the irregular patches of emphysema and collapsed tissue already referred to.

If the inflammation spread to the intralobular and terminal bronchi, the acini of a lobule or group of lobules also become involved in the same process, and give rise to the patches of consolidation known as broncho- or catarrhal-pneumonia. The vesicles are filled with large epithelial and small lymphatic cells, and occasionally contain some ciliated epithelium which has been sucked into them during violent inspiration.

The lymphatics of the small bronchi are in immediate relation with those of the alveoli, and they all become filled with lymph cells, so that on section a small tube looks as if surrounded by a circular zone of connective tissue stuffed with leucocytes. The muscle-cells may be so compressed by this infiltration as to waste and even permanently disappear.

The bronchial glands are enlarged in all cases of bronchitis alike, whether acute or chronic.

Should the inflammation subside, the inflammatory products may be gradually removed, the epithelium gradually regenerated from Debove's membrane, and complete recovery take place. Recovery is, however, a process of growth, and requires time, and when growth and repair are sluggish, as in old people, recovery must be slow and may often be incomplete. Until the new epithelium is, as it were, seasoned, it will not be able to offer its normal resistance to irritants, and a delicacy may be left which will show itself in the tendency to relapse which is

so characteristic of the disease. With each relapse recovery will take longer and be less complete, and at last the patient will pass into the condition of chronic bronchitis, in which, though there may be periods of better and worse, the affection is never entirely absent.

THE MORBID ANATOMY OF CHRONIC BRONCHITIS.—In chronic bronchitis the secretion in the tubes varies much in character and quantity in different cases, as will be described later.

On laying the bronchi open the surface is of a purplish or slaty hue, but occasionally dull gray; it is smooth and shiny, and below it are visible coarse strands of connective tissue which give it a trabeculated and occasionally ribbed appearance like that of coarse canvas. The mouths of the mucous ducts are usually much dilated and easily seen. The basement membrane is as a rule thickened and sometimes projects in small bud-like outgrowths.

The epithelial covering is reduced in most cases to a single layer of irregularly formed cells (*a*, fig. 33), but there may be more than one layer, and then most of the cells are of the goblet form and have undergone mucoid degeneration.

The other coats are much thickened, partly from cellular infiltration and partly from connective tissue growth.

The cellular infiltration is so considerable as almost to conceal the muscular and fibrous tissue. It involves all the coats and extends along the course of the lymphatics even to the root of the lung, and following the same course there is in long-standing

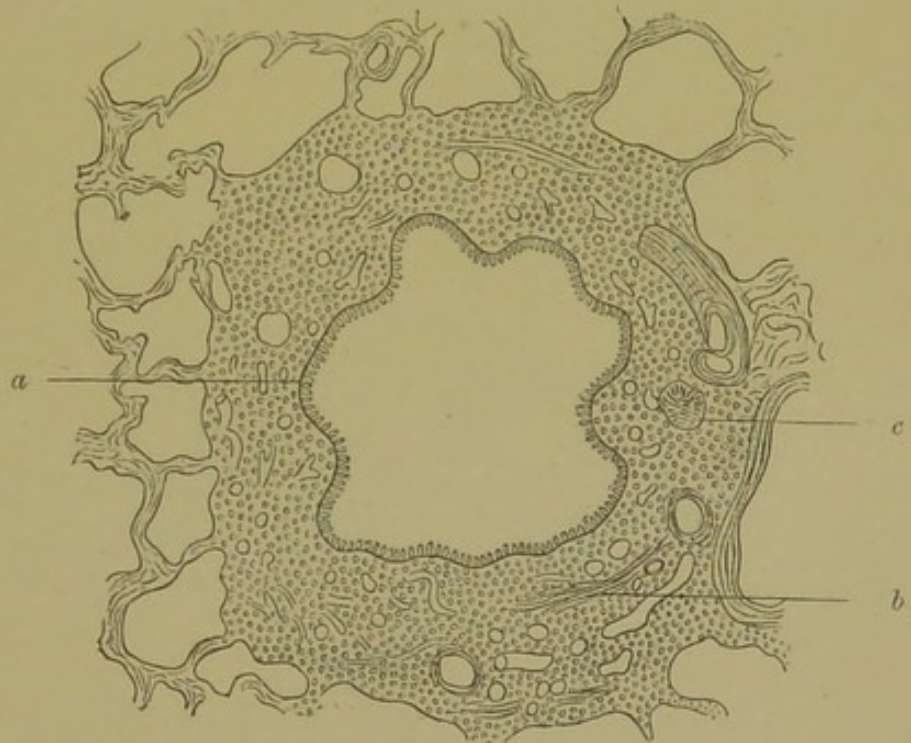


Fig. 33.

Section of a bronchus in a condition of chronic catarrh. *a*, germinating epithelium placed on basement membrane; *b*, remains of muscularis; *c*, a small nerve (*Hamilton*).

cases great increase in the fibrous tissue (interstitial pneumonia). The blood-vessels are dilated in all the coats and their muscular layer hypertrophied, owing, it is supposed, to vascular obstruction consequent on the tissue changes.

The muscular coat of the bronchi is as a rule atrophied (fig. 31), and replaced by fibrous tissue, but it is said to be sometimes hypertrophied (*Hamilton*). The mucous glands commonly atrophy and disappear, being replaced by fibrous tissue, but they may remain for a long time enlarged and active.

The cartilages are generally gradually absorbed, but they may calcify or are said to be sometimes even ossified. It is stated that they occasionally necrose, and being discharged into the tubes are coughed up; I doubt if this ever occurs

as the result of simple bronchitis, though it is not infrequent in the course of tubercle or syphilis.

In the chronic bronchitis which attends heart disease, the blood-vessels are greatly distended and tortuous, and there are many small hæmorrhages and patches of pigment. All the coats of the bronchi are œdematous, and there is but little of the cellular infiltration which is the most striking feature of true bronchitis. Rupture of the distended capillaries is of frequent occurrence, both into the tubes and into the vesicles, and though the epithelium is shed and the

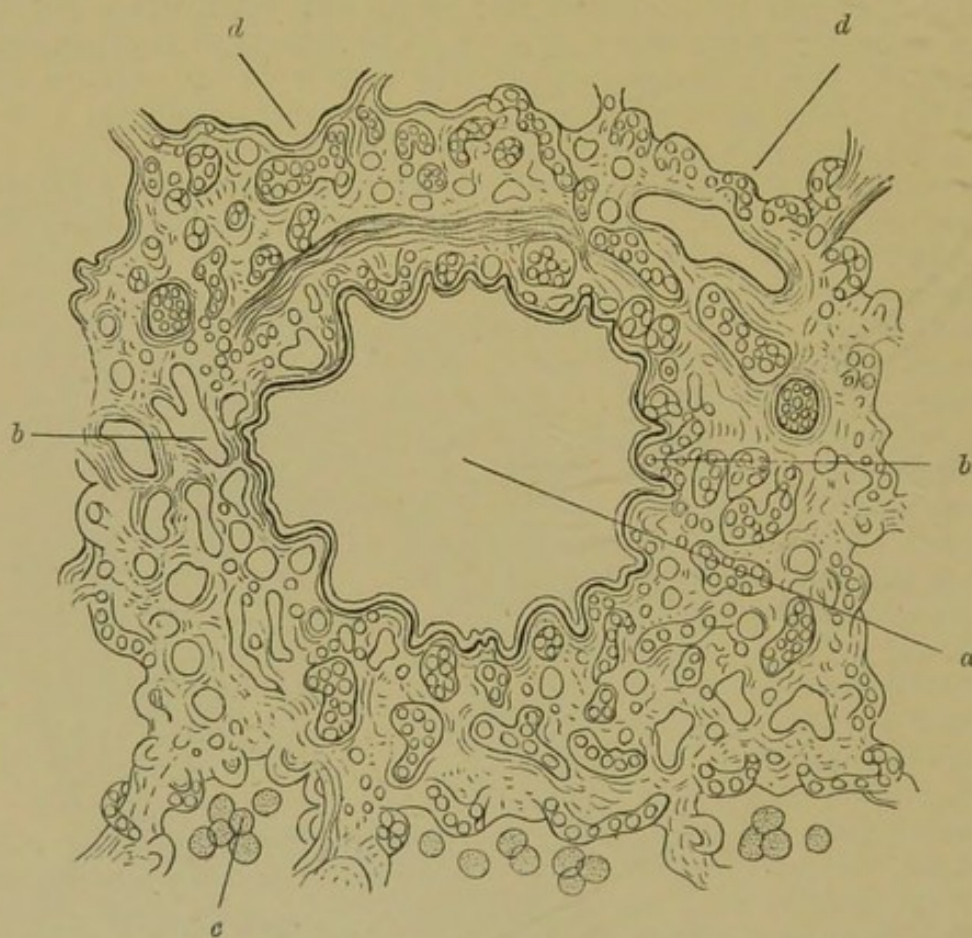


Fig. 34.

Section of a bronchus from a case of mitral regurgitation (*Hamilton*). *a*, lumen of bronchus, with mucous membrane thrown into folds; *b*, dilated capillaries; *c*, desquamated and pigmented alveolar epithelium; *d*, alveoli.

basement membrane thickened, this may be the simple mechanical result of œdema.

For these reasons, this form of bronchitis is regarded by many as the result of venous congestion, and not as a true inflammatory bronchitis in the strict pathological sense.

In the bronchitis associated with the inhalation of dust of any kind, the particles are found round the vesicles, infundibula and finest bronchioles, and not at all in the coats of the large bronchi, except in the outer part of the adventitia, where it is in immediate relation with the walls of the vesicles. They are especially abundant round the walls of the pulmonary artery (fig. 45), and in the interlobular septa (fig. 44), but beneath the pleura they are found only in the deep lymphatic plexus. It is clear, therefore, that the particles gain

access to the lung through the vesicles, infundibula, and smallest bronchioles, and reaching the perivascular, and in part also the peribronchial, lymphatics, are carried thence with the lymph stream to the lobular septa and deep layer of the pleura, and so on to the root of the lung, where they are found massed in large numbers in the glands. The bronchitis which follows is of the ordinary kind—the secretion is mucopurulent, and the cells in it are not pigmented.

The **pathological complications** to which bronchitis may lead in the lungs are—(1) transient or permanent emphysema; (2) transient or permanent bronchiectasis; (3) collapse; (4) bronchopneumonia; (5) interstitial pneumonia; and (6) chronic pleurisy. These affections will all be further referred to in their proper places.

PHYSICAL SIGNS.—The physical signs of bronchitis are few, and almost entirely auscultatory, viz., rhonchus, sibilus, and crepitation.

Rhonchus and Sibilus are the wheezing sounds produced in the large and small tubes respectively by the air as it passes over the surface of the swollen mucous membrane roughened by secretion.

Both may be musical—rhonchus frequently, sibilus rarely—and have been then described as cooing, piping, or whistling. They are frequently audible to the patient and to others at a distance, and may be felt also with the hand laid upon the chest. In both rhonchus and sibilus the expiration is prolonged, and is often as loud and even longer than inspiration; it may be sometimes not unlike true bronchial breathing, but it never has the harsh guttural high-pitched tone such as is heard over pneumonic consolidation. The pause which in health follows expiration may follow inspiration instead, and this in adults as well as in children.

The vesicular murmur is obscured or overpowered by the rhonchus or sibilus, but it is not abolished. If the tubes are plugged by secretion, the respiratory sounds in the corresponding parts may entirely disappear, returning again as soon as the tubes become pervious.

The **crepitation** is of the large, medium, or small kind, according to the size of the tube in which it is produced, and it may, in some forms of capillary bronchitis, be even so fine as to resemble the fine-hair crepitation of pneumonia, and it has then probably the same origin. In the other cases it is due to the bursting of bubbles in the fluid. It will therefore be absent where the secretion is scanty and viscid, as in early bronchitis, and most abundant where the secretion is fluid and copious, as in the later stages. It is sometimes absent, however, where the secretion is profuse. Crepitation varies with the amount of fluid in the tubes, the depth of respiration, the size of the tubes, and the distance of the seat of production from the surface.

Stokes has observed crepitation produced in the tube from the agitation of the secretion by the movements of the heart.

The most striking characteristic of the physical signs of bronchitis is the capricious way in which they vary in character and position within short spaces of time; an attack of coughing or a few deep inspirations may entirely remove all physical signs from one place and produce them in another. As would be expected, sibilus varies less than rhonchus.

A persistent localised bronchitis is a very suspicious sign, and points with great probability to some permanent local disease, and if at the apex is often one of the earliest signs of phthisis.

The **percussion-note** is unaltered in uncomplicated bronchitis, but if there be emphysema it may be hyper-resonant, and if there be collapse or con-

solidation dull. Patches of collapse and consolidation when small are often entirely masked by the complementary emphysema which develops, but if extensive, yield the ordinary signs. Extensive collapse occurs usually at the bases behind, and especially in children.

Inspiratory Recession.—If there be much collapse the soft parts of the chest may be forced in on inspiration. This is seen in the intercostal spaces and supra-clavicular and episternal fossæ, both in children and adults. In little children, where the ribs are yielding, the whole lower part of the chest may be sucked in to a considerable degree, though hardly ever to the extreme degree seen in laryngeal obstruction.

When bronchitis frequently recurs in weakly—especially in rickety—children, permanent traces are often left behind in various deformities of the thorax, *e.g.*, those known as the rickety and syphilitic chests; and the same explanation is to be given of Harrison's furrow, when it is abnormally developed.

The Sputum.—The essential constituents of the sputum in bronchitis are—

1. Mucus; with a few mucous cells, derived from the mucous glands or from mucoid degeneration of the epithelial cells of the respiratory passages.
2. Serous fluid; exuded from the blood-vessels of the bronchi.
3. Cells; some epithelial, from the mucous glands or from the epithelial layer of the mucous membrane, others small and round like pus cells, derived from the transitional layer of the epithelial coat or by migration from the blood-vessels.

These elements, combined in varying proportions, and mingled with more or less of saliva and air-bubbles, constitute the different kinds of sputa.

In a case of simple acute bronchitis the secretion is at first diminished, but soon increases in quantity; it is at first scanty viscid, and composed almost entirely of mucus. It soon becomes abundant and more fluid, being mixed with serous effusion from the vessels. It contains few cells and resembles saliva or white of egg, and is then described as "*sputa cruda*." At a later stage, usually when resolution is commencing, it becomes rich in cells, which have undergone fatty change and give it a yellow colour and an opaque appearance, "*sputa cocta*."

These changes are similar to those which are familiar in the secretion from the nose during an ordinary catarrh.

The chief named varieties of bronchitic sputa are the following:—(1) *Sputa cruda*; (2) *Sputa cocta*; (3) *Sputa-muco purulenta*; (4) *Sputa pituitosa* or *sero-mucosa*.

1. **Sputa Cruda.**—Transparent, colourless or pale grey, like white of egg, or a solution of gelatin or gum, viscid, sticking to the bottom of the vessel, without smell, of a salty or somewhat metallic taste, mixed at times with a few streaks of bright blood, and more or less frothy with air bubbles. It presents but few formed elements, and these chiefly large mucous cells with a few small round cells, and now and then some columnar ciliated epithelium and a little squamous epithelium from the mouth. In chemical composition it is rich in mucin, and contains a little albumen; the salts are almost entirely chloride of sodium.

2. **Sputa Cocta.**—More opaque, of a yellow colour, and less viscid, it differs from the *sputa cruda* only in the larger number of cells, small and round, as well as epithelial, both in a condition of fatty degeneration to which the yellow colour is due. Not infrequently the yellow substance occurs in lumps or streaks swimming in a clearer fluid. In chemical composition it is essentially the same as the former.

3. **Sputa Muco-purulenta**, usually nummular, *i.e.*, in small lumps or pellets, some floating and others sinking in a viscid muco-serous fluid; sometimes this form of sputum is scanty, and consists of little else than these pellets in moderate number. It then closely resembles the sputum in some forms of phthisis.

Microscopical examination shows nothing beyond an overwhelming number of pus cells.

As compared with pus, sputum contains more mucin and more chlorine, and while in pus the salts are chiefly potash, in sputum they are chiefly soda.

In other cases it may not be nummular, but form a fairly uniform liquid often of a dirty colour, which, on standing, separates into layers, the upper frothy, from which ragged shreds hang down into the opalescent fluid of the middle layer, and the lowest opaque and green, like a sediment of pus, as indeed in great part it is, mixed with detritus.

In this form the sputum may be fetid, and in it are found stinking lumps, composed of the granular detritus derived from decomposing cells, crowds of putrefactive bacteria of all kinds, and, not infrequently, fat crystals.

As would be expected, chemical examination shows a number of the usual decomposition products, butyric, acetic and formic acids, ammonia and sulphuretted hydrogen, caproic acid, margarine, cholesterine and leucine.

4. **Sputa Pituitosa, S. Muco-serosa** (Bronchorrhœa serosa).—A generally abundant synovia- or saliva-like fluid, often frothy, and with a few shreds of mucus. This, the pituita of Laennec, is often very profuse. Besides occurring in one rather rare kind of bronchitis, it is occasionally met with in empyema and in acute miliary tuberculosis, and in smaller amount in the asthma humidum of emphysema and morbus cordis, as well as in whooping-cough.

I have seen this kind of sputum more than once in cases of pressure upon the trachea, by an aneurysm as well as new-growth. In these cases it is not as a rule mixed with air and might be mistaken for saliva.

Andral¹ quotes a case in which a sudden discharge of an enormous quantity of serous fluid from the bronchus occurred in a case of hydrothorax at the same time that the fluid in the pleura was being rapidly absorbed. This subject will be again referred to under the heading of Albuminous Expectoration.

Hæmoptysis.—The sputum in bronchitis very often contains blood, but usually, only in the form of bright red streaks upon its surface, due to the rupture of some small vessels in the larger bronchi, as the result of the violent coughing [*Streaky hæmoptysis*].

In morbus cordis, not infrequently small dark lumps are expectorated; these are formed almost entirely of altered blood, and come from the vesicles, into which some of the distended pulmonary capillaries have given way.

Hæmoptysis in either form has but little importance in bronchitis.

SYMPTOMS.—The access is usually gradual. It is characterised by general lassitude, aching or even rheumatic pains in the limbs. The patient is very sensitive to cold and complains much of chilliness, especially in the evening, but there is rarely any shivering severe enough to be called a rigor.

A cough often precedes the actual attack for weeks or months. It is rare for bronchitis to give no premonitory symptoms.

Cough is never absent. It is due to the irritation of the secretion, but it stands in relation not with the amount so much as with the difficulty of expectoration, being often most trying where the expectoration is scanty, and least troublesome where it is profuse. Where expectoration is difficult, the cough is often loud, ringing or barking, and may occur in paroxysms of even some minutes' duration. It is generally preceded and attended by a feeling of tickling or irritation referred to the trachea or to the upper part of the sternum, and an attack is easily determined by slight changes of temperature, as by passing from one room to another, whether it be warmer or cooler. It is occasionally brought on by eating, but drinking hot things relieves it. It is usually worse at night, and may be absent often during the whole of the day, to come on so soon as the head is laid upon the pillow; this is explained by the suggestion that in the prone position the secretion gravitates to the posterior membranous portion of the trachea—that is, to the most sensitive part.

¹ *Clinique medicale.*

Where the secretion, even if copious, is easily expectorated, the cough though frequent may not be noisy or distressing. The worst attacks often occur in the morning on waking, especially after a good night's rest. At this time it is sometimes so distressing that the patient prefers sleep broken by short fits of coughing to a good night with the violent morning paroxysm which follows waking, and experience often leads them to decline sleeping medicines on this account.

A severe paroxysm may lead to retching or even vomiting, to the involuntary escape of urine or wind and fæces, to epistaxis, to hæmorrhage into the conjunctiva or skin, to protrusion of a hernia, or to prolapse of the bowel or womb.

Pain.—Actual pain is unusual in bronchitis. There is often a feeling of *soreness* beneath the sternum, increased by coughing, or of *tickling* and *irritation* referred to the larynx, trachea or upper part of the sternum, but pain is absent, unless it be due to laryngitis, pleurisy, or to muscular strain from coughing. The muscular pain is generally referred to the edge of the ribs, or to the epigastrium, *i.e.*, along the attachments of the diaphragm and abdominal muscles, while that of pleurisy is felt higher up in the axilla, and this fact is of some diagnostic importance.

Dyspnœa.—Owing to the swelling of the mucous membrane and to the secretion in the tubes, the passage of air in and out of the lungs is impeded. This is met by increased rapidity of breathing. If the increased rapidity be not sufficient to compensate for the obstruction, the movements also become more vigorous. The accessory muscles then enter into play, and in order that they may act to full advantage the patient assumes the sitting position (*Orthopnœa*).

For orthopnœa, a certain amount of consciousness and muscular power are requisite, and it will be absent where these are wanting.

If the dyspnœa increase, the patient becomes very restless, tossing from side to side in the bed, now sitting up, now lying down, until at last, when exhaustion comes on, he lies prostrate upon his back with rapid shallow respirations, and passing gradually into a drowsy and unconscious state, dies at last comatose.

The breathing may, towards the end, become very irregular and assume the characters of Cheyne-Stokes's respiration. This is a very grave symptom under whatever circumstances it occurs. In bronchitis it is to be referred generally to a failing heart, and occurs as a rule only a short time before death.

The dyspnœa may come on in severe *paroxysms*, which are sometimes incorrectly called asthma. Of course, a bronchitic patient may also be asthmatic, but the paroxysmal dyspnœa occurring in bronchitis is not as a rule true spasmodic or nervous asthma, but is the result of the accumulation of secretion in the tubes and is relieved by its evacuation. The same is true of the paroxysmal dyspnœa of plastic bronchitis. In bronchitis of the aged and of those suffering from morbus cordis, the paroxysmal dyspnœa is often of cardiac rather than pulmonary origin.

Where the dyspnœa is severe, it is in the earlier stages when the strength is still maintained that the patients suffer most; in the later stages, they fortunately to a great extent lose consciousness of their sufferings.

Cyanosis.—This depends, as a rule, upon the amount of dyspnœa. It varies from a mere dusky flush in slight cases, to a deep purple colour where suffocation is imminent. The cyanotic tint is most marked on the lips, the tips of the nose and ears, and beneath the nails of both fingers and toes.

Deep cyanosis is rarely seen except where bronchitis is complicated with emphysema, and it may then be almost as extreme as in congenital morbus cordis.

When cyanosis is permanent, Traube taught that a fatty condition of the heart in all probability existed.

In other severe cases cyanosis is replaced towards the end, when exhaustion is setting in, by an ashy pallor; the skin becomes of a leaden colour, is cold and covered with a clammy sweat. This change, which is most frequently seen in children, may occur very quickly; it is of the gravest significance and usually portends death.

Fever.—The temperature rarely rises above 101 or 102, except in children, and in some cases, even as many as half according to Lebert, it may not rise at all. It is of no especial type, but is moderately remittent. There are often, especially in children, irregular exacerbations of fever which frequently end with sweating. Continued fever indicates severity. In the young and the old the fever is usually high, and it may then not be easy to exclude pneumonia, which is, as a matter of fact, not an uncommon complication.

When fever is present all the other symptoms of fever accompany it, the tongue is coated, the appetite lost, the bowels constipated, and the skin dry; the pulse and respirations are rapid, the urine is concentrated and deposits a sediment of urates, the cheeks are flushed, and there is often slight delirium, but all these symptoms may be present without any elevation of temperature at all.

The pulse is quickened, especially in children and old people, and that beyond what the fever would account for. It is at first full and the tension high, but later it becomes weak and ill-sustained, of low tension, and often irregular.

The heart's action is at first laboured, but later, in bad cases, becomes weak, irregular and fluttering.

The skin is often dry to begin with, especially if there be fever, but it soon becomes moist, or even sweating, and in the collapse stage is bathed in cold clammy sweat. The sweats when profuse are, however, not only sleep sweats, as in phthisis, but occur at all times of the day, and especially after coughing, and are most marked over the head and chest, especially in children.

The face.—The eyes are suffused, the cheeks flushed in the acute stage, cyanosed in severe cases, and ashy pale in the suffocative stage; in chronic cases venous stigmata are present on the cheeks and nose.

The digestive system hardly ever fails to be affected even in slight or in chronic cases.

The tongue is coated, usually with a moist creamy fur, but in children and old people it is often dry. A dry red tongue in old people, Gintrac thinks, indicates pneumonia, but this is not generally the case; it depends chiefly upon the patient breathing through the mouth, especially during sleep.

The appetite is impaired or lost, but vomiting is rare except in children.

The bowels.—Constipation is the rule, and may be very obstinate, but in children the attack is often ushered in with diarrhœa.

Gastro-intestinal catarrh is in young children as common a result of a chill as bronchitis is, and it is not therefore surprising that the two should be frequently associated, and that it should sometimes be difficult to say which is the more important affection.

The urine is scanty, of high specific gravity, deposits urates on standing, and it may sometimes contain a trace of albumen; but it may present all these characters even when there is no fever.

A trace of sugar has been detected during the acute stage, disappearing when this has passed. It is of very rare occurrence, but I have met with this transient glycosuria a few times.

Headache is common, especially at first. It then depends partly upon the condition of the stomach and partly upon the fever. In the later stages it is greatly due to the congestion and straining of coughing.

Delirium, usually of the quiet wandering type, is common in children and in old people, but only during the acute stage or towards the end.

The sleep is broken and unrefreshing, and often much disturbed by the cough.

The patient may be drowsy during the day and wakeful at night. Constant drowsiness is a bad sign in little children and old people, for it often indicates the commencement of carbonic acid poisoning. In children it may become so deep as to suggest the coma of meningitis.

Pain, other than headache and that already described in the chest, is not common, but Barthez and Rilliet describe a form of neuralgia, which they associate with bronchitis, round the orbits and occurring in paroxysms occasionally of great severity.

I have met with paroxysmal headache as severe as migraine in the course of a severe cold associated with bronchitis, which proved to be rheumatic and yielded at once to full doses of salicylate of soda.

Lastly, **Convulsion Fits**, which may commence the attack in children, may also in them usher in the end; but in adults suffocation rarely ends with convulsions.

The effect upon the heart.—In all but the slightest forms of bronchitis the circulation through the lungs is impeded, not so much from the bronchitis itself as from the obstruction to the entrance of air, and the consequent defective aëration of the blood; while in the chronic forms there is added the further impediment caused by the morbid changes produced in the lung tissue.

The first effect of the obstruction is to increase the tension in the pulmonary artery, hence the accentuation of the pulmonary second sound and the frequent reduplication of the second sound at the base. To overcome this increased resistance, the right ventricle beats more forcibly, and the first sound is prolonged over the right ventricle and occasionally also reduplicated.

At this time, the beats of the heart as well as the respirations are greatly increased in number.

The further effects depend upon the amount of obstruction and upon the rate of its development.

If the obstruction rapidly increase, as in a case of suffocative catarrh, the heart-beats increase at first still more in rapidity, but at the same time become irregular and less powerful, losing in force as they gain in frequency. Gradually, as exhaustion sets in, they become slow and fluttering, and at the last the heart is paralysed by over distension and stops in diastole.

If the obstruction develop gradually, as in chronic bronchitis, the right side of the heart, upon which the extra work is thrown, hypertrophies, and thus compensation may be complete; but compensation, being a process of growth, requires time.

The healthy heart has ample reserves of force from which it can draw when demand is made upon it, but these reserves are not unlimited, and if part of them be used for growth, less is left for emergencies; hence the explanation of the paradox that a hypertrophied heart is a weak heart; though apparently strong, it is actually weak, for it cannot stand a strain. These reserves are at their maximum in the young adult, and are small in little children and old people. Thus in any case of chronic bronchitis where hypertrophy has occurred and compensation been complete, a very slight cause may be sufficient to render

compensation inadequate, owing to the small reserves left, and the signs of cardiac failure will then appear. The final breakdown may result from causes which are not to be found in the lungs or heart at all, such as over-fatigue, bodily or mental, or an illness of some general kind, a pregnancy and so forth.

As soon as compensation ceases to be complete, and the heart becomes unequal to its work, dilatation occurs, those parts dilating most upon which the chief strain falls. Thus, in the case we are considering, the right ventricle dilates first, then the right auricle and large veins near the heart. These conditions may be recognised during life by the increase of the cardiac dulness to the right, by venous and epigastric pulsation, and if the dilatation be extreme by a systolic murmur. The congestion then slowly spreads backwards:

1st. Along the portal system, leading to enlargement of the liver and ascites, and often associated with gastro-intestinal disturbance and piles.

2nd. Along the systemic veins, as evidenced by œdema of the extremities, especially of the feet and legs, and by albuminuria. The earliest symptom which the patients themselves observe is usually swelling of the feet, but the liver is often much enlarged before the feet swell, and may give so much pain as to be the cause of chief complaint. In these cases the hepatic enlargement and pain often greatly diminish when œdema or ascites develop.

As the heart grows weaker the œdema slowly spreads upwards to the thighs and trunk, then to the abdomen and chest, and the patient dies at last waterlogged, almost exactly as if the victim of mitral disease.

When the right heart fails, to whatever cause that failure be due, the venous congestion to which it leads affects the lungs and the heart itself as well as the rest of the body.

The effect of venous congestion upon the lungs.—The bronchial veins, opening as they do into the vena azygos and superior intercostal vein, will very early feel the effect of venous congestion, and the obstruction to these veins will still further aggravate the bronchitis which exists. The congestion may relieve itself to some extent through the anastomatic communications with the pulmonary veins, and thus part of the blood may reach the left side of the heart without passing through the pulmonary circulation at all, as is especially the case where there is much emphysema. This mixing of the arterial and venous blood may be a part cause of the considerable cyanosis which occurs in some cases.

The effect of venous congestion upon the heart.—The coronary veins will feel the congestion still earlier, for they open into the right auricle itself, and as the result the circulation through the heart will be impaired and the nutrition of the whole organ will suffer, on the left side as well as on the right side. The effects will, of course, be most marked in the parts upon which the heaviest work falls, *i.e.*, on the right side, and consequently fatty change, which is the pathological evidence of overwork, is often found to be most advanced on the right side.

As soon as the left ventricle fails, a fresh cause of pulmonary congestion is added, for there can be no greater obstruction to the circulation than a left ventricle which cannot drive the blood onwards. The pulmonary veins become congested, and the aëration of the blood is still further interfered with. This adds to the congestion of the right side, embarrasses still more the coronary circulation, and by further impairing the nutrition of the heart makes the left ventricle weaker still.

The vicious circle thus established explains the rapid failure of the heart, which is so striking a feature in the later stage of many cases of bronchitis.

Peacock showed that as the result of bronchitis—

(1) Hypertrophy and dilatation of both the right and left ventricle occurred, the cavity of the right ventricle being enlarged and the walls increased in thickness, the left ventricle being increased in size, but retaining the usual thickness of its walls.

(2) The pulmonary orifice was wider than normal.

(3) The fossa of the foramen ovale was much enlarged, and might even bulge. He thought it might possibly even become patent again when the valve is not adherent, but had never actually met with this.

I have met with one instance of this in a woman, in whom the extreme cyanosis suggested a congenital affection of the heart. Such a diagnosis was not made, as except for the cyanosis there was no evidence of it. On the *post-mortem* examination the foramen ovale was found closed completely in the upper part, but for about a quarter of its circumference at the lower part the membrane was unattached and formed a sort of valve, which under ordinary conditions no doubt closed the orifice, but under the altered conditions of pressure on the right side of the heart allowed the passage of blood from the right to the left auricle.

Peacock's¹ measurements show that dilatation of the orifices of the heart occurs where there has been long-standing obstruction to the circulation either in the heart itself or in the lungs, but it does not follow necessarily that there must therefore have been regurgitation, for the valves may be able also to stretch and adapt themselves to the change, and thus still be competent to close the orifice.

The result upon the heart is evidenced during life by the signs of dilatation, the most constant being displacement of the apex to the left with epigastric and venous pulsation. In spite of great dilatation the cardiac dulness is often entirely masked by the emphysema which has developed.

Murmurs are common.

A *tricuspid murmur* is spoken of as frequent, but this I cannot endorse. Systolic murmurs audible over the right ventricle, especially at the bottom of the xiphoid cartilage, are not rare, but they are in most cases localised and not propagated so distinctly towards the right as they ought to be if they were really the result of tricuspid regurgitation, and as is observed in cases of organic disease of the tricuspid orifice.

In the same way *systolic murmurs at the apex* are not infrequent, but, again, they are usually audible only over the left ventricle and not in the axilla, and behind, as the common regurgitant mitral murmur is.

This is not the place to discuss the theory of dilatation murmurs, or to criticise the application to disease of the physiological observations of Ludwig and Hasse, which go to show that muscular action is necessary to complete the closure of the auriculo-ventricular orifices, and that where this is deficient regurgitation will occur. It will be enough to state the fact that these dilatation murmurs are very rarely heard behind; they are sometimes, but although my attention has been drawn for several years to this point, the two cases which follow are the only instances I have met with. I am therefore not ready, without further evidence, to assume that systolic dilatation-murmurs are always necessarily due to regurgitation.

John S., aged 41, a labourer, came as an out-patient to the chest hospital on 14th February 1879, suffering from severe bronchitis. He had suffered from winter coughs for many years, but had been worse for the last four months.

He was much cyanosed and had great dyspnoea. Examination disclosed general emphysema with severe bronchitis and abundant crepitation over the whole of both lungs. The cardiac dulness was masked by the emphysema, but the apex was about two inches outside the nipple. A loud systolic murmur was audible at the apex and for some distance into the axilla, as well as behind. This was thought to be due to mitral incompetence. Another murmur of the same

¹ *Weight and Dimensions of Heart*, 1854, p. 42.

character and also systolic was present at the xiphoid cartilage. This was attributed to tricuspid regurgitation, and the case was regarded as one of mitral disease with secondary bronchitis. The patient was admitted, and the signs remained unaltered for some days, but when, after a fortnight, the bronchitis subsided, the murmurs grew fainter, and on 11th March they had entirely disappeared both at the apex and at the sternum. The apex, though not so much displaced, was still outside the nipple. The patient was much better but still somewhat cyanosed.

When he left the hospital a few weeks later, the bronchitis being gone, the apex had returned to its formal place, the sounds were healthy and the murmurs absent.

On 10th December 1879 the patient came back with a similar attack, and was again admitted. The bronchitis was almost as severe as before, but the apex was not quite so far displaced and there was no apex murmur, but at the xiphoid cartilage a blowing systolic murmur was present, and in the epigastrium there was marked systolic pulsation and a slight presystolic thrill.

This attack was less prolonged, as it was also less severe. The murmur disappeared with recovery, and on leaving, the apex was again in its normal place.

On 6th February the patient was admitted for the third time, with most severe bronchitis and with the heart dilated more than ever. The apex was in the sixth space, three inches outside the nipple, and the apex murmur was as loud behind and in the axilla as over the apex. At the xiphoid cartilage there was also a loud systolic murmur. The patient was deeply cyanosed, and the veins in his neck were much distended and pulsated forcibly. The feet and legs were cedematous, and the abdomen distended with fluid. This time the patient did not rally, but died gradually, deeply cyanosed, on 3rd April.

The *post-mortem* examination was made the next day.

The body was cedematous, with considerable ascites.

The lungs very emphysematous, and exhibited the ordinary lesions of chronic bronchitis. They were adherent to the thorax wall in many places, the spaces between the adhesions being occupied by serous fluid. The heart was greatly dilated, measuring round the auriculo-ventricular sulcus twelve and a half inches, and from the base of the pulmonary artery over the apex to the auriculo-ventricular sulcus behind twelve inches. On section the right ventricle was enormously dilated, measuring from the pulmonary valves to the apex five and three-quarters inches, the walls being very thin and in most parts not more than a quarter of an inch thick. The left ventricle was also dilated, but was not much more than half the size of the right. The muscular substance was pale and fatty, especially on the left side. The valvular orifices were dilated somewhat, but the valves themselves were perfectly normal and seemed unusually healthy for a man of his age, and there were only one or two small patches of atheroma in the aorta.

The other organs were congested, but otherwise normal.

The patient died, it was clear, from cardiac failure, of which the physical signs had been distinct during life, viz., dilatation and murmurs. The dilatation affected both sides of the heart, the left as well as the right, though the signs of dilatation of the left ventricle which were so evident during life disappeared to a great extent, as is usual, after death. The fatty change so frequently associated with dilatation was found on both sides, though in this case most marked on the left. There was no disease capable of producing this fatty change in either the arteries, the kidneys, or the heart itself, and there was nothing left to associate it with except the condition of the lungs. The direct relation between the dilatation and the bronchitis is clearly shown by the history of the different attacks, which also demonstrates the order in which the cavities of the heart yielded—first the right heart giving the signs of dilatation and ending with the tricuspid regurgitant murmur; then the left heart, which, as in the second attack, remained for a time dilated without any murmur, but at last as the dilatation increased a murmur also developed. The systolic murmur must in this case at any rate have been truly regurgitant, for it had all the characters usually associated with mitral incompetence, being audible at the apex and also in the axilla and behind, differing in the last respects from the ordinary dilatation murmur, which is confined to the region of the left ventricle and is not propagated beyond it. As the bronchitis subsided the signs disappeared in the inverse order, first the left ventricle murmur and some of the left-sided dilatation, then the right side murmur and finally the whole dilatation.

I have only met with one other case in which, as the result of bronchitis, undoubted mitral regurgitation occurred without any organic valvular disease, and this was in a child fourteen years of age, also the victim of chronic bronchitis for years, and with some consolidation of the left upper lobes. She was admitted into the chest hospital with great dyspnoea and cyanosis, and with the signs of severe general bronchitis. At the apex of the heart a systolic murmur was heard, which was also audible in the axilla and behind, and, as in the previous case, the diagnosis was made of bronchitis secondary to mitral disease. But this diagnosis had to be modified when the apex returned to its normal place and the murmur disappeared as the bronchitis got better. The patient left the hospital without any evidence of heart disease, and none developed as long as she remained under observation.

PROGNOSIS IN GENERAL.—The prognosis differs so much in the various forms of bronchitis that it is difficult to make any statement of general value, either as regards risk to life or prospect of cure.

Bronchitis is most dangerous when it attacks the small tubes, but in all forms age is the most important factor in prognosis, the risk to life being so much greater at the extremes of life. Acute bronchitis is, of course, more dangerous than chronic, but it offers greater prospect of cure.

Much will also turn upon the previous health and actual strength of the patient, hence in the secondary forms the prognosis depends most upon the nature of the primary disease, and upon the extent to which the strength has been reduced by it.

When death occurs, if it be not due to some complication, it results either from asphyxia, from asthenia, or from failure of the heart.

These points in prognosis will be further dealt with in connection with the special forms of Bronchitis.

DIAGNOSIS IN GENERAL.—The physical signs are so characteristic that the diagnosis of bronchitis is easy. But when the term bronchitis is used, it implies that there is bronchitis only and nothing else, and there the difficulty lies, for bronchitis is often secondary or the sign of some other affection, and these other affections it is often by no means easy to exclude.

The conditions with which special forms of bronchitis are likely to be confounded will be dealt with in their proper place, and the means of diagnosis then considered. Perhaps the best point in diagnosis is this, that localised bronchitis, *i.e.*, bronchitis persistently localised in one part of the lung, is strong presumptive evidence in favour of a local lesion there. Thus bronchitis limited to the apex may be the earliest evidence of phthisis: when limited to one side is explained by some unilateral lesion, *e.g.*, widespread pleural adhesions or interstitial change in the lungs; when present at both bases is probably connected with hypostatic congestion, especially if associated with morbus cordis.

Considering the close relation in which the heart stands to the lungs, a careful examination of the heart should be made in all cases.

Even general bronchitis may be but an evidence of some more serious disease, *e.g.*, acute disseminated tuberculosis of the lung, but in this case the general constitutional disturbance and the hectic character of the fever will suggest the correct diagnosis.

In a similar way in typhoid fever, where the abdominal symptoms are slight, and the pulmonary symptoms pronounced, the diagnosis may be difficult. As a matter of experience, typhoid fever, acute tuberculosis, and bronchitis are not infrequently confounded for a time; and when the fever is high, the diagnosis may be still further complicated by the possibility of pneumonia.

Speaking generally, the diagnosis of bronchitis is easy, and the chief difficulty lies in determining whether the bronchitis is the primary and only affection, or secondary, and a complication or consequence of some other disease. The best way to avoid mistakes is to bear these possibilities of error in mind.

THE TREATMENT OF BRONCHITIS IN GENERAL.—The general indications for treatment are clear:

1. To remove the causes which have excited or predisposed to the disease or which maintain or aggravate it.
2. To remove, control, or modify the pathological processes which accompany or directly follow the disease, and the symptoms which result from them.

3. To treat the general conditions to which the disease may give rise and any complications with which it may be associated.
4. To take such measures of prophylaxis as will prevent the disease developing where predisposition exists, or if it has already developed, will prevent its recurrence.

The ways in which these different indications can be best fulfilled will vary greatly in the different forms of bronchitis. It will be convenient in the first place to consider the general lines of treatment which may be followed, and subsequently, as the special forms of the disease are dealt with, to indicate in each more particularly those from which advantage may be expected.

The bronchi are accessible to direct medication only through the air inspired; and this may be modified in respect of its temperature or of the amount of moisture it contains, or by the addition to it of therapeutic remedies, either in the form of gases or of the vapours of volatile substances, or in the form of finely divided powders or sprays.

Gases or vapours mixed with the air are easily carried, of course, to the most distant parts of the respiratory organs. Powders and sprays are chiefly deposited in the mouth and pharynx, and though they may reach the most distant parts of the respiratory organs, their use is more uncertain and less under control.

The modifications in the air may be effected either in the general atmosphere surrounding the patient, *i.e.*, in the room or in a tent surrounding the bed, or only in the air actually inspired by the patient by means of some form of inhaler. In the latter case the medication is intermittent, while in the former it may be continuous night and day.

For the general medication of the air of the room, if the drugs be volatile at ordinary temperatures, it will be sufficient to expose them in saucers or upon sponges; if heat be necessary to make them volatile, they may be placed in saucers upon a water bath, or sand bath, or over a spirit lamp. If, as in most cases, moisture be required as well, the common bronchitis-kettle answers every purpose, either placed on the fire or heated by a lamp, the various medicaments being added to the water. For stronger medication the steam-spray is useful.

Inhalation.—For simple inhalation the apparatus varies much with the nature of the substance used. For those volatile at ordinary temperature a very simple and effective inhaler may be made out of a piece of glass tubing, 3 or 4 inches long and of about $\frac{3}{4}$ of an inch bore, with or without an open bulb on it. This may be lightly stuffed with cotton wool, sponge, or even blotting-paper, soaked with the substance selected.

Another simple form consists of a double respirator, between the two halves of which cotton, wool, sponge, or flannel is placed, similarly impregnated.

For the inhalation of warm moist air, impregnated or not with drugs, it is often sufficient to take a jug or can filled with boiling water, and after covering the mouth and nose, as well as the jug, with a towel, to breathe in the vapour which rises; or one of the common forms of inhaler may be used, in which the air is drawn over or through the water, the temperature varying with the drug employed.

Lastly, if a spray be employed the common hand apparatus used for æther will be sufficient for an air-spray, while Siegel's apparatus is the most convenient for the steam-spray.

Temperature.—The air inspired, unless it be very cold, is returned on expiration raised to the temperature of the body.¹

Temperature of the air inspired.	Temperature of the air expired.
°C.	°C.
-6.3	+29.8
+17 to 19	+36.2 to 37
+41	+38.1
+44	+38.5

¹ Landois and Stirling, p. 270.

In health great variations in the external temperature of the air can be tolerated, but that is not so in disease, and the temperature of 60° to 65° F. is that which experience shows to be on the average the most comfortable to the patient.

It is also important that the temperature should be kept uniform, and that by night as well as day, *i.e.*, that no sudden changes of temperature should occur. This is especially to be guarded against if the air be at the same time kept moist, for then a damp chill is likely to be produced which may be very detrimental.

Although the temperature of 60° to 65° is the best for the air surrounding the patient, still by means of inhalers the air inspired is often presented at a higher temperature than this. It is then usually impregnated with moisture or mixed with the vapour of drugs, so that the simple action of hot dry air has not been fully investigated. Cold air has been rarely used, but it has been administered at a temperature below zero, and is said in some cases of chronic bronchitis to have relieved the cough and other symptoms for a time. Its use is risky, for in most cases cold air aggravates the disease.

Moisture.—Air is most pleasant to breathe when it contains from 50 to 70 per cent. of aqueous vapour, complete saturation being reckoned as 100; with more than 70 per cent. the air feels too moist, and with less too dry. It is this degree of saturation that it is desirable to maintain in the air surrounding the patient.

A saturated atmosphere is not pleasant at any temperature, and a fall of a very few degrees will then lead to the production of a damp and dangerous chill.

Fortunately it is not easy to saturate the air of a room completely. When, as in the ordinary sick room, a bronchitis-kettle is used, the steam does not do more than make the air agreeably moist, something will depend upon the external temperature, for if the day be cold the air when raised to 65° becomes relatively dry and the steam added remedies this, but if the day be warm and moist the kettle is often found too much and is discontinued.

If the room be large, as in the ward of a hospital, the result may be obtained by surrounding the bed by a half tent and allowing the kettle to steam gently under it.

There are grave objections to enclosing the bed almost entirely, for under these circumstances the air becomes completely saturated, and the moisture condensing hangs in drops upon the inside of the tent. In such a tent with its saturated atmosphere the patients often lie in great discomfort. I have seen many cases, which had long remained *in statu quo* when treated in such a tent with steam, improve at once rapidly as soon as the tent was converted into a half tent or the kettle removed. There is no doubt that the treatment with such a saturated atmosphere had in these cases done harm.

The medical substances used for inhalation fall into two groups according as they are volatile at ordinary temperatures or not. They may all of them be used either as *moist* or *dry* inhalations, *i.e.*, mixed with water or not. In action they are for the most part stimulant and somewhat antiseptic, or sedative.

Iodine in concentrated form is a violent irritant, but when well diluted acts as a powerful stimulant as well as an antiseptic, and if absorbed as an alterative and resolvent, it is found useful in some of the chronic forms of bronchitis.

For dry inhalation 10 drops may be inhaled through a tube or respirator. Iodine is not often inhaled alone but in conjunction with other drugs of similar action.

For moist inhalation the ordinary Vapor Iodi is used, consisting of one drachm of the tincture with one ounce of water placed in a suitable apparatus and warmed. More often weaker solutions are employed, *e.g.*, half a drachm to a drachm in half a pint of warm water at about 100° F.

Iodide of Ethyl (M x-xv) inhaled from a handkerchief or respirator several times daily is useful in chronic bronchitis, especially in asthmatic forms. It renders the breathing deeper and easier, is somewhat exhilarating and has no soporific anæsthetic or depressant effect. Containing as it does four-fifths of its weight of iodine, it forms a rapid means of saturating the system by absorption with iodine.

Creasote is stimulant and antiseptic. **Dry.**—About 10 drops for each inhalation. **Moist.**—One or two drachms may be added to the water in a bronchitis-kettle for general medication, or for ordinary inhalation, half a drachm to a pint of boiling water.

The next following drugs have all a similar action to creasote:—

Carbolic Acid.—**Dry.**—About 10 drops for each inhalation. **Moist.**—Half a drachm to the pint of boiling water, or 2 drachms to the pint of water at 80°–100° F.

<i>Turpentine</i> <i>Oleum Pini Sylvestris</i> <i>Do. Pumilionis</i> <i>Eucalyptol</i> <i>Thymol</i> <i>Benzoin (Tr. Benzoini Co.)</i>	}	<i>Dry.</i> —20–30 drops at a time. <i>Moist.</i> —1 or 2 drachms to the pint of water at 140° F., usually with a drachm or so of light carbonate of magnesia added.
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Camphor.—Stimulant. The most convenient preparation is the Spiritus Camphoræ. *Dry.*—10–15 drops. *Moist.*—A drachm to a pint of water at 140° F.

Ammonia.—Stimulant. *Dry.*—5 drops of the Liquor Ammonia (B.P.). *Moist.*—A drachm to the pint of water at 80° F.

Sulphurous Acid.—Antiseptic. *Moist.*—1 drachm of the acid (B.P.) to the pint of water at 80° F.

The following drugs are sedative in action, and are most useful when the cough is violent:—

Chloroform, Aether, and Acetic Aether may be inhaled pure, but are generally diluted with rectified spirit, 1, 2, 3, or more parts and combined with other drugs. If for moist inhalation one drachm is mixed with a pint of water at 80° or 100° F.

Conium.—One or two drachms of the Succus Conii to the pint of boiling water, or the Vapor Conii (B.P.) may be used.

Hydrocyanic Acid.—A drachm of the dilute acid in the pint of water at 80° F.

Opium is sometimes employed as a vapour, three grains of the extract being heated and the vapour inhaled, but this is not a satisfactory way of administering the drug.

As a moist inhalation the extract may be mixed with hot water, or the Tr. Camphoræ Co. may be used instead. In the latter, the camphor and other constituents are the really active remedies.

Chloride of ammonium fumes are useful in all forms of chronic bronchitis where the secretion is abundant.

The fumes may be obtained—

1. By heating some of the salt in a spoon and inhaling the products.
2. By placing some ammonia in a saucer, in the centre of which a watch-glass is supported, containing fuming hydrochloric acid; and
3. By using a special form of apparatus in which the ammonia and hydrochloric acid fumes are produced in separate vessels and mixed in a third from which the inhalation is made.

For dry inhalation as stated, a combination of drugs is preferred, of which the following is in common use, or some modification of it:—

Tr. Iodine Carbolic Acid Aether Spirit	}	aa 2 parts.
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Creasote, Thymol, Eucalyptol, or Turpentine, etc., one part.

Sixty drops of this may be sprinkled on the sponge or cotton wool of an appropriate respirator, and the inhalation employed either continuously or for half-an-hour at a time several times a day. A respirator worn at night with such inhalation may greatly allay the cough and promote sleep, but it must be lightly packed so that the breathing may be quite easy through it, otherwise it will not be tolerated.

Oxygen.—Inhalation of oxygen is usually reserved for those cases in which there is much cyanosis, but as the cyanosis depends upon the mechanical obstruction offered to the entry of air into the vesicles of the lungs by the secretion in the tubes, and as there is the same obstruction to the entry of oxygen, the use of oxygen in such cases is very disappointing.

In less severe cases the inhalation of oxygen often does good (1) by improving the general aëration of the blood, (2) by its stimulant effect upon the circulation, as shown by the greater regularity and force of the heart's beat, and by the improved tone of the pulse, (3) by its general sedative effect, so that under its action the patient often loses the constant restlessness which has been so distressing, and gets snatches of quiet and refreshing sleep.

Atomization, Nebulization, Sprays.—There is no doubt that sprays are for the most part deposited in the mouth and pharynx, and that only a very small part at all reaches even the larynx. This, according to Riegel, is not more than about $\frac{1}{3}$ to $\frac{1}{12}$ part.

If the air-tubes be free they may, however, reach even the most distant parts of the lungs, as has been demonstrated by Demarquay,¹ by the use of an inhalation of perchloride of iron, for which he tested with cyanide of potassium. By these means he was able to show the presence of the inhalation in the vesicles of the lungs, as Lewin² also did in a cavity.

When sprays are so applied as to reach the bronchi they may have a direct action upon the mucous membrane, but this can hardly be much, for stronger solutions are necessary for this purpose than it is safe to use as sprays. The local action is more probably due to local absorption, but as most of the drugs employed have a similar action on the mucous membrane if administered in the usual way by the mouth, and as a large quantity of the spray is almost inevitably swallowed, it is impossible to apportion to each its due share of action.

If the spray is to reach the air-tubes, much depends upon the method of inhalation, and this is not easy for a patient to acquire. The head should be thrown back slightly, the mouth be kept widely open, and the breathing be deep and easy. If a funnel for the mouth be used, care should be taken that the tongue does not arch up behind it and partially close its aperture.

The first effect of an inhalation, especially if an astringent solution be employed, is to produce a feeling of tickling and cough with a sense of tightness and oppression beneath the sternum, which soon passes off. The first inhalation should not last longer than 5 or 6 minutes, but later ones may be extended to 15 or 20 minutes. Astringent sprays are not altogether harmless at times, for they may, it is stated, determine an attack of asthma or even hæmoptysis, but I have never myself seen either of these results.

The spray may be produced either by means of air or steam. For the former the ordinary hand spray is sufficient, and for the latter some form of Siegel's apparatus is the best.

If steam be used it does not materially dilute the solution employed, for it is almost entirely dissipated in the air and does little more than saturate the air inhaled at the temperature of the room. The duration of the inhalation is better determined by the quantity of solution used than by time, and for most drugs of the strengths commonly employed one ounce is sufficient for each inhalation.

Sprays have been administered as astringents, stimulants, antiseptics, alteratives, and sedatives.

The list of remedies used at different times in the form of spray is far too long to reproduce with advantage, but some of those most frequently used, with the strength per ounce, are given below.

As astringents.—Sulphate or perchloride of iron (gr. i-x); sulphate of zinc or copper (gr. i-x); alum (gr. x-xx); tannic acid (gr. x-xv); ergot (℥ x-xx).

Most of these solutions are too weak to produce any local action at all.

As stimulants.—Oils of turpentine, pine, cade, etc., creasote, carbolic acid, eucalyptol, thymol, etc. (℥ i-v); salicylic acid (gr. x-xx); tt. of iodine (℥ i-xx); iodide or bromide of potassium (gr. i-x); chlorinated soda and chlorine water (℥ v-lx); common salt (gr. xx-lx); nitrate of silver (gr. $\frac{1}{2}$ -v).

As alteratives.—Carbonate or bicarbonate of soda or potash (gr. i-x); chloride of ammonium (gr. xx-lx); the natural saline, alkaline or sulphur waters; lime water pure or diluted to one half; sulphurous acid (℥ x-xx); glycerine (3 i); even corrosive sublimate (0.01 per cent. = gr. $\frac{1}{200}$).

¹ *Gaz. Médic. de Paris*, 1861, p. 616.

² *Die inhal. therapie*, Berlin, 1865.

As sedatives.—Ipecacuanha wine or Tr. camphoræ co., pure, or diluted to one half; Tr. of hyoscyamus or belladonna (m ii-x); even oils in emulsion (m x-xxx) with solution of gum arabic. Parolein, a fluid, odourless petroleum, is an excellent medium for many of the substances used as sprays, e.g., menthol, pinol, eucalyptol, camphor, etc., 1 in 10; carbolic acid, 1 in 20; iodoform, 1 in 60.

Atomization is an unsatisfactory method of treatment in bronchitis. Many of the solutions are far too weak to produce the actions claimed for them, even if it could be certain in a given case that they reached the parts affected in sufficient quantity. The more powerful drugs are unsafe, as it is impossible to know what amount of the solution will be swallowed, and most of the remedies can be better administered in other ways.

I do not, therefore, advise the use of sprays under ordinary circumstances in bronchitis; the other methods of inhalation are far preferable.

Intratracheal Injections.—These are rarely used in ordinary bronchitis, though they have been employed a good deal in the bronchitis of phthisis, but rather with a view to the treatment of the lung than of the bronchial affection.

Expectorants.—This is a group of remedies which it is not easy to define. Strictly speaking every method of treatment which leads to increased expectoration may be called expectorant, whether the result be due to an actual increase in the amount of secretion or to the greater ease with which it is brought up. Other remedies are often included in this group, which decrease the need of expectoration either by diminishing the secretion or by mechanically expelling it, such as astringents and emetics, but these remedies are better treated of under other headings.

When, as in acute bronchitis, the mucous membrane is congested, and the secretion diminished, remedies which relieve the inflammatory tension and increase secretion will give great relief, but when, as in chronic bronchitis, the mucous membrane is lax, and the secretion profuse, remedies which tone or brace up will do most good. Accordingly the expectorants fall into two groups, the *sedative* or *depressant* and the *stimulant*; the former diminishing vascular tension and increasing secretion, and the latter increasing vascular tension and for the most part diminishing secretion.

As a general rule it may be laid down that expectorants of the first class are to be employed in the early stages of acute bronchitis, and those of the second class in the later stages or in the chronic forms of the disease, but as these two conditions merge gradually into one another, there is scope for great variety in the combinations of drugs employed.

The chief *sedative* or *depressant expectorants* are antimony, ipecacuanha (emetine), lobelia (lobeline), jaborandi (pilocarpine), and apomorphine.

Their general action is to increase secretion, and to reduce blood pressure.

Antimony has long been, and I think deservedly, a favourite remedy. Stokes taught that no other remedy has such a power over acute bronchitis. The preparations available are the oxide, the sulphide, and the tartrate. The two former are but little used in the present day, the tartrate is chiefly employed in the form of the Vinum Antimoniale.

Antimony acts powerfully on the mucous membrane of the bronchi and on the skin. It is most useful in those cases of bronchitis where the symptoms are acute, and the skin hot and dry.

In full doses it acts as a powerful cardiac depressant, and produces faintness, nausea, and vomiting. On account of its depressant effect, and its action on the intestines, it is contra-indicated in children and in cases where there is gastro-intestinal disturbance.

In small doses it is frequently added with advantage to the prescription in many of the chronic forms of the disease where the expectoration is copious, frothy, and difficult to expel.

Antimony when freely administered may cut short an acute attack, as in a case described by Wood,¹ in which small doses were given at short intervals (a quarter of a grain every fifteen minutes), until vomiting was produced, this being followed by a good meal and stimulants, with the result that the attack aborted. Such free administration of antimony, however, requires careful watching.

Stokes recommended its administration in such cases with opium, as Laennec proposed; for example:

Antimonii Tartr., gr. vi.
Tt. opii. ℥ xii.
Aquæ cinnamoni ad ℥vi.

One tablespoonful every hour or two hours, until the whole be taken, if possible within the twenty-four hours.

This treatment sometimes produces vomiting and purging, sometimes nausea only on moving; sometimes tolerance is established, but improvement occurs in all cases.

Although such active treatment may cut short the attack in its initial stage, it more frequently only rapidly induces the second stage, when stimulation will be found advantageous.

Ipecacuanha is also a very useful expectorant, and may be given to children, and in other cases where antimony is unsuitable. It increases the secretion from the mucous membranes even in small amounts, though some authors have stated that this occurs only with nauseating doses. It is the best expectorant for children, but the dose administered is often too small.

The pharmacopœia preparations are the powder (1 to 2 grains) repeated from time to time, and the wine, the last being the favourite form of prescribing it.

The Pulv. Ipec. Co. contains opium, and the Pil. Ipecac. C. Sc. squill and ammoniacum also; they are both very good preparations, but are administered more for the opium they contain than for the *ipecacuanha*.

Lobelia is but little employed except in bronchitis associated with asthma. In full doses it is a very depressing and uncertain drug. It may be given in small doses of the tincture (℥ x-xx) every two hours or so, but it must be carefully watched.

Jaborandi, or its alkaloid *pilocarpine*, is useful in some cases, though it is stated that its action on the skin and salivary glands makes it unpleasant to patients. I have frequently given *pilocarpine* in small doses ($\frac{1}{4}$ of a grain of the nitrate two or three times a day) with advantage; it seemed to me to loosen the secretion, and I have not seen it produce any disagreeable symptoms.

Apomorphia is more often given as an emetic *sub cutem*, but in small doses it is of use as an expectorant (administered by the mouth).

The stimulating expectorants raise the blood pressure and stimulate the mucous membrane. Their action on the expectoration varies, some increasing it and others diminishing it.

Senega is one of the most powerful of this group, and it is given in the later stages of bronchitis or in the chronic forms; if it be used in the more acute stages it is best combined with Vinum Antimoniale. Its taste is not agreeable, but it may be in part disguised by spirits of chloroform.

Senega contains a large quantity of saponine upon which its effect chiefly depends, and the presence of which, in *Saponaria* and in *Quillaia*, explains the use of these drugs in bronchitis.

Chloride of ammonium is useful in the later stages of bronchitis both in adults and children, but it should not be given until the inflammatory action has been reduced by the sedative expectorants. It is best adapted to cases where the secretion is abundant and the strength low, and is of special service in the broncho-pneumonia of children. It may be given in doses of 5 to 10 grains in liquorice water.

The carbonate of ammonia is a diffusible cardiac stimulant, and though often well combined with stimulant expectorants, has only an indirect action on the bronchi through the circulation.

Scilla is more often employed than almost any other drug of this group, both in acute and in chronic cases, but it is of especial benefit in the subacute and later stages, where the secretion is scanty and tenacious. If given in full doses for long it may impair the digestion and appetite.

It may be prescribed as the tincture or the powder. For children the Acetum Oxymel and Syrup are favourite preparations, but they are of course incompatible with carbonate of ammonium. The Pil. Scillæ Co. is a good combination with ammoniacum; and the Pil. Ipecac. C. Sc. is given for the opium it contains.

Coxe's hive syrup was a popular American remedy, consisting of equal parts of the syrups of squill and *senega* with a grain of tartrate emetic in the ounce. In full doses it acted as an emetic, and was sometimes given to children for this purpose.

¹ *Therapeutics*, 1874.

Resins and oleo-resins.—Although resin is of itself a slight stimulant to the mucous membrane, still the activity of the pharmacopœia resins depends chiefly on the volatile oils and other substances they contain. They fall into two groups—the balsams, which contain a large amount of benzoic acid, and the oleo-resins, which contain none.

Balsams.—*Benzoin* contains 12 to 20 per cent. of benzoic acid. It acts upon the skin and all mucous membranes, but especially upon that of the bronchi. It is generally given in the form of the compound tincture, *i.e.*, the old-fashioned friar's balsam. This has been long a popular remedy, and it deserves its reputation. It may be prescribed in water as an emulsion, upon sugar, or in the form of lozenges.

The balsam of Peru contains besides benzoic acid some cinnamic acid, and is an active remedy, but though more powerful is not so agreeable to take as the balsam of Tolu, which, on that account, is more often used.

Oleo-resins owe their activity to the volatile oils they contain. These oils are excreted by the mucous membranes generally, but freely by the lungs, and many give their odour to the breath.

Turpentine is not used in the crude state, but in the form of the oil.

Oleum Terebinthinæ is a powerful stimulant and irritant, and though not palatable is useful in some chronic forms, especially where the secretion is profuse and purulent, *i.e.*, in broncho-blenorrhœa, but some of the other pine oils are preferable, such as the *Oleum Pini Sylvestris* or *Pumilionis*. They may be administered in emulsion (10 to 15 drops for a dose), or like terebene, in water, and in a lozenge.

Copaiba and *cubeba* were once much used but have gone out of fashion, although Trousseau especially speaks highly of *copaiba*.

Ammoniacum contains but little volatile oil, and has only a feeble action. It may be given as the *Mistura Ammoniaci* (B.P.), and is one of the components of *Pil. Scillæ Co.* *Santal oil*, \mathfrak{m} v, 3 to 5 times daily in emulsion. *Myrtol*, \mathfrak{m} v, in capsules.

Garlic and *onion* used to be favourite popular remedies, especially for children, both as poultices and internally, but are not much used now.

Myrrh and *anise* have been also recommended for chronic bronchitis, and *Grindelia robusta* for those cases which are associated with asthma.

Another group of stimulant expectorants is formed by tar and its derivatives.

Tar is a very complex substance containing a number of volatile bodies, many of which have an action on the mucous membrane, but the various preparations of tar are now being gradually superseded by those of its derivatives, of which creasote is the most important.

Creasote may be given in solution in spirit (1 in 40) or in cod-liver oil, and as a lozenge with balsam of Tolu. As an inhalation it has been already referred to.

Tar water was an old-fashioned remedy made by shaking up tar with water (1 part in 10) and decanting after standing.

Tar may be administered as the Water (1 part in 10), as the Syrup (tar 1 part and sugar 25), or in the form of Pills or Perles.

Demulcents.—*Demulcents* are preparations of a gummy, starchy, or saccharine nature, which, applied locally to a part, have a direct soothing effect. When taken for bronchitis or catarrh this action can be only produced upon the pharynx, but when administered warm, as they usually are, they combine the local effects of heat upon the pharynx and adjacent parts of the respiratory tract, with the stimulating effect of heat upon the circulation in general, and upon the skin and kidneys. *Hot demulcent drinks* are therefore part of the routine treatment of acute catarrhs of any kind, and are very useful in acute bronchitis. Hence the use of hot milk, thin gruel, and arrowroot, linseed tea, black currant tea, etc.

When taken cold they produce little action, unless made sharp with acetic or citric acid, as in the various oxymels; but they often serve as vehicles for other remedies, *e.g.*, decoction of althæa and liquorice; and as demulcent drinks, barley water, or solution of Iceland moss, are in common use.

In the form of *jujubes* demulcents are of great service in allaying cough, and offer a good basis for the administration of other drugs.

Derivatives are methods of treatment based on the theory that internal congestion may be relieved by drawing away the blood from the organ affected to

other parts of the body. On a large scale this is only possible by acting on two organs, viz., the skin and the intestines, in which the system of vessels is large enough to contain for the time almost the whole blood of the body.

The effect produced is, however, not due to the simple mechanical detention of blood in the dilated vessels, for it has been shown that the dilatation of the superficial vessels is attended by active contraction of the deep vessels, and that this is a reflex phenomenon. The deep-seated contraction, moreover, bears, it appears, a fairly definite relation in distribution and extent to the superficial dilatation, being widespread where this is extensive, and limited where it is local, and in the latter case the effect is most marked in those parts which bear a direct anatomical relation to the surface acted on.

Derivatives acting on the skin belong with few exceptions to the class of *irritants* or *counter-irritants*, and when applied more or less extensively to the thorax are of great service in bronchitis. What is required is an irritant strong enough to produce well-marked and not too transient dilatation of the vessels of the skin. Most of the remedies used belong to the group of rubefacients; the vesicants, *i.e.*, the more violent irritants, which produce blistering, are too powerful, and are rarely used, though Stokes states that they do good in the later or chronic stages of the disease.

Rubefacients.—One of the simplest and best is *heat*. This may be applied either in the form of hot, dry cloths, or of cloths wrung out in hot water, or as *poultices*, but as they all cool rapidly and require to be constantly changed, they are usually combined with other rubefacients such as mustard or turpentine. (A tablespoonful or two of mustard to a fairly large linseed poultice is sufficient to prolong the stimulating action, and to make the poultice feel warm and comfortable for some hours.) Poultices, if of any size, are now almost invariably made of linseed meal, but, when small, may be of bread.

To make a poultice.—First lay out upon a flat surface, a board or table, the towel or paper upon which the poultice is to be spread. Then into a basin of sufficient size, previously warmed, pour some boiling water enough for the whole poultice, and into it sprinkle the meal slowly, stirring rapidly all the time, so that no lumps may form, until the right consistency has been reached. Then spread it quickly out on the towel with a spatula (a large carving knife or paper knife will do well). Sprinkle over the surface a few drops of oil to prevent the meal sticking to the skin, and apply at once. If the patient is not quite ready, roll the poultice up and place it before the fire or in the oven to keep it hot.

Mustard may be added in any proportion. The common Cataplasma Sinapis consists of equal parts of mustard and meal, but if made with the white powdered mustard it is very strong and often blisters. It should not be applied for more than twenty minutes. If a single sheet of thin paper be placed upon the surface, so that the meal is not in direct contact with the skin, the poultice can be applied hotter, and will retain its heat longer, while its effect will not be impaired. Mustard must always be used with caution in children, for they blister readily, and a blister may leave a very troublesome sore; and again in young women with tender skins, for in them the application of a mustard leaf or strong poultice may leave behind it a pigment-stain which may take months to disappear. If the patient be restless or suffer from orthopnoea, it is often not easy to retain the poultice in place. When the patient is sitting up it is best supported from the shoulder by a shoulder strap, or by bringing the poultice over it. A towel carried round the chest and pinned in position is better than a bandage to keep the poultice in place, and if supplied with shoulder straps is all that is wanted.

A large double or jacket poultice, it must be remembered, is of considerable weight (1½ to 2 lbs. it may be), and is often more than patients seem able to bear, if they have much dyspnoea.

The poultice may be replaced by *spongiopiline* or *folded flannel*, cut like a waistcoat, but open at one side and at the shoulders, where it may be tied with strings. This may be wrung out in hot water or hot mustard and water, or in a decoction of capsicum (℞ i to O i), but it is better kept dry and sprinkled with some rubefacient preparation, as turpentine, spirits of camphor, or the domestic spice tincture.¹ The camphor is a very pleasant preparation and not so irritating as turpentine.

¹ Two ounces of unground ginger with one ounce each of cloves, cinnamon, and chillies are placed in a bottle, and one pint of whisky added. After standing for some days, the supernatant fluid may be poured off and is ready for use.

The jacket is very light and warm and may be left on for hours at a time, requiring only to be refreshed with the preparation two or three times in the day.

When the poultice or spongiopiline is removed, the chest must be enveloped with cotton wool or flannel, as it will be for some days very susceptible to cold.

A small mustard plaister or a mustard leaf applied over the sternum or between the shoulders gives much relief in the early stages of an acute attack, especially when the cold has affected the large tubes only. For gentler counter irritation the emplastrum calefaciens (containing 1 in 25 of cantharides) is useful, while as a protective a simple pitch plaister may be worn.

Friction, whether with the hand, a rough cloth, or a brush, is an excellent rubefacient, but its effect is transient. It is more commonly combined *with some stimulating liniment*. It is a popular and efficacious method of treatment, especially for little children. There are several excellent rubefacient liniments in the pharmacopeia, Lin. Ammoniae, Camphorae, Camphorae Co., Sinapis Co., Terebinthinae, Terebinthinae Aceticum, all of which may, if necessary, be diluted with soap liniment or olive oil.

The rubbing with the liniment should be carried out night and morning for about fifteen to twenty minutes, and the chest wrapped afterwards in cotton wool.

If moderate and sustained counter-irritation be desired, the various *iodine preparations* may be painted on once or twice a day (the tincture 1 in 40, the liniment 1 in 24, or the liquor 1 in 9). The application of the stronger preparations often causes pain, which may be relieved by washing the part with spirit or a solution of iodide of potassium to remove the excess of iodine and then applying a poultice or some simple ointment.

The prolonged use of iodine often makes the skin harsh and tender, so that it has to be discontinued for the time.

More powerful irritation even up to vesication or pustulation is hardly ever required. It may be obtained by means of *croton oil* applied pure, or better as the liniment (1 in 8). This may be made still more active by the addition of turpentine (7 parts to 1 of lin. crotonis), or less active by dilution with 1, 2, 3, or more parts of soap liniment.

The Liquor Epispasticus, in the same way, according to the strength employed and the duration of application, will produce any degree of counter-irritation from a simple erythema up to vesication.

Hot baths and diaphoretics.—Nothing is more beneficial in the commencement of an acute catarrh, especially in children, than a *hot bath*. It should be taken at a temperature of about 105, and after 10 or 15 minutes the patient should be quickly placed in a warm bed and the action of the skin encouraged. A *turkish* or *vapour bath* is equally useful.

To increase the stimulating effect salt or mustard may be added. A hot mustard bath is a most useful remedy for children in the early feverish stage of bronchitis or of broncho-pneumonia. Together with the bath a few doses of an active *diaphoretic mixture* containing, for instance, sweet spirits of nitre and acetate of ammonia, are of advantage, and the domestic treatment of catarrh by a hot mustard foot bath, warm drinks, and extra clothes on the bed can hardly be improved upon in principle.

Energetic diaphoresis, whether produced by drugs or baths, is, however, not actively employed in bronchitis, except in the early stage, where the skin is hot and dry and the temperature raised.

Derivatives acting on the intestines are rarely used except in the early stage of acute bronchitis, and then only in robust persons, on account of the depression produced. Still, in suitable cases brisk purgation does good. For adults the favourite remedy is the compound jalap powder. For children small doses of gray powder frequently repeated, gr. $\frac{1}{4}$ – $\frac{1}{2}$, two or three times daily, have been especially recommended for the bronchitis of teething.

Bleeding is not an *indicatio morbi* but an *indicatio symptomica et vitalis*, and the symptoms which call for it are rapidly-increasing dyspnoea and cyanosis with over-distention of the right side of the heart. The object is to relieve the heart, and pulmonary circulation, and bleeding will be useless under these circumstances unless free. In the adult it will be necessary to remove 15 to 20 ounces at least from the arm, or sometimes from the external jugular.

It has even been proposed to tap the right ventricle directly, and this operation is reported to have been several times performed with success. I hesitate to condemn an operation of which I have had no experience, but the risks are apparent, and I can hardly believe that it has any real advantage over the ordinary methods of venæsection.

In appropriate cases free bleeding may save life, and I believe I have seen this result more than once, but it is not a method of treatment to be played with.

"Free or not at all" should be the rule. If the patient be in such a state of health as not to bear the loss of many ounces of blood, it will be best to remove none.

Except under peculiar circumstances, bleeding is contra-indicated in children and in feeble adults. It does most good in robust full-blooded adults in acute attacks; but even in the late stages of chronic bronchitis, especially if associated with emphysema, it may be of service, when, as not rarely happens, the symptoms become suddenly aggravated, and though in such cases there is no hope of permanent cure, still life may be prolonged and made more comfortable.

The local abstraction of blood by *leeches* or *wet cups* is of little use in bronchitis; it is better suited to the local acute inflammations of the lung or pleura.

Dry cupping over the lower part of the back or between the scapulæ certainly often gives great relief, and when, as usual, there is orthopnoea, the cups can be applied without much disturbance. The therapeutic action of dry cupping must be reflex in nature and similar to the effect of counter irritation, but of the good effect it produces there is no doubt, whatever the explanation be.

Junod's boot, which is really a gigantic dry cup applied to the whole leg, was formerly used with the object of mechanically retaining the blood away from the diseased part. It is now almost forgotten. There is, or was, one at St. Bartholomew's Hospital, but it did not work, and I doubt if at most hospitals such an apparatus even exists.

Emetics.—The old teaching was this: "If the dyspnoea be due to overloading of the lungs with blood, bleed; if to choking of the tubes with secretion, give an emetic." It not infrequently happens that the choice of treatment seems to lie between bleeding and emetics.

Emetics give in many cases most marked relief. They are equally applicable to children as to adults, and are not contra-indicated even by considerable prostration, so that they may be used when bleeding is contra-indicated. The effect is mechanical for the most part and due to the expulsion of the secretion from the air-tubes by the forcible expiratory efforts made in the act of vomiting.

For an adult the best emetics are *ippecacuanha* and *tartar emetic* given in full doses, as, e.g., Pulv. Ipec. 20 grs., Antim. Tartr. gr. 1. In children tartar emetic is contra-indicated and ippecacuanha alone employed. The great advantage of both these central emetics is that they have an expectorant action which continues for some time after the emetic action has subsided.

Of the mechanical emetics the commonest and the best is *sulphate of zinc*. *Sulphate of copper* is more unpleasant to take and often disorders the bowels, while *mustard and water*, the simplest of all, is also the most unpalatable.

It is not easy to make a child sick by any drug administered by the mouth; even full doses of ippecacuanha often fail. The introduction, however, of *Apomorphine* has furnished a safe and also a reliable emetic suitable alike for children and adults. It is rapid in action and is followed by no marked depression. Some persons, however, seem very susceptible to the influence of the drug, for cases are recorded in which the administration of ordinary doses has been followed by alarming collapse, but this result seems rare. It may be administered by the mouth or *sub cutem*. For the adult gr. $\frac{1}{2}$ by the mouth, or gr. $\frac{1}{10}$ by the skin, is sufficient, and proportionate doses for children.

In a little child in whom suffocation seemed imminent, gr. $\frac{1}{2}$ sub cutem produced free vomiting in three minutes, with immediate relief and subsequent rapid recovery.

Laennec stated that he had cured many chronic cases by repeated emesis when other measures had failed.

Narcotics and Sedatives.—The only really trustworthy sedative in bronchitis is opium with its preparations. Although codeine, conium, hydrocyanic acid, hyoscyamus, lactucarium, and the bromides stand upon the list, they are of far inferior value.

Opium and morphia diminish the excitability of the respiratory centre, as hydrocyanic acid also does, though to a less degree, but they also diminish secretion and in both ways check cough, which is the chief indication for their use, and they form the active element in nearly all adult cough mixtures.

Belladonna and stramonium, while stimulating the respiratory centre, diminish the excitability of the nerve endings of the vagus in the lungs, but they are little used in bronchitis, except in the asthmatic forms.

Among the preparations of opium the favourite remedies for bronchitis are the Pil. Ipec. c. Sc. and Dover's powder; the latter especially has great influence upon recent catarrhs, and if administered early enough may cut them short—10 grains at bedtime is the common dose. A grain or two of camphor added to the Dover's powder increases its efficiency.

Opium is contra-indicated in children, for whom chloral or the bromides are more suitable. It is also contra-indicated in adults where the secretion is profuse and the strength low. The cough in these cases is conservative, and if it be checked the secretion accumulates in the tubes and increases the dyspnoea, which can only be relieved by prolonged and exhausting coughing. Many patients prefer a broken night to the distressing paroxysms of coughing which a few hours' unbroken sleep entails.

Opium is of most service where the cough is constant with little expectoration. For mild cases the lozenge of ipec. and morphia is a good remedy, or the pulv. ipec. co. combined with some camphor (1 gr.) or with tartar emetic (gr. $\frac{1}{4}$). Where the cough is very violent and prolonged it may yield to nothing short of 5 or 10 drops of laudanum on the tongue.

If opium does not suit a patient, *codeia* may be substituted for it in the form of the syrup. The *bromides* and *chloral* are also useful sedatives.

Hydrocyanic acid is an ingredient of many cough mixtures, but its action is feeble.

If *aqua laurocerasi* and *prunus virginiana* have any influence in bronchitis, it is probably due to the hydrocyanic acid they contain.

Astringents and Exsiccants.—Astringents or exsiccants are remedies employed for the purpose of diminishing the amount of expectoration. When administered in the form of inhalation they can have but little local effect, and when taken by the mouth it is probable that they often act more as general tonics than as local astringents.

Iron is the drug most commonly used, and, as Graves recommended, in the form of the Mistura Ferri co. The more astringent preparations are often not tolerated, as they increase the feeling of tightness in the chest.

The acetate of lead was advocated by Stokes in doses of 2 or 3 grains three times a day, and other authors speak well of it.

Alum also in doses of 5 to 40 grains in syrup or in powder with sugar is useful. It may be given in the form of alum whey, made by adding 120 grains to a pint of milk and straining off the curd.

Tannic and gallic acids (grs. 10 to 15) do good sometimes; or the preparations of *catechu*, which owe their efficacy to the tannin they contain.

Turpentine and its allies, *terebene*, *terpene*, etc., are useful in the profuse expectoration of chronic bronchitis. Other valued remedies are the *mineral acids*, nitric, sulphuric or hydrochloric, and some of the bitters such as *quinine*, *gentian*, and *chiretta*. Acids and bitters alike probably act chiefly as tonics.

Digitalis and *ergot* might appear to be rational remedies, but it is by no means clear that they have any direct effect upon the secretion.

The following remedies are also stated to be of service, though their action is not understood.

Oxide of zinc in 5 grain pills twice daily.

Myrrh, 5 to 10 grains in emulsion, but it is to be avoided where there is fever.

Agaricus albus, 2 or 3 grains every two or three hours.

Tonics.—*Tonics* are not tolerated in the early stages, but are generally required later in the disease. The most useful are *quinine*, *iron*, *acids*, *bitters*, *arsenic*, and *strychnine*.

Alteratives.—Alteratives are given with the object of modifying the processes of the disease.

Iodide of potassium, *calomel* and *arsenic*, in small and repeated doses, are most to be relied on. *Cod-liver oil* does much good in the later stages. The *alkaline carbonates* are of service where the secretion is viscid, and *colchicum* where there is an evidence of gout.

Sulphur does good in many chronic cases, and may be added to many of the cough mixtures. Plummer's pill (Pil. Hydrarg. Subchlor. Co.), containing as it does sulphide of antimony as well as calomel, is a useful combination, though rarely prescribed now.

Stimulants.—The condition of the heart and pulse is, in bronchitis as in other diseases, the indication for the use of the ordinary diffusible stimulants, viz., *alcohol* and *ether*.

The respiratory stimulants, *ammonia*, *senega*, *camphor*, etc., have been already referred to under the head of expectorants.

The cardiac stimulants of most use are *digitalis* and *caffeine*.

The special central respiratory-stimulants are *strychnine* and *belladonna*, especially the former.

The choice of drugs or combination of drugs will be determined by the special features of each case. In old people, however, and in severe acute attacks of little children, stimulants will probably be required from the first, in other cases only when the special symptoms calling for them arise.

Diet.—The diet should be according to the needs and capacity of the patient. In the early stage of an acute attack, when the tongue is coated and the appetite lost, nothing can be taken but liquid food, i.e., milk and beef tea, with warm demulcent drinks, but when the tongue cleans and the appetite returns, any simple food may be given, and probably some stimulant will be of advantage.

Purgatives.—There is a tendency to constipation in bronchitis, and at such times the appetite will be impaired.

The constipation is best combated by a mild Rhubarb pill, Plummer's pill, Confection of Sulphur and Senna, or some other mild purgative. Calomel, gr. 1 to 3 at night, followed by some Carlsbad or Epsom salts the next morning—Jalap in the form of Pulv. Jalapæ Co., or in combination with calomel.

Climate and change of air.—Owing to the cold, fogs, and constant changes of temperature, many patients are unable to spend the winter in this country, and choice must be made of a place abroad to winter in. The essentials of a good climate for bronchitis are that it should be warm, of uniform temperature, free from damp, changes, raw winds, and, as far as possible, dust.

The winter climates fall into two groups, the soft or sedative, and the stimulating or bracing. For the majority of cases the stimulating climates are in my experience the best, but some patients do well in a moist, relaxing climate.

Of the *warm, sedative climates* may be mentioned Torquay, Bournemouth, Penzance, and the Channel Islands in this country; the Pyrenees, Mont Doré, Pau, or Madeira abroad. Of the *stimulant and bracing climates*, the Riviera di Ponente is the most frequented. The warm, dry air of Egypt, as at Cairo or the higher parts of Algiers, have been strongly recommended. In America the parts most in repute are the southern half of California, the western part of Texas, Mobile in Alabama, Aiken in South Carolina, and most of the interior parts of Georgia and Florida.¹

In all these places alike, much depends upon the aspect of the residence, its altitude, and its proximity to the sea.

A *sea voyage* or residence near the sea level has been advocated because of the more uniform temperature, of the salt in the air, and of the greater atmospheric pressure; but, as a matter of fact, the sea does not suit many cases. The salt may make the air too irritating, and the moisture too oppressive. Many patients, as at Torquay or in the Riviera, do well on the hills who cannot tolerate the sea-level.

In summer the hills at home or the mountain resorts abroad are good, but the place must be selected with care, with a view to avoiding as far as possible rapid changes of temperature.

The various summer baths or cures of reputation owe their influence, I believe, more to the climate than to the cure, and in sending patients to such places regard must be paid to the climate and to the season of the year.

¹ Pepper, *Syst. of Medicine*.

The *water cures* supposed to be specially suitable for bronchitis are those in which the springs contain alkalis or sulphur.

1. *Alkaline springs*, such as Salzbrun, Vichy, Vals, Marienbrunnen.
2. *Alkali-muriatic springs*, such as Seltz and Ems.
3. *Salt springs*, such as Soden, Ischl, Reichenhall, where there are elaborate arrangements for baths and inhalation.
4. *Sulphur springs*, such as Aachen and Weilbach.

The other cures, viz., whey, milk, grape, vegetable, which have been advocated at one time and another, owe all their reputation to the climate and to the regimen insisted on in the establishments, where the patients are made to live according to rule, especially as regards diet and exercise, in a way which it appears impossible to get them to do at their own homes or in their own country.

Causal treatment.—Most of the *external* causes which influence the attack, such as cold, unhealthy occupations, etc., are put aside as soon as the general indications of treatment are carried out by placing the patient in bed in a warm room. It remains only to consider the causes *internal* to the patient which favour the disease, to remove them where possible, and failing that, to modify their effect by appropriate treatment. How to deal with these and other conditions in the patient not actually suffering at the time from bronchitis will be considered under the head of prophylaxis.

When bronchitis arises in the course of pleuritic effusion, pneumothorax, and ascites, little good will be done until the fluid has been removed by paracentesis.

In morbus cordis there should be added to the treatment for bronchitis some remedy appropriate to the condition of the heart, *e.g.*, digitalis, strophanthus, or caffeine.

In the acute congestion of pneumonia, or occasionally of morbus cordis, the question of bleeding, and in the case of high fever the question of cold bathing, may arise.

General constitutional defects, such as rickets, scrofula, obesity, gout, Bright's disease, chronic alcoholism, etc., must be dealt with by appropriate treatment, not so much at the time as after the attack.

When the chest is ill-developed, much may be done by properly regulated gymnastics to strengthen it.

Prophylaxis or Prevention.

1. All unsuitable occupations must be abandoned.
2. Chills must be avoided, by warm clothing, by wearing thick stockings and flannel next to the skin winter and summer, and by avoiding exposure to all sudden changes of temperature, as by passing from hot rooms to the outer air without sufficient covering, by being out of doors when the sun is setting and the mists are rising, or by exposure at any season of the year to raw, damp, and cold winds, and in summer to draughts, when the skin is acting freely.
3. In cold and foggy weather respirators should be worn to warm and filter the air.
4. The body must be put into the best condition of health and kept in it; and where constitutional weakness exists, whether congenital or acquired, it must be dealt with by appropriate treatment. So far as age is concerned, old people, young children, and also convalescents from illness must be treated like weakly adults, for their powers of resistance are low, and they should not be kept too long indoors. Coddling is bad, yet irrational hardening is often worse. Much

may be done by judicious management to brace up the body to stand changes of temperature, and nothing is much better for this purpose than a cold douche on rising.

5. Lastly, when patients can afford it they are best sent to winter in a good climate.

CLASSIFICATION AND VARIETIES. — The classification of bronchitis presents many difficulties, and is variously given by different authors, some taking, as Laennec did, the characters of the sputum for their basis, others the nature of the cause, and others arranging the cases in groups according to certain clinical types.

Each of these systems presents some advantages, but none are entirely satisfactory. The last method seems, on the whole, to be the most convenient.

Whatever classification be adopted, the features of the case will vary according as the disease is primary or secondary, acute or chronic, and according as it attacks the large or the small tubes.

Among the influences that affect the type, none is more important than age. Bronchitis has its own special features and dangers in the very young and in the aged, and in both it differs in important respects from bronchitis in older children and in adults in the middle period of life. The chief reason for this difference is to be found in the fact that in the adult it is chiefly the large tubes, and in the very young and the aged the small tubes especially that are liable to be affected, and thus bronchitis of the large tubes becomes the clinical type of the disease in the adult, and bronchitis of the small tubes that in the young and aged.

But besides these common forms of bronchitis, there are others which do not fit into any of these types, such as plastic bronchitis, putrid bronchitis, the bronchitis associated with morbus cordis, pituitary catarrh, and some others, which must be dealt with separately.

The different forms of bronchitis will be dealt with in the following order, a convenient arrangement, though it cannot be strictly called a classification.

Primary	{ ACUTE	Of large tubes. Tracheitis or Tracheobronchitis.
		Of small tubes. Capillary bronchitis.
		Suffocative bronchitis.
		Bronchitis in the aged. Peripneumonia notha.
	{ CHRONIC	Common mucopurulent catarrh.
		Bronchitis sicca.
		Bronchorrhœa serosa.
		Bronchitis putrida.
		Bronchitis plastica.
Secondary	{	MECHANICAL OBSTRUCTION. Morbus cordis, mediastinal tumours, emphysema, whooping cough, etc.
		COLLATERAL HYPERÆMIA. Phthisis, pneumonia, general tuberculosis, chronic affections of the lung, tumours of the lung, pleuritic effusion, ascites, pregnancy.
		FEVERS. The exanthemata, influenza.
		GENERAL DISEASES. Gout, rheumatism, and rheumatic fever, Bright's disease, alcohol, syphilis, ague, pregnancy.
		ASTHMA.
		IRRITATION. Gases, sprays, dusts. The passage of secretions or food from the pharynx or larynx, as in diphtheria, cancer, etc.

20. ACUTE BRONCHITIS OF THE LARGER TUBES—TRACHEO-BRONCHITIS ACUTA.

Simple Bronchitis of the Adult.—The following may be taken as a simple case of acute bronchitis of the large tubes in the adult:—

The patient, a man of 32 years of age, robust, and of previous good health, got wet through; the day was very cold, and he was unable to change his clothes for some hours. He felt thoroughly chilled, and on reaching home could not get warm until he had been in bed some time. He passed a restless, uncomfortable night, and woke in the morning with a headache, and with a sense of tightness and oppression in the chest. His whole body seemed sore and bruised as if he had been beaten.

Second day.—Though feeling unfit for work he went to his business, but returned early in the afternoon, finding himself good for nothing. The headache and oppression on the chest increased, and he had a slight dry cough. He felt constantly chilly, and was sensible of the slightest draught. That evening and night were spent like the last, and the next morning he sent for the doctor.

Third day.—The patient complained of great lassitude, and of aching all over the body, especially in the limbs; the head felt full and heavy, and the chest tight, so that it seemed difficult to get a full breath, the chest felt raw and sore beneath the sternum, and seemed to be scraped and rasped by the cough, which was frequent, violent, and noisy. The cheeks were slightly flushed, the complexion thick and muddy, the eyes suffused, the tongue coated with a creamy fur, the appetite lost and the bowels confined; there had been no motion since the commencement of the illness. The urine was scanty, high coloured, and deposited a sediment of urates; the skin was harsh, rough, and dry; the temperature was 99° F., the pulse 100, and the respirations 30. The movements of the chest were free, and there were no physical signs to be detected, nor was there any expectoration.

The patient was placed upon low diet, the milk and broth being given as hot as he could take them; a diaphoretic mixture containing some Vin. Antimoniale was prescribed, with a calomel and jalap purge; mustard poultices were ordered to the chest, back and front, *i.e.*, over the sternum and between the shoulders.

That evening the temperature reached 100. The general condition was the same, but the cough was more troublesome; after a fit of coughing a little viscid expectoration would be brought up, occasionally streaked with bright blood; rhonchus was now heard on both sides of the chest.

The night was weary, and rest was frequently broken by coughing.

Fourth day.—The skin was less dry, the temperature 99. The bowels had been freely relieved, the cough was looser and less painful, and the patient felt better. Rhonchus, loud, cooing, sonorous, was now audible over the whole chest, the expectoration was more abundant, mixed with a few large air-bubbles, and presented the ordinary characters of the sputa cruda.

Fifth day.—The expectoration was of the same character, but much more copious. As it became free, the tightness and oppression on the chest were greatly relieved, and the patient felt more comfortable. The temperature was still 99, the pulse and respirations remaining the same. Rhonchus continued, and crepitation of medium size was heard here and there.

Sixth day.—The expectoration began to be streaked with yellow, and by the ninth day the sputa cruda had been converted entirely into sputa cocta. During this time the patient had rapidly improved. The tongue had become nearly clean, the appetite had returned, though the physical signs had not much changed, except that large and medium crepitation was more abundant, still the cough was much less troublesome and had almost ceased to be paroxysmal, the expectoration being brought up without difficulty.

The subsequent history of the case consists in the gradual abatement and disappearance of the symptoms.

By the fourteenth day the physical signs had almost entirely vanished, and the patient seemed well, except for the cough, which was still easily brought on by talking or by change of temperature.

The patient was kept for a few days longer in the bedroom, and after a short holiday returned to business, being troubled only with a little cough on rising in the morning, which ceased for the day as soon as a few pellets of viscid expectoration had been coughed up. He remained in good health except that he found himself very susceptible to cold, the slightest chill seeming to fly to the chest and bring back the cough. He had one slight relapse at the end of April, after being exposed to the east wind, and did not lose the morning cough entirely until the end of the summer. He took care of himself during the winter following. It is now some years since the attack, and there has been no return.

Although it may be, as in the preceding case, that the first attack of bronchitis may develop without any previous evidence of chest weakness, still it more commonly happens that the patient has suffered for a longer or shorter time from cough on taking cold, and the acute attack often seems only an aggravation of his usual symptoms, and to be, as the patient often says it is, due to a neglected cold.

This recurrent cough, or winter-cough, as it is commonly called, from its being most frequent in winter, is in many cases due to a catarrh of the trachea, and might be described as tracheitis, but it is not found to be convenient or indeed possible to separate tracheitis from bronchitis, and as tracheitis runs so easily into bronchitis, and in severe cases is always associated with it, the term tracheo-bronchitis, or simply bronchitis, is used to cover both forms of affection.

Tracheo-bronchitis, though most common in adults, is by no means rare in children, but in them it differs in certain important respects.

1. The onset is more acute, and there may be shivering or even convulsions.
2. The general symptoms are more severe. The fever is higher, the temperature rising often to 103 or 104, though it does not remain continuously at such a height, but fluctuates considerably during the twenty-four hours. The pulse and respirations are more rapid and beyond what the fever would account for. The children are more seriously ill, and are often delirious at night.
3. The cough is more severe and more paroxysmal. There is no expectoration, for little children swallow any secretion they cough up. There is often some dyspnoea in very young children, even when the inflammation is restricted to the large tubes.
4. There is much greater risk of the small tubes becoming involved.

The **prognosis** of tracheo-bronchitis, both in the adult and in children, as regards life is good; indeed, in the absence of complications death hardly ever occurs. It is also good as regards complete recovery, and I think in this respect better in children than in adults, probably on account of the greater care taken of them after the attack.

The **duration** of this affection is about a fortnight, but it will be some time before the health is completely restored, for a certain delicacy of the bronchi is generally left behind, which shows itself in a tendency to cough on the slightest chill, or during cold weather. As long as the cough continues, the liability to relapse remains, and extra care is necessary. If the symptoms do not clear up completely, and expectoration as well as cough continue, the case will pass into one or other of the groups of chronic bronchitis.

Complications.—Except in those cases where the disease runs on into capillary bronchitis, there are but two complications of any importance which are likely to arise in tracheo-bronchitis, viz., laryngitis and pneumonia.

It is common enough, as would be expected, for the larynx to be affected together with the trachea, and hoarseness is a frequent concomitant of severe catarrh both in adults and children. It is, however, only in children that laryngitis assumes any great importance, but it may in them produce symptoms severe enough to lead to difficulty in diagnosis from the membranous form.

In the adult it is by no means rare for even a slight case of tracheo-bronchitis to run on into an attack of pneumonia. This is common in the more severe form of bronchitis in the aged and weakly, but it may occur in the course of a quite slight attack in a perfectly healthy person, as in the following case.

A young woman, aged twenty-five, of excellent health, caught cold, and for a fortnight was greatly troubled with cough. Expectoration was scanty, and the patient did not think it worth while to remain indoors, except for the last three days, at the end of which, without any great chill that she was aware of, she was suddenly seized with rigor, the temperature rose to 104, and apex pneumonia developed, ran its ordinary course, and ended in complete recovery.

Treatment.—The treatment of acute bronchitis is similar to that of severe catarrh.

The patient should be put to bed in a warm room and the air kept moist by means of a bronchitis kettle containing water with some aromatic such as Friar's balsam.

A hot bath, a vapour bath, or, at any rate, a hot foot bath may be given, after which, by means of hot drinks and a diaphoretic mixture, the skin should be induced to act freely. The effect of these remedies is often increased by a glass of hot grog or punch, but otherwise stimulants are not necessary in the early stage.

A purge of some simple kind will probably be required. The best is a grain or two of calomel followed by a dose of Mist. Sennæ Co. or of Carlsbad or Epsom salts.

It is at this stage that the free administration of antimony may cut the attack short, or that a full dose of Dover's powder (gr. 10 at night) may give so much relief. Neither of these remedies is, however, advisable for children.

Mustard poultices should be applied freely, or the chest rubbed with a stimulating liniment and wrapped in cotton wool.

The diet should be bland and unstimulating, and consist chiefly of milk and beef-tea, supplemented by hot demulcent drinks.

If the cough be troublesome, it may be controlled by small doses of opium or sedative inhalations.

For medicine it will be well to give some mixture like the following :—

Pot. Nitratis, gr. x.
 Vin. Ipecac., ℥ xx.
 Vin. Antimon., ℥ xx.
 Spir. Ætheris Nitros, ℥ss.
 Tt. Camph. Co., ℥ xx.
 Aquæ Camphoræ ad ̄ i.

4th horis.

To this draught may be added a few minims of Tt. opii if the cough is troublesome.

After a day or two, when the expectoration is fully established, the poultice may be replaced by a stimulating embrocation, *e.g.*, spirit of camphor or Lin. Terebinth Acet., the antimony stopped, and stimulating expectorants substituted for it.

Ammon. Carb., gr. v.
 Sod. Bicarb., gr. xv.
 Syr. Tolut, ℥ss.
 Vin. Ipecac., ℥ xv.
 Tt. Senegæ, ℥ss.
 Aq. Camph. ad ̄ i.

4th horis.

The general line of treatment laid down by Stokes cannot be much improved on for ordinary cases. First of all antimony; then ammonia with the stimulating expectorants, combined with opium and ipecacuanha; and lastly, during convalescence, iron, especially in the form of Griffith's mixture.

It is wise not to allow a patient to leave bed until the wheezing has entirely disappeared for some days.

Great judgment must be exercised during convalescence in the choice of days and time of day for going out of doors, but it often happens that, with care, fresh air expedites the cure.

In the child the same general treatment may be pursued, except that antimony will be avoided and opium not be given except in the form of Tt. Camphor. Co., and that it will be convenient soon, or from the first, to substitute for the poultices a stimulating embrocation.

21. CAPILLARY BRONCHITIS—ACUTE BRONCHITIS OF THE SMALLER TUBES—BRONCHIOLITIS—SUFFOCATIVE CATARRH.

Just as tracheo-bronchitis is the type of acute bronchitis in the adult, so is capillary bronchitis in the child. It is not only more common in children, but produces more severe symptoms, owing to the ease with which the air-tubes become obstructed and suffocation-symptoms develop.

This is to be attributed to two causes.

1. The small size and incomplete rigidity of the tubes as compared with those of the adult, so that a small amount of secretion may produce a large amount of obstruction.

2. The difficulty which children experience in expelling the secretion from the tubes. This is due—

- (i.) To their smaller chest capacity, *i.e.*, to the smaller volume of air which can be used to force the obstruction out.
- (ii.) To their feeble muscular power.
- (iii.) To the want of rigidity in their thorax; and
- (iv.) To their ignorance of the proper way of coughing; for effective coughing, *i.e.*, coughing which shall be effective in dislodging an obstruction from the air passages, is a habit unconsciously developed in the adult only by practice, and it is an art which little children have not had time to acquire.

Capillary Bronchitis in the Child.—Capillary bronchitis, beginning as such, is rare in both adult and child. It is in both nearly always secondary, and due to the spreading of catarrh from the larger to the smaller tubes.

As a rule the child has had a slight cold for a day or two with some running from the eyes and nose, and perhaps a little hoarseness, to which no importance has been attached, when often quite suddenly, and especially at night, severe symptoms set in.

The child becomes restless and feverish, and is disturbed by a frequent, dry, hacking cough, the respirations are rapid and panting, and the breath short.

The access of capillary bronchitis may thus be often apparently sudden, and it is sometimes ushered in by shivering or by a rigor, or even by a fit of convulsions which in infants so often replaces the rigor.

The fever is well marked, the face flushed, the eyes lustrous, the skin dry and hot, the tongue coated, and the urine highly coloured. The temperature may reach 103 or 104, but it fluctuates much on different days and even during the twenty-four hours.

The pulse is much above its normal rate (140–180), and often of high tension at first, but like the respiration, it stands in no definite relation to the height of the fever.

The tongue is moist and coated with a creamy fur; there is considerable thirst, but the appetite is lost, and there may be much sickness and diarrhœa.

The cheeks, hands, and body are dry and hot, but the head and chest often sweat freely.

The urine is febrile, *i.e.*, high-coloured, of high specific gravity, and deposits a copious sediment of urates.

The cough is dry, hacking, frequent, but not as a rule painful. It occasionally occurs in long and violent paroxysms. Like the voice, it is often hoarse from catarrhal laryngitis, which commonly accompanies bronchitis in children.

Dyspnœa is always present to some extent, and it is to this that much of the restlessness may be attributed. It usually comes on gradually as the bronchitis develops. There are not infrequently severe paroxysms, during which the child will jump up in bed, as in croup, in great excitement and throw itself about from side to side, until after a violent fit of coughing, ending often in vomiting or in the evacuation of urine or fæces, the dyspnœa is relieved, and the child sinks back exhausted and falls asleep. In such cases there is always cyanosis, but its degree varies with the amount of dyspnœa. Sometimes, as in whooping cough, such a paroxysm may end in spasm of the glottis, and the child die suddenly, suffocated, but such an event is rare.

The *physical signs* in a slight case may be limited to rhonchus and sibilus heard over the whole thorax, but in more severe cases changes may be observed in the shape and movements of the chest, as well as in the percussion note. The upper ribs and sternum down to the level of the nipple are markedly prominent and move but little, remaining on expiration in a state of expansion, while the lower parts are retracted somewhat and recede on inspiration. Some amount of inspiratory recession of the lower ribs and spaces is hardly ever absent in any case of capillary bronchitis in infants, and in very bad cases it may be almost as extreme as in laryngeal obstruction. Its extent may be taken as a measure of the gravity of the case, for it indicates obstruction to the tubes of the lower lobes, and ends often in collapse of those portions of the lungs.

The percussion note is hyper-resonant, especially over the upper part, but if there be collapse it may be dull in places, especially at the bases behind.

Wheezing is heard all over the chest, both rhonchus and sibilus, with more or less of crepitation, medium and fine, the wheezing being most marked above and in front, and the crepitation below and behind. Where, however, the tubes are completely plugged, as is most likely to occur at the bases, the breath sounds and crepitation may entirely disappear, and the diagnosis from pleuritic effusion become difficult.

There is no sputum, for children under five hardly ever expectorate.

The nervous system plays an important part in this disease in children, as is shown by the fits which often usher in the attack; by the delirium which is hardly ever absent to some extent; by the bending of the thumb into the palm and clenching of the fist (tetany), which is common in even early stages of severe cases; by the undue rapidity of the pulse and respiration, which are out of all proportion to the fever and local symptoms; and lastly, by the unconsciousness and fits with which bad cases often end.

The symptoms described as a rule develop gradually in the course of two or three days from the commencement of the attack. The chief danger to be dreaded is obstruction of the air tubes and consequent suffocation. Of this cyanosis is the best criterion.

Mere rapidity of breathing, contrary to what is the case in the adult, counts for little in young children, for the respirations may be rapid quite early in the disease when there is little dyspnoea, and become less rapid when the dyspnoea increases. Rapidity of breathing in the child may even be rather a good than a bad sign, as indicating activity of the nervous system, for with increasing cyanosis the sensibility of the nerve centres is diminished and the rate of respiration falls.

Alarming symptoms may develop at any time, and often with but little warning. In its most severe and acute form capillary bronchitis has been described by the name of *suffocative catarrh*, i.e., of bronchitis with early signs of suffocation, and some of these cases have terminated fatally within a few hours from the commencement of the illness.

The evidences of impending suffocation are these—the flush on the face fades and is succeeded by pallor; the complexion becomes of an ashy leaden hue, and the lips, nose, ears, and finger-nails blue; the eyes lose their lustre and grow heavy and dull; the cough gradually diminishes in frequency and in power, and is of no use for the purpose of expelling the secretion from the tubes. The restlessness passes off, and the child lies in a drowsy apathetic condition, taking little notice of what is going on around it; the respiration becomes less rapid, less deep and often irregular, a few hurried respirations being succeeded by a pause, and in some cases well-marked Cheyne-Stokes breathing may develop; the pulse becomes more rapid, fluttering, running, and difficult to count, irregular and ill-sustained. It gains in frequency and loses in power.

The dyspnoea grows greater, but the efforts to overcome it less. Though the internal temperature continue high, the skin may not feel hot, but be bathed in a cold clammy sweat. The child becomes more and more drowsy and difficult to rouse, until at last it passes into a comatose state and dies unconscious, the heart continuing to beat, for some minutes it may be, after respiration has stopped. Death is often preceded by convulsions.

The **prognosis** of capillary bronchitis is bad, and the younger the child the worse it is. Under five years of age the mortality is high (*cf.* fig. 30, p. 117). Still, hope should be maintained to the last, for the most desperate cases sometimes recover. Alarming symptoms rarely last more than a few hours, or perhaps a couple of days, but there is always the risk of their return.

Recovery is slow and frequently interrupted by relapses.

The **duration** in simple acute cases is about a fortnight (Fauvel, 5–8 days), but cases are recorded in which death has occurred within a few hours from the commencement of the illness. Such a rapid ending is, however, rare in the secondary form.

Diagnosis.—It is obvious from the description given that it is not easy, or indeed in some cases possible, to distinguish clinically between capillary bronchitis and broncho-pneumonia. Both affections result from the spreading of the inflammation along the air-tubes, and in many cases both conditions no doubt co-exist. Yet it is not necessary to go so far as some authors do and deny the existence of capillary bronchitis as a disease, apart from broncho-pneumonia.

The existence of capillary bronchitis as a special form of disease is, in my opinion, established conclusively by pathological as well as clinical evidence.

The difference of opinion is largely due to the fact that there are two clinical forms of broncho-pneumonia which are not clearly distinguished from one another, the one secondary to bronchitis, the other not.

In the first there is antecedent bronchitis, and the inflammation extends from the larger to the smaller bronchi, producing bronchiolitis or capillary bronchitis, and may in parts also extend to the vesicles of the lungs, in which case broncho-pneumonia becomes associated with the capillary bronchitis.

In the second there is no antecedent bronchitis, the onset is very acute, and the whole course of the case is that of an acute inflammation of the lung, *i.e.*, of pneumonia, or primary broncho-pneumonia.

It is these cases which have been described as acute suffocative catarrh, and which have proved fatal in a few hours. They are indeed but a special form of acute pneumonia or broncho-pneumonia in the child, and in fatal cases the usual acute changes are found in the vesicles and widely disseminated through the lungs.

It is to the first form alone that the use of the term capillary bronchitis should be restricted, for it is a form of bronchitis. The second belongs to the category of primary pneumonia or broncho-pneumonia, and not to that of bronchitis at all. It will be fully dealt with in the section on Broncho-pneumonia, *q.v.*

Complications of Capillary Bronchitis.—The chief complications are emphysema and collapse, broncho-pneumonia, and bronchiectasis.

Emphysema.—This is hardly ever absent even in moderately severe cases. It may be recognised sometimes by the hyper-resonant note on percussion, but it may exist without any characteristic change in the percussion note. It is almost always complementary, *i.e.*, associated with collapse of other parts; and as collapse is most marked in the lower lobes, emphysema is most marked in the upper, and to this is due, in young children, the prominence and impaired movement of the parts in front already described. In adults, the emphysema may be general, and give the usual signs of displacement of the liver and disappearance of the cardiac dulness.

Acute complementary emphysema of this kind is of no special significance, and quickly disappears as the bronchitis subsides.

Collapse also is rarely absent except in the mildest cases. If it occur in small patches its presence is unrecognisable, being masked by the emphysema which is its almost necessary accompaniment, but if it be extensive, as when the whole lower lobe is involved, percussion may yield a dull note. The best evidence of collapse is inspiratory recession, and the amount of recession may be regarded as the measure of the extent of collapse. Collapse, from the time which the affected portions of the lung take to recover themselves, is a common cause of delayed convalescence. It also not infrequently runs on into inflammation, *i.e.*, to broncho-pneumonia.

Broncho-Pneumonia, like collapse, may, if extensive, be recognised by the physical signs of consolidation, but more often the patches are small and masked by the emphysema, so that they yield no physical signs at all. The diagnosis must then be made by the persistence and character of the fever. Catarrhal pneumonia, though one of the commonest complications of bronchitis, does not stand in any direct relation with the extent and amount of the bronchitis, for it is often well marked where the bronchitis is slight.

Bronchiectasis is of the acute form (*cf.* p. 184). It gives no physical signs, and produces no symptoms. It cannot be recognised during life. If it be of common occurrence, as has been stated, it must completely disappear when the bronchitis gets well.

Where the cough is severe and paroxysmal, it may be associated in infants and very young children with spasm of the glottis, as in whooping cough, and thus produce a fatal termination; but this is a rare accident.

Capillary Bronchitis in the Adult.—Capillary bronchitis is not common in a robust adult, and when it occurs the fever is lower and the general symptoms less severe. It develops more gradually and runs its course more slowly. Complications are less frequent; emphysema is, however, the rule, but collapse is not common nor is it so extensive as in children; the mortality is much less, but the affection is more likely to end in incomplete recovery, and to lead to one of the forms of chronic bronchitis.

Treatment.—(i.) **In Children.**—The general treatment of capillary bronchitis is the same as that of tracheo-bronchitis, but as there is more fever and the skin is hot and dry, hot baths (especially the mustard bath) give great relief, and may be repeated if necessary every evening during the acute feverish stage.

Although, as a rule, depressants and opium should be avoided in children, still Stokes spoke highly of the use of small doses of Dover's powder and James's powder, a grain of each every six hours. Charles West recommended small doses of calomel and antimony every four hours for the first twenty-four to thirty-six hours, after which he advised the calomel to be omitted and a saline mixture containing a little opium and antimony to be given.

In most cases, however, a simple febrifuge mixture with some squill and ipecacuanha is sufficient.

If suffocation threaten, emetics are indicated, and they have been administered even two or three times a day.

Ipecacuanha, $\bar{3}$ ss– $\bar{3}$ i every twenty minutes until vomiting occurs, was the old-fashioned favourite remedy; but this is being gradually replaced by apomorphia, which is much more certain, while it is also, as far as my experience goes, a safe remedy even for quite little children (gr. $\frac{1}{30}$ – $\frac{1}{25}$ sub-cutem).

When emetics are necessary, stimulants also should be given. The best is a little brandy in milk.

If suffocation be imminent, it has been recommended to use cold effusions with a view to staving off carbonic acid narcosis, but this remedy is risky, and in my judgment has only a transient effect, if any.

If cyanosis be considerable, leeches have been used, but free bleeding is, as a rule, contra-indicated on account of the age, and a few leeches only are of little good.

Dry cups applied between the shoulders and to the loins are much more useful.

The continued inhalation of oxygen may be of great benefit; I have seen cases kept alive by this means for some days after death seemed imminent, and possibly if life be thus maintained by the use of oxygen, it might actually save cases which would otherwise have died. It is difficult, however, to establish this by clinical evidence.

Convalescence is often retarded by collapse of the bases of the lungs. This may be in some measure obviated by frequently changing the position, in which the child lies, from the back first to one side and then to the other.

One of the advantages of taking sick babies on to the lap or in the arms from time to time is the frequent change of position which this involves.

(ii.) **In the Adult.**—If the patient be robust and full blooded, capillary bronchitis may be treated more vigorously, either by the free administration of antimony, or of antimony and ipecacuanha, to nausea even if not to emesis, or if cyanosis develop, by free bleeding and depressant emetics.

In weak adults, emetics must be used with caution, stimulants must be given with them and the action of the heart maintained by digitalis, strophanthus or caffeine.

22. ACUTE BRONCHITIS OF THE AGED— ASTHENIC BRONCHITIS — PERIPNEU- MONIA NOTHA.

In the aged, acute bronchitis, whether of the large or small tubes, is liable to be associated with extreme asthenia; often in the course of what seems at first a simple cold. The patient loses all vigour, prefers to lie in bed, and though irritable and restless, is often inexplicably drowsy and lies the greater part of the day asleep.

The face is flushed, the complexion muddy, the skin dry and hot; the tongue has a dry central streak, the appetite is lost, there is much thirst, and the bowels may be loose; the heart-beats are rapid and feeble and the pulse tension low; the respirations are hurried and often panting, there is a frequent cough with but little expectoration, and nothing but slight rhonchus to be heard. In other cases the signs of bronchitis are more marked; there is more expectoration, it may even be profuse, and as a consequence the cough is more troublesome.

Though so drowsy during the day, the patients sleep badly at night, and there is usually slight wandering delirium.

There is as a rule not much fever, though the temperature may rise at night to 101 or 102, or even sometimes higher, so as to suggest pneumonia.

A dry central streak on the tongue has been regarded as of diagnostic importance in indicating pneumonia, but this is not correct, for the tongue has been dry where the absence of pneumonia has been demonstrated by *post-mortem* examination.

The physical signs may remain unchanged for some days, but all the while the asthenia continues to increase, the heart-beats grow more rapid and feeble, and the respirations less deep; the patient becomes more drowsy and can be roused only with difficulty to take food and answer questions, dropping off again at once into a heavy uncomfortable sleep, in which the breathing is panting and laboured, and there is low wandering delirium. After a time sibilus and crepitation show that the bronchitis has spread to the smaller tubes, and to the signs of asthenia are added those of dyspnoea and suffocation. The patient now sinks rapidly, the drowsiness deepens and ends in unconsciousness, and death comes usually without a struggle or any apparent suffering.

The tendency of this form of bronchitis is to involve the smaller tubes sooner or later. When this happens early the only result is to hasten the end, but in either case alike the patient dies more of exhaustion than of suffocation, rather as it seems from the heart than from the lungs.

Although, in the aged and feeble, bronchitis tends to assume the form just described, still in vigorous old persons it may present its usual features, while in younger persons, who are enfeebled by disease or other causes, it may be of the asthenic type.

These cases have been all grouped together since Sydenham's time under the title of *peripneumonia notha*, and though the term asthenic bronchitis fits in better with the present nomenclature, it is interesting to be reminded by the old name that the same clinical group of cases was recognised at a time when nothing was known of bronchitis as such.

The **prognosis** of asthenic bronchitis, whether in the aged or not, is extremely grave. If the patients do not die, convalescence is slow, and often interrupted by relapses, while recovery is rarely complete.

The **treatment** of bronchitis in the aged is like that in the child. All depressing remedies and emetics also are to be avoided, though good is said to result, even in cases of considerable prostration, from the use of antimony.

Stimulants will be required throughout, and it may be in considerable quantity. Strychnia and digitalis will be found of great use, the former to stimulate the respiratory centre and the latter the heart.

To avoid hypostatic congestion it is also important not to allow the patient to lie too long in one position, but to change the position frequently from one side to the other.

23. CHRONIC BRONCHITIS.

Chronic bronchitis is the term applied to cases of bronchitis of long duration, or of frequent recurrence; recovery being incomplete, as indicated on the one hand by the persistence of symptoms, and on the other by anatomical changes in the lungs.

From chronic bronchitis, therefore, must be distinguished those cases of acute bronchitis in which, though the duration has for some reason been prolonged, or in which relapses have occurred, recovery has in the end been complete; and again from cases in which, after a considerable interval of good health, bronchitis has recurred. In such cases the affection will be more exactly described by the terms *protracted*, *relapsing*, or *recurrent bronchitis* respectively.

The different kinds of chronic bronchitis are named according to the character and amount of the expectoration.

In the ordinary forms the sputum is mucopurulent in character and moderate in amount. Where the sputum is very profuse, the cases are described as *broncho-blennorrhœa* if it be mucopurulent, and *bronchorrhœa serosa* if it be serous; where it is very scanty or absent, as *dry catarrh* (*Catarrhe sec.* of Laennec).

ORDINARY CHRONIC BRONCHITIS—CHRONIC MUCOPURULENT CATARRH.—This may develop in one of two ways—either commencing with an acute attack of bronchitis, from which recovery is never complete, or, as is more commonly the case, with a cough which recurs at first every winter, then lasts longer and longer into the summer, until at last it is never absent.

Chronic Tracheitis or Tracheo-bronchitis (Winter Cough).—Chronic bronchitis in its slightest form is limited to the trachæa and main bronchi, and yields therefore no physical signs, except it may be a little rhonchus now and then. The cough is troublesome in the morning on rising, and continues until the secretion which has accumulated in the tubes during the night has been expectorated. After this it may give but little trouble during the day until bedtime, when it may disturb the rest during the early part of the night, but often the sleep is quite unbroken, and there is no cough till the next morning.

The sputum may be small in amount, but is not infrequently fairly abundant. It is expectorated in mouthfuls or lumps, yellow and nummular, often containing few if any air-bubbles, and it may sink in water. It may be so nummular as to suggest chronic phthisis, but is of course free from tubercle-bacilli.

Except for the annoyance of the cough, and for the breaking of the rest, the affection produces no symptoms, the general health and nutrition are preserved, and the patients are capable of an active and vigorous life.

This is the common form of winter cough. It disappears in warm weather and in a good climate. The affection may continue as such for many years, but it tends with each recurrence to get worse as time goes on, and to involve the smaller tubes.

In old people the secretion is usually more abundant, but the cough and expectoration may continue for many years without change or producing much discomfort, as in the following case :—

Mr A., aged 78, had had his cough for more than twenty years. It had always been worse in the morning, and for the rest of the day had been more an annoyance than a discomfort on account of the inconvenience of expectorating. The sputum was mucopurulent, and had always been of the same character, but had become a little more abundant of late years, otherwise the patient was perfectly well and had never been laid up with his chest, though he had frequently suffered from gout. The patient was in excellent health, and still in active work as the manager of a very large business calling for much bodily activity as well as mental power.

On two occasions of late years, when business required an absence of some months in California, the cough entirely disappeared, but this had never happened in this country even in summer, except when the weather was unusually settled, and that not of late years.

Drugs had been of little service.

The patient was robust and active up to his death, at the age of 84.

Chronic Bronchitis.—In the more severe forms, which are usually meant when the term chronic bronchitis is used, the smaller tubes are involved as well as the main bronchi. The physical signs are therefore more marked, the expectoration is more abundant though of the same character, and the effect of the disease upon the health more serious.

There is always a certain amount of permanent dyspnoea, increased by exertion, and due chiefly to the pathological changes in the lungs, which result from long-standing bronchitis, viz., emphysema and chronic interstitial fibrosis. The dyspnoea is subject to exacerbations if the secretion increase or accumulate in the tubes, and may be paroxysmal or even asthmatic in type (*Bronchitic asthma*).

The cough and expectoration vary much in different cases.

In chronic bronchitis of the slightest degree, it is the cough that is chiefly troublesome. The patients wheeze a good deal at times, but still are able to get about and continue capable of active work.

In the second degree, there are exacerbations of the disease in which the patients are obliged to lie up for a few days at a time, these attacks being easily brought on by slight exposure or even by over-fatigue.

In both these forms the general nutrition is usually unaffected.

In the more severe degree the patients are confined entirely to the house, or it may be to one room. The cough is constant and often most troublesome at night. There are frequent intercurrent febrile attacks, during which the temperature may rise to 101 or 102. The fever is explained in most cases by the recurrence of bronchitis, but may be due to broncho-pneumonia.

The health suffers greatly partly from the disturbed rest and constant fatigue of coughing, but still more from the confinement, *i.e.*, from the want of air and exercise. The patients may grow fat and flabby and be incapable of any effort; but they frequently lose flesh and may become extremely emaciated.

If, with the emaciation, there be intercurrent febrile attacks and copious puriform nummular expectoration, the diagnosis from phthisis becomes difficult. These are the cases which have been described as *Catarrhal Phthisis*, but they are

not phthisis, for there are no physical signs of phthisis during life, no bacilli in the sputum, nor are the signs of phthisis discovered after death. Pitiabie as the state of such patients seems to be, still after a time toleration is gradually established, and they may live for many years and remain capable of many enjoyments. I have had a case of this kind under my care for nearly twenty years. The patient, though confined almost entirely to one room and chiefly to bed, manages her house and family actively.

Complications and Course.—With the severer forms of chronic bronchitis, various complications are certain sooner or later to be associated.

General *emphysema* is hardly ever absent. It yields its ordinary physical signs, and only so far modifies the case as to increase the dyspnœa as well as the tendency to recurrence.

Interstitial Fibroid Changes in the lung are common. These again chiefly increase the dyspnœa, but they also not infrequently lead to chronic *bronchiectasis*, the diagnosis of which may sometimes be made by physical signs, but more often only by the character of the sputum.

To these may be added *acute inflammatory lesions*, such as pneumonia or broncho-pneumonia.

Chronic bronchitis may also end in *phthisis*, but not so frequently as has been thought.

In the later stages of chronic bronchitis, it is the failure of the heart that is the chief feature. The right side of the heart becomes dilated, and the results of venous obstruction show themselves—the feet first begin to swell, the œdema gradually increases until general dropsy develops; finally the lungs also become œdematous, and death occurs with the mixed symptoms of asthenia and suffocation.

Treatment.—It is not easy to lay down rules for the treatment of chronic bronchitis, for the cases differ widely.

As a rule *stimulating expectorants* are indicated, though they are often advantageously combined with small doses of antimony and ipecacuanha. If extra stimulation be required, carbonate of ammonia and senega may be added.

Iodide of potassium, arsenic, and sulphur are very efficacious in many cases, and they act probably as *alteratives*.

Among *tonics* the most useful are iron, strychnia, and cod-liver oil.

Inhalations are much employed, but the choice of them will be largely determined by the nature and amount of the sputum.

For the treatment of the cough, much will depend upon its relation to the expectoration; if this be abundant, drugs are required which will diminish the secretion; if it be scanty and the cough irritable, alkalis, expectorants, and opium are indicated.

For chronic winter cough, Friar's balsam, turpentine, and terebene are in common use, both by the mouth and by inhalation, and Dover's powder or the Pil. Ipec. c. Sc. may be required at night.

The chest should be rubbed with a *stimulating embrocation*, and even blisters are sometimes found of use.

Emetics are rarely necessary, but Laennec said that with emetics he frequently cured obstinate cases which had resisted other methods of treatment.

If asthma complicate the bronchitis, belladonna or stramonium, the iodides or iodide of ethyl may be prescribed, or other asthma-remedies employed.

For the chronic bronchitis of old people, ammoniacum, galbanum, and assafoetida are specially suitable.

Prevention as well as cure will be greatly assisted by a good climate, but a single winter abroad will rarely be sufficient for cure.

24. SPECIAL FORMS OF CHRONIC BRONCHITIS.

I. Broncho-blennorrhœa.—With the previous symptoms the expectoration may be extremely profuse, and amount to 1 or 2 pints or even more in the twenty-four hours, so as to suggest the existence of a large cavity or of an empyema bursting through the lung. To such cases the names of *broncho-blennorrhœa* or *blennorrhagia pulmonum* have been given.

There is always dyspnoea, which may be asthmatic in type. This affection is one of those included under the term *asthma humidum*. As a rule the patients lose flesh and strength early, but this is not due to the discharge alone, for in other cases with profuse expectoration the nutrition may be preserved for years.

Lebert describes a case in which the health was fair and the strength good in an old man of 80, who had been the victim of this affection for more than thirty years.

The cases are usually very chronic and develop slowly out of one of the forms of ordinary bronchitis.

In the course of typhoid fever I have on two or three occasions seen this condition develop in an *acute* form. The patients have brought up in the twenty-four hours a pint or more of purulent sputum. It has been a very anxious complication, but, in the cases I speak of, it did not prove fatal, and rapidly subsided when the fever passed away.

Treatment is directed towards reducing the expectoration. The resins, balsams, and turpentine have a great reputation in this respect, but the most valuable remedies are found among the astringent group—iron, tannic acid, acetate of lead, and also ergot and digitalis.

II. Catarrhe sec.—This term was invented by Laennec to describe a class of case in which, though there was more or less cough, there were few signs of bronchitis and little, if any, secretion. The description which he gives would include all the slighter forms of trachæo-bronchitis already mentioned. By some authorities the term is limited to a group of cases in which the lesion is in the small tubes and not in the large, and is by them defined to be *dry catarrh of the fine tubes*, the mucous membrane of which is swollen, but the secretion scanty. The change is, as a rule, widespread through the whole of both lungs.

The few cases I have seen of this affection have occurred in old persons. It is said to be common in children, in whom it is more serious in its results, but this has not been my experience.

The affection is difficult to diagnose from asthma, but it is more often, I believe, confused with plastic bronchitis, and this would explain Laennec's statement that it might be local and associated with complete absence of respiratory murmur over the affected part.

The sputum, if there be any, is scanty, viscid, and consists of small pellets, grayish in colour and translucent (*sputa margaritacea*, *pearly sputum*), in which Curschmann's spirals and the Charcot-Leyden crystals may be found.

The cough is very severe and exhausting, produces much muscular pain from straining, and may end with vomiting. Violent paroxysms may last an hour or more without intermission, and produce intense exhaustion, which in feeble aged persons may become alarming.

If there be any physical signs at all, they consist of rhonchus and sibilus only, but there is no crepitation.

Fever is absent unless there be some inflammatory complication.

There is always some permanent dyspnoea, due to the emphysema which is an almost invariable accompaniment of the disease, but during the paroxysm it is greatly aggravated. The paroxysms are often asthmatic in type.

The respirations vary with the dyspnoea, and in children may be very rapid, even as many as 80 in the minute.

It is invariably associated with emphysema, and may become complicated with collapse or with broncho-pneumonia. In most cases it passes gradually into one of the ordinary forms of chronic bronchitis.

The general condition varies, but as the affection may last for months, the health usually suffers considerably, especially in children, many of whom die of exhaustion.

The cause of the affection is obscure. It runs a chronic course, and obstinately resists all forms of treatment. In the young and aged it may prove fatal from the exhaustion which the violent paroxysms of coughing causes.

The chief symptom in this affection is cough, which will have to be controlled by opium, belladonna, and bromides, and by sedative and stimulating inhalations.

III. Bronchorrhœa Serosa.—Pituitary Catarrh—Phlegmor-rhagia Pulmonaris.—For the original description of this affection we are again indebted to Laennec.

The diagnosis is made from the character of the expectoration, which is muco-serous, like saliva in appearance, colourless, translucent, containing a few large cells, frothy on the surface, the fluid beneath being clear, like white of egg diluted with water.

The disease occurs both as a *primary* and a *secondary* affection, and in an *acute* and *chronic* form.

The *acute form* is very rare as a primary disease; but in a slight degree it is not very uncommon in the course of many pulmonary affections.

It presents itself under the guise of suffocative catarrh, and in children may be very rapidly fatal.

Laennec¹ gives one such case, in which after death the tubes were found filled with clear fluid, but otherwise appeared normal, and no other pulmonary lesion existed.

Laennec also quotes the case of a woman who was suddenly seized with intense dyspnoea, having been previously perfectly well, and after a few hours of great suffering expectorated an "enormous quantity" of serum, after which the symptoms disappeared and the patient recovered. Six months later she experienced a similar attack, which came on suddenly at night. This was followed by the expectoration of four pints of similar fluid, and the patient recovered.

Stokes² states that he has seen similar cases, but adds the remark, with which all authorities agree, that they are extremely rare.

The *chronic form* is not quite so rare.

¹ *Traite d'auscultation*, 1831, vol. i. 155.

² *Dis. of Chest*, *Syd. Soc.*, 1882, p. 60.

The following case I had under my care a short time ago :—The patient was a man of about 36 years of age, well nourished, and fairly strong, who had suffered from the affection for some months. He was admitted into the hospital because he looked so dusky and ill. The physical signs were those of bronchitis. The expectoration was copious and characteristic, and amounted to a pint daily.

After the first day or two the duskiess passed off and the patient felt well, but he continued to spit up about the same quantity of fluid daily all the time he was under observation.

One peculiarity in this case was that the temperature was raised, and though it fell somewhat, was still above normal when he left the hospital. He did not present himself again, and was lost sight of.

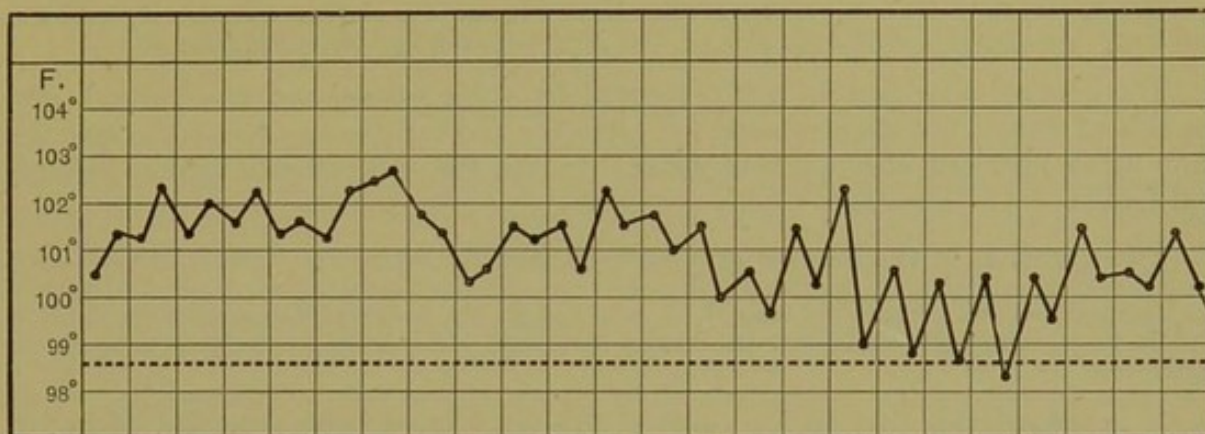


Fig. 35.

Temperature chart of case described above.

Gre¹ records the following case :—

A Japanese, 25 years old, came over as a ship's steward to England, and immediately upon his arrival began to suffer from shortness of breath and cough—a new thing for him. After these symptoms had lasted for fourteen weeks he was admitted to the hospital. There was much dyspnoea and a most abundant expectoration of puita. Tubercle-bacilli were not found. The physical signs were those of severe bronchitis. For days together he seemed about to die from suffocation; but under very careful treatment he improved until, without any obvious cause, the symptoms recurred in full severity. Another improvement was followed by another relapse, and more than four months had passed away since his admission ere he could be discharged fairly convalescent. This was on May 8th; but on June 16th he was readmitted with all his former symptoms; he went out on July 16th. On December 5th he was admitted once more, and the time of the year was probably the reason why we could not get rid of him till February 25th. He had now been a patient on and off for more than a year, and as there seemed no end to this sort of thing, the sister of the ward got up a small subscription and shipped him off to the antipodes.

The chronic form rarely commences suddenly, but is usually preceded by symptoms of ordinary catarrh. When the disease is once established it usually becomes intermittent, and, with irregular intervals between the attacks, continues for years.

The patients who suffer from the ordinary symptoms of chronic catarrh, and present the usual physical signs, bring up during the twenty-four hours, it may be, several pints of secretion having the characters already described. The expectoration may continue fairly uniform throughout the day, but more often there are bouts in which several ounces may be brought up at once, the interval being almost free.

During the attack the patients have dyspnoea, but in the intervals the breathing may not be very short.

¹ *Lancet*, March 29, 1899.

The secretion does not change in character in the course of each relapse, as in other forms of bronchitis, but remains throughout of the same character.

The quantity expectorated may be very great, as in another case of Laennec's.

The patient, a man over 60, brought up from 3 to 6 pints in the day in several attacks, separated by short intervals, after which he was not further troubled till the next day; he was active and went about much on foot.

Andral¹ records two cases in which *post-mortem* examinations were made with entirely negative results: the one, an old man, died after expectorating daily for five months about 2 pints; the other, a man of 45, had brought up 3 pints daily for three years.

Biermer also states that he has seen similar cases, but not of so extreme a kind. He considers that they may improve, if properly treated, but rarely, if ever, recover completely.

The general nutrition is well-preserved for a long time, and the patients are capable of work; but require care, for exposure and fatigue aggravate the attacks, as in other forms of chronic bronchitis. In the end the patients become anæmic and feeble.

The duration of the disease may be reckoned by years.

Laennec² records a case in a man of 70, who had for ten or twelve years expectorated about 4 pints daily, bringing it up in two paroxysms each day, being fairly comfortable between the paroxysms.

It is remarkable that so few cases of this kind have been recorded of recent years, and this may be taken as evidence of the rarity of the affection.

Though rare as a primary affection, serous bronchitis is not uncommon in the course of other diseases, and Laennec considered it to be often of the nature of a critical discharge. As such it has been described as occurring at the end of a bronchial catarrh, or of an acute pneumonia, and again in the course of disappearance of a pleuritic effusion or of an ascites. In these cases it does not, as a rule, last long, *i.e.*, more than a few hours, and is not in sufficient amount to produce severe symptoms of dyspnœa. It is now more commonly described as *serous* or *albuminous expectoration*, and will be more fully considered under this heading.

More commonly it is a sign of bad omen, and may of itself be the determining cause of death. It is thus met with as one of the last symptoms in acute pneumonia, in acute tuberculosis, and occasionally in phthisis, in the course of pleuritic effusions both serous and purulent, in asthma, and in morbis cordis. Many of these secondary cases have been spoken of as acute œdema of the lung, which in some respects is a good name for them.

One other group of cases belonging to this class remains to be referred to, *viz.*, that in which there is a copious discharge of serous fluid occurring in the course of thoracic aneurysm or mediastinal tumour.

The fluid expectorated may amount to many ounces in the twenty-four hours. It is like saliva in appearance, and is mixed with a few large air-bubbles, but it comes from the large air-tubes and not from the mouth. It is not rare in the course of thoracic aneurysm or new-growth to find the patient expectorating a considerable amount of thin, clear, serous, saliva-like discharge, often streaked with blood for some weeks before death. Death may result from actual bursting of the aneurysm through the trachea, but if death result from other causes, the trachea is found ulcerated and thinned so that it was on the point of giving way. The expectoration is attended with no dyspnœa, and with but few physical signs, and the cough with which it is associated is not specially frequent or difficult.

¹ *Clinique médicale.*

² *Loc. cit.*

Although in the cases which I have seen the trachea alone has been affected, it is possible that the same symptoms may accompany erosion of one of the main bronchi.

Sputum of this character occurring in such cases is, I believe, of some clinical importance in respect of prognosis, as indicating threatened perforation.

Treatment is very unsatisfactory in all these cases, but the use of the astringents and tonics is the most promising.

25. BRONCHITIS PUTRIDA, FETIDA, SEPTICA.

Putrid bronchitis is the name given to those cases of chronic bronchitis in which the secretion has a fetid or gangrenous odour. The diagnosis cannot be made by the smell alone, for fetid expectoration occurs with gangrene of the lung, with a fetid empyema discharging through the lung, or with cavities in the lung in which the secretion has undergone fetid decomposition. In these cases the fetor may be traced *post-mortem* to a local lesion, while the secretion in the rest of the bronchial tubes retains its ordinary characters. It is only where the bronchial secretion itself is fetid that the affection can be rightly named putrid bronchitis.

The diagnosis is determined (1) by the physical signs of bronchitis, and by certain characters of the sputum; and (2) the exclusion of those other affections upon which the fetor might depend.

The sputum is generally copious, and may amount to many ounces in the twenty-four hours. It is mucopurulent in character, and of an intensely fetid odour. The smell does not differ from that of gangrene, although it has been described as peculiar.

The sputum separates on standing into three layers.

The upper, the most bulky of the three, is frothy, opaque, and of a greenish colour. It is composed chiefly of mucus mixed with epithelium and pus cells.

The middle is an almost colourless, slightly cloudy, serous, saliva-like fluid, containing few formed elements.

The lowest forms a sediment of a yellowish white colour and granular appearance, in which are small lumps with an extremely fetid odour.

These lumps or pellets, originally described by Dittrich and Traube, and regarded by them as pathognomonic, are soft, friable masses, smooth on the surface, of a dirty, grayish-yellow colour, varying from the size of a millet seed to that of a bean, and of an extremely fetid odour.

Microscopical examination shows, chiefly of course in the sediment—

Epithelium derived from the air-passages, with much pus and a few red blood-cells.

There may, however, sometimes be a good deal of blood, as Traube¹ stated, and that without any gangrene.

Fat-drops and fat-crystals.

Needles of tyrosin and globules of leucin.

Pigment-granules, derived from the blood colouring matter; and sometimes a few crystals of hæmatoidin.

Charcot's crystals, transparent, diamond shaped, and of an undetermined nature.

Various organisms of putrefaction. *Leptothrix pulmonalis* (Leyden's and Jaffé's² specific organism). *Oidium albicans*, etc.

Besides all these, sequestra from the lung, if there be any destruction of the lung, infiltrated with prodigious numbers of bacteria.

¹ G. Sée, *Gaz. méd. de Paris*, 1882, 6. s. iv. 138.

² *Arch. f. Klin. Med.*, 1866, ii. 488.

Chemical examination shows the presence of

Many different forms of albumen.

All the common salts of the body.

Neutral fats and fatty acids, cholesterin, margarine, etc.

Butyric and valerianic acids, to which the smell has been attributed.

Leucin and tyrosin.

Glycerin in small quantity, and even a little glycogen.

A special ferment (Filehne) which has the property of decomposing the elastic fibres of the lung.

Lastly, some substance which gives a blue colour with iodine.

Most, if not all, of these products are the result of the putrescence of albuminous fluids, and could be obtained by allowing such fluids to decompose in contact with the air outside the body. Leyden and Jaffé actually demonstrated this to be the case with bronchitic sputum. Most of the organisms met with are those of putrefaction. Whether there be any truly specific organism peculiar to the disease, as Leyden and Jaffé suppose, is a question which remains unsettled.

Even at the temperature of the body such putrefactive changes require time, and will be most likely therefore to occur where there are cavities or pouches within the lung in which the secretion may lie stagnant for a time. In the great majority of cases of putrid bronchitis, such cavities are found to exist, and to have been in all probability the seat or origin of the affection. Out of the twelve cases which Traube records, in only two was chronic pulmonary disease with cavities absent.

It is possible, however, that under special conditions putrefactive changes might occur in the bronchitic secretion without any such cavities to start them, and there are a few cases recorded in which the absence of any lesion of this kind and of gangrene has been proved by *post-mortem* examination.

Chronic Bronchitis for 16 years—Sputum fetid twice—Putrid Bronchitis 17 days before death—Consecutive Pneumonia and Pleurisy.

P.M.—Putrid Bronchitis—No cavities—No gangrene—Recent Pneumonia and Pleurisy—Diphtheritic inflammation of smaller bronchi.

V. N.,¹ 28 years of age, was admitted into the hospital on June 20th. He had been troubled with cough and expectoration from the age of 12, and he stated that on two occasions the sputum had been fetid for a few days. On the 6th of June his present illness began with repeated shivering fits and a great increase in the expectoration, which became fetid. A week later a severe stitch in the side developed. On admission the patient suffered with considerable dyspnoea and could not lie down in bed on that account. The chest yielded evidence of general bronchitis only. The respirations were 28, the pulse 132, small and weak, the temperature 105, the sputum very fetid and containing plugs with the usual characters, amounting to about 10 ounces in the twenty-four hours.

June 22nd.—But little sleep, much weaker, some sweating during the night, great thirst, face pale, sputum of the same character (about 20 ounces), pulse 132, respirations 36, morning temperature 103.

Towards evening the patient became extremely weak and very pale, the skin dry, the tongue coated, the thirst great, pulse 124, respirations 36, temperature 105·8.

That night he slept badly and was delirious, and the next morning he died.

Post-mortem examination showed some recent pleurisy over the lower parts of both lungs; the lower lobes on both sides almost airless from a mixture of oedema and hepatisation, but not a trace anywhere of tubercle or gangrene; the mucous membrane of the larger bronchi injected, but otherwise of natural appearance; the smaller bronchi of the lower lobes in places in a condition of cylindrical or spindle-shaped dilatation, but their walls nowhere thickened; the mucous membrane everywhere pale but in many places roughened with membranous inflammation, most marked in the dilated parts. The finest bronchi yielded on pressure the dirty yellowish white, brittle, fetid masses observed during life in the sputum, and giving the same microscopical appearances.

¹Traube, *Ges. Beitr.*, ii. 573.

Of the twelve cases which Traube describes, that just quoted is the only one where the putrid bronchitis was not associated with gangrene or chronic cavities in the lung.

The next case has a special interest as being, I believe, the first of the kind recorded.

Chronic Bronchitis 12 years—Sputum fetid for years—Sudden Death.

P.M.—No lesion other than fetid bronchitis. (Andral.)

A cook, 60 years of age, was admitted into La Charité in March 1822 in a state of exhaustion and great emaciation. For the last ten or twelve years he had been troubled with cough and shortness of breath in the winter. During the preceding summer he had spat a little blood. From the time of admission he coughed much, and expectorated a great quantity of greenish fetid sputum, which might have been thought to come from the pleura or from a large tubercular cavity. According to the patient's own statement he had expectorated similar sputum for several years. On percussion the chest was resonant everywhere; on auscultation the breathing sounds were coarse, with sibilus at times. The patient was without fever and had never sweated.

For the next ten or twelve days the patient remained in the same condition. The sputum was always fetid, the appetite good, but the weakness extreme.

On the 28th of March he grew suddenly much worse. The face became livid, the eyes dull, and the dyspnoea extreme; the pulse frequent, irregular, and hardly to be felt.

For the next two days suffocation was imminent, and on the 31st he died.

On *post-mortem* examination the lungs were found very cedematous and in some places non-crepitant, the large bronchi filled with secretion having the same characters as the sputum and very fetid. The mucous membrane of the smaller bronchi was reddened, but there was no other lesion of the bronchi or lungs. The spleen was very large, soft, and dark.

In the course of diphtheria the sputum may become fetid.

In a fatal case recently seen in a child the membrane did not extend far along the tubes, but the medium-sized tubes, though containing no membrane, were filled with a quantity of extremely fetid secretion; there was no other lesion in the lungs except the bronchitis.

When putrid bronchitis has once developed, whether in association with cavities or not, it is able of itself to set up grave secondary lesions in both the bronchi and the lung. As the direct result of the irritation of the fetid secretion the walls of the bronchial tubes may be attacked with inflammation, sometimes of a character so intense as to be rightly called gangrenous. The adjacent vesicles may become involved and patches of pneumonia develop, which often end in suppuration or in gangrene. Traube¹ records a case in which this occurred and death resulted from pneumothorax due to the bursting of the gangrenous lung into the pleura. Leyden and Jaffé actually produced all these lesions experimentally by injecting into the air passages of rabbits the sputum from cases of putrid bronchitis, but similar lesions are constantly met with wherever putrid discharges find access to the lungs from any source.

The **local signs** of the disease are those of chronic bronchitis, with the addition of those peculiarities of the sputum already described.

The **general symptoms** are septic in character, and probably depend upon the absorption into the blood of the poisonous products of putrefaction.

The common history of putrid bronchitis is this—the patient, usually in middle life and the victim of chronic bronchitis, is seized more or less suddenly with symptoms of grave illness, ushered in it may be with one or more rigors and with the fetid change in the sputum. Fever sets in, and with it develop signs of profound prostration. After but a short time, a few days perhaps, the

¹ Marfan, Charcot, *Traité de méd.*

patient passes into the typhoid state and dies collapsed, death being often preceded by suppression of the expectoration and consequent suffocation.

In severe cases the general symptoms are intense, but they do not differ from those of septicæmia. They are almost invariably fatal and constitute the class of case which has been most frequently recorded, and to which G. Sée proposed to give the name of septic bronchitis.

Case of Septic Bronchitis after parturition.

Marie S., aged 33, had suffered from bronchitis since the birth of her third child, six years ago. She was confined of her fourth child on September 26th. The accouchement was normal, but the cough, which had been more troublesome during the last four months of pregnancy, grew worse after delivery. On December 15th she felt pain in the left side, and the expectoration became profuse and yellow. A week later hæmoptysis occurred; it lasted for several days, but was not profuse. Since the beginning of January there had been fever, highest at night, and slight œdema of the hands and feet. On admission on January 12th the patient was extremely ill and very prostrate, expectorating a large amount of fetid sputum, which presented all the characters typical of fetid bronchitis. On the next day she died of exhaustion.

The *post-mortem* examination showed emphysema of the upper parts of the lungs with œdema and congestion of the lower, no pleurisy and no consolidation. The trachea and great bronchi were violet-red, covered with thick fetid pus and with patches of false membrane here and there; the small bronchi were obstructed with thick pus of the same characters. There was some slight general dilatation of the smaller bronchi, but no pouches.

The lymph glands were enlarged and contained fetid fluid, but were not suppurating. The kidneys and liver were large and congested, and the spleen three times its normal size, friable and like the spleen of typhoid.

Such attacks may even occur at irregular intervals, and in many of the fatal cases a history is obtained of previous attacks, similar in nature but without other grave symptoms.

If the poisonous substances happened to be not absorbed at all, or not in sufficient quantity, septic symptoms might be entirely, or to a great extent, absent. Accordingly there are mild cases recorded in which the fetid expectoration has continued for weeks or months without the general health suffering in any special degree. These mild cases are probably more frequent than would be gathered from the published records. Indeed, Trousseau attached a relatively benign character to the disease in general, while Traube, who chiefly described the severe cases, regarded it as an affection of the utmost gravity and danger. It is possible, however, as Lebert suggests, that some of these mild cases may depend upon a different cause, and be the result of a peculiar form of mucous decomposition, comparable, I suppose, with those forms of ozæna which are not connected with any definite local lesion.

There are, at any rate, the two classes of case, and the prognosis depends not so much upon the local affection as upon the general symptoms which accompany it.

The **diagnosis** rests, as already stated, upon the evidence of general bronchitis with fetid sputum, and next upon the proof that the other causes of fetid expectoration are absent. It is usually not difficult to put aside the causes of fetid discharge which exist in the nose, mouth, or pharynx, but it is extremely difficult to exclude those which have their seat in the lung. The physical signs and the history will probably be sufficient to dispose of fetid empyema, but the diagnosis of putrid bronchitis from gangrene or fetid cavity may be absolutely impossible without a *post-mortem* examination, and may not be quite certain even then. The presence of elastic fibres in the sputum is not of much value, for destruction of the lung may follow, as well as precede, the putrid bronchitis.

Treatment will be directed in the first place towards supporting the patient's strength, and in the next towards diminishing the fetor.

Abundant food, with stimulants, and, as medicine, quinine and strychnine, will fulfil the first indication. The latter will be best met by antiseptic inhalation or by the internal administration of creasote, or turpentine, but especially of musk. A grain of musk as a pill or a few minims of the tincture two or three times a day is on the whole the most efficacious remedy.

History.—The earliest cases were recorded by Andral¹ and Laennec.

The association of putrid bronchitis with gangrene of the lung and with chronic cavities was described by Dittrich and Traube,² who also gave the first description of the plugs, to which they attributed a pathognomonic importance.

Lasèque³ showed that gangrene was not a necessary concomitant.

The most complete observations on the subject are those of Traube,⁴ upon whose monograph most of the subsequent accounts of the disease are based.

Trousseau⁵ drew attention to the mild forms of the disease.

Leyden and Jaffé⁶ demonstrated the presence of putrefactive organisms and described one form to which they attributed specific properties, viz., the *Leptothrix pulmonalis*. Lastly, G. Sée⁷ laid stress on the septic character of the symptoms in the grave cases. I do not know that bacteriology has so far added much to our knowledge of the affection.

26. PLASTIC BRONCHITIS — FIBRINOUS OR MEMBRANOUS BRONCHITIS—BRONCHIAL CROUP—BRONCHITIS CROUPOSA—BRONCHIAL POLYPUS.

The essential characteristic of this remarkable disease consists in the expectoration of fibrinous casts of the bronchial tubes after a paroxysm of more or less severe dyspnoea. Such casts are common in diphtheria, and are not altogether rare in acute pneumonia, but as in these cases the bronchial cases are secondary to the inflammation of the larynx on the one hand and of the lung on the other, the term plastic bronchitis is not applied to them, but is reserved for another and entirely different group in which the bronchi themselves are the primary seat of the affection.

The disease is a rare one, so much so that Lebert,⁸ writing in 1869, could only find records of 44 unquestionable cases. Since his paper other cases have been recorded, which, together with a few more of earlier date omitted from Lebert's series, brought the total in the year 1889 up to 98. It is upon an analysis of these 98 cases that the following account of the disease is based, but reference by the way will be made to a few other cases which have come under my own observation.⁹

The cases are described according to the suddenness of access and severity of the symptoms as *acute* or *chronic*. It is often difficult to draw so sharp a distinction between them, but whether acute or chronic it is the tendency to relapse and to recur that forms a prominent feature in the disease.

The patient suffering from this affection, often without other premonitory symptoms than those of ordinary bronchial catarrh, is usually suddenly seized

¹ *Clin. Méd.*, iv. 144.

² *Über Lungenbrand in folge von Bronchialerweiterung*, Erlangen, 1850.

³ *Arch. de Méd.*, 1857, vol. ii.

⁴ *Clin. Méd.*

⁵ *Gaz. Méd. de Paris*, 1882, 6. s. iv. 138.

⁶ *Gaz. Méd. de Paris*, 1882, 6. s. iv. 138.

⁷ *Ges. Abh.*, vol. ii. 556.

⁸ *Arch. f. klin. Med.*, 1866, ii. 488.

⁹ *D. Arch. f. klin. Med.*, 1869.

⁹ The complete literature up to 1889 will be found in my paper in the *Practitioner*, vol. 43, 1889.

with dyspnœa, and after a violent paroxysm of coughing brings up one or more casts, after which the symptoms subside, if they do not entirely disappear, until the next attack occurs.

The **casts** are expectorated as fleshy pellets or masses which, on being floated out, prove to consist of a stem with many minutely ramified branches. The larger casts are generally tough and yellowish-white in colour, but the smaller may be soft and gelatinous. Though usually solid they may contain a lumen into which a small probe can be inserted; even when there is a lumen it is usually filled with mucus and is not permeable to the air. The stem and branches correspond with the divisions of a bronchial tube, and it may be even possible to diagnose, from the method of branching, the place from which they come. Thus casts from the lower lobe are longer than those from the upper; while if the cast come from the main bronchus it is more stumpy, and more rapidly branching when formed on the right side than on the left. Upon the branches are sometimes found small pouches or protrusions, which have been supposed to have their origin in bronchiectatic cavities, but they are more probably due to irregular distension of the cast from within by mucus and air.

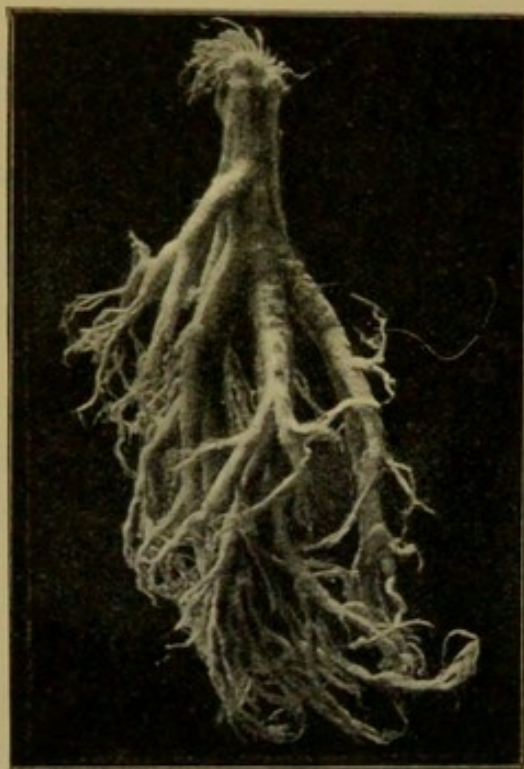


Fig. 36.

Photograph of a cast from a case of plastic bronchitis (St. B. H. Museum, 1685*b*).

The branching may be so fine as to correspond with the terminal bronchi, or even with the vesicles themselves.

On section the larger casts are often found to be formed of two or three concentric layers.

Microscopical examination shows them to be composed of a structureless substance through which are scattered numerous white cells and occasionally a few ciliated cells. Fat-drops and the Charcot-Leyden crystals have been described, but are not common. Diligent search has been made for specific germs, but so far nothing but putrefactive bacteria have been found.

Curschmann described peculiar spiral threads upon which he laid stress, but they have not been found by other observers. They are certainly not constant, nor are they pathognomonic. Chemically the casts consist of fibrin and yield the usual reactions.

The casts are not, as Laennec and others have asserted, simply clots of coagulated blood. Streaks of blood are common enough upon the surface of the cast, but red blood cells are not found in their substance, as would be the case if they were blood coagula. True blood casts of the bronchi do occur, but they differ in colour and are not minutely branched. One very interesting instance of true bronchial blood cast came under my own observation, and is preserved in the museum of the College of Surgeons.¹

In structure and composition the clots are identical with the membrane of ordinary croupous inflammation, and are no doubt produced in the same way, viz., by exudation from the blood, and not, as Kretsky supposed, by a peculiar degeneration of the cells of the mucous membrane.²

¹ Catalogue Prep., 3451A.

² *Wien. Med. Woch.*, 1893, xxiii. 315.

In size the casts vary greatly; commonly they do not measure more than 2 to 2½ inches in length, the size of the stem being that of a goose quill. They may be much larger, even reaching the length of 6 or 8 inches and measuring ½ inch in the stalk, but they may also be very small, and are then easily overlooked.

Morbid Anatomy.—The disease being rare and by no means very fatal, the number of *post-mortems* has been small, so that statements as to the morbid anatomy of the affection must be made with some reserve. The process appears to be essentially circumscribed, and though the bronchi in any part may be involved, those of the lower lobes are especially liable to attack. The affection commences as a rule in the medium-sized bronchi, whence it spreads to the finer, and subsequently it may be to the larger, tubes. It sometimes happens that no casts are found *post-mortem*, even when they have been freely expectorated during life, and occasionally the converse occurs, and casts are found *post-mortem* when none had been expectorated and when the diagnosis therefore could not be made.

The casts are usually quite loose in the bronchi, and are easily removed; at other times they are more difficult to detach, but it is only very rarely that they are adherent.

Except for the evidence of slight bronchitis, the bronchi are intact, and even the ciliated cells may be retained in the affected tubes. It is quite exceptional to find any local lesion in them. Biermer¹ records one case in which an ulcer was found, but it was in all probability syphilitic; in another case² a caseous gland had broken into the bronchus, and it was in connection with this lesion that the cast had formed.

Associated Lesions.—Although in many cases the changes described constitute the whole pathology, still plastic bronchitis is very frequently associated with other affections.

Thus the disease not infrequently occurs in a patient who has for some time suffered from *chronic general bronchitis*, but even then the plastic bronchitis is localised, *i.e.*, the casts are formed only in certain parts of the lung.

Phthisis is not uncommon.

Thus in 7 out of the 51 recent cases *phthisis* existed, or was reasonably suspected. Of these 4 died, in 3 of these recent tuberculosis was found, and in the fourth chronic *phthisis*.

Considering, however, how frequent a disease *phthisis* is, and how rarely plastic bronchitis is found *post-mortem*, it is clear that the association between these two diseases, if it be not accidental, must be remote.

Out of Lebert's 32 chronic cases 10 died, and in 7 of them *phthisis* was found; while of the other 3, 1 had fetid expectoration with anthracosis, 1 had stricture of the trachea, and the last had emphysema and a fatty heart.

Of the other acute affections of the lungs, plastic bronchitis is commonest after *acute pneumonia*. The cast may appear weeks or even months after the pneumonia is past, as in a case of Moeller's,³ where the pneumonia preceded the expectoration of the cast by ten weeks, and in Adersen's,⁴ where the interval was four months. These casts must be distinguished from the group already referred to, in which casts are expectorated during the attack of pneumonia. When the interval is short it may well be that the cast was formed at the time of the pneumonia and only got rid of later, but it seems unlikely that this is the correct explanation when the interval is so long as in the cases mentioned. In consider-

¹ Biermer, *Virch. Handb. der spec. Path. u. Ther.*, V. Abt. 1, Hft. 4.

² Weigert, quoted by Sax, *loc. cit.*

³ Schmidt's *Jahrb.*, vol. 204, p. 162.

⁴ *Ibid.*, vol. 201, p. 154.

ing the relation of plastic bronchitis to pneumonia, it should be borne in mind that pneumonia may be the result as well as the cause of plastic bronchitis and may then lead to death.

Plastic bronchitis may also be associated with *acute destructive disease* of the lungs, probably of a septic nature.¹

It is stated to occur also with *pleurisy*, both acute and chronic, but this combination is so rare as to be in my opinion only accidental.

The association with *morbus cordis* is more remarkable.

In the 51 recent cases it was met with no less than 5 times, and always with mitral disease. Alarming as such a complication must be, strange to say only two of the patients died, and neither of them from the plastic bronchitis; death being due in one case to tuberculosis, the heart lesion not having been suspected during life, and in the other to a general septic condition, acute pericarditis and endocarditis, together with acute pneumonia, being found after death.

Starck² records a case in which extensive *infarction of the lung* occurred and a cast was expectorated five weeks later, the patient making a good recovery.

Plastic bronchitis sometimes follows *acute specific fevers*. It occurred in one case³ five weeks after *typhoid fever*, and in another ten weeks after an attack of *scarlet fever* and *pneumonia*. Cases have also been described in connection with *influenza* and *measles*.

It is noteworthy that in no less than 4 out of the 51 recent cases⁴ plastic bronchitis was associated with some well-marked *affection of the skin*:

With pemphigus in two⁵; with impetigo in a third⁶; and with impetigo and herpes zoster in the fourth.⁷ Of the two cases of pemphigus one died, and in both the eruption affected the mouth and pharynx, and was detected in the larynx and trachea. Mader even goes so far as to suggest that all cases of plastic bronchitis are of the nature of pemphigus, but there is little if any evidence to warrant such a conclusion.

Lastly, there appears to be sometimes a relation between the plastic bronchitis and the *catamenia*.

In a case recorded by Brik⁸ such a relation, it was thought, could be clearly traced in the first three attacks, but the fourth occurred at the age of 60, after the catamenia had been long absent. Oppolzer's⁹ case is more marked. The first attack lasted two years with monthly intervals, occurring regularly at the times of the catamenia: pregnancy followed and the attacks ceased. In the two subsequent attacks the same relation was observed, but the attack was of shorter duration, lasting only five weeks and three months respectively.

Results.—Plastic bronchitis, even when of long standing, often has but little effect upon the lungs. It may lead to emphysema, both transient and permanent. Collapse in the obstructed portion is not uncommon, and this may pass on into pneumonia. It is also stated that the plugged tube may even be obliterated and the corresponding portion of collapsed lung become fibrotic.¹⁰

Ætiology.—Of the actual determining or producing cause we know nothing. The affection is connected with no diathesis, and is peculiar to no locality, race, or occupation. It is comparatively common in this country, in Germany, in Switzerland, and in some parts of America. On the other hand it is rare in Italy and warm countries, in these respects resembling bronchitis in distribution, and being, like it, most common in the cold damp seasons of the year.

¹ Janeway, *Med. Rec. N.Y.*, xxii. 102.

² *Berl. klin. Woch.*, 1886, xxiii. 221.

³ Möller, *loc. cit.*

⁴ Monat, *Arch. Gen. de Méd.*, 2nd Ser., xiv. 238.

⁵ Mader, *Wien. med. Woch.*, 1882, xxxii. 301; Escherich, *D. med. Woch.*, 1883, ix. 108.

⁶ Waldenburg, *Berl. klin. Woch.*, 1869, p. 208.

⁷ Streets, *Amer. Jour. Med. Soc.*, N.S., lxxix. 148.

⁸ *Wien. Med. Pr.*, 1882, 828.

⁹ *Vorles. ü. spec. Path. u. Ther.*, i. 3 Lf. 446. Schnitzler, *Wien. Med. Halle*, 1864, v. 450.

¹⁰ Biermer, *loc. cit.*

As already stated, it is liable to be associated with certain affections, but its occurrence in them seems to depend upon some accidental condition, and what this is has not been ascertained. The search after specific organisms has hitherto proved unsuccessful.

Some of the acute cases not associated with croup, or diphtheria, or with pneumonia, it might be urged, are really diphtheritic in nature, a theory which some French authors would express by the use of the term "croup bronchique ascendante," in contradistinction to the common form which they would describe in contrast by the term "croup laryngienne descendante." Plausible as such a theory may seem when applied to the acute cases, it is quite inappropriate to the chronic cases, which have nothing whatever in common clinically with diphtheria, except the formation of a membrane.

Sex.—The affection is, at least, twice as common in men as in women: thus of the 98 cases, 65 occurred in men and 33 in women.

Age.—The following table shows the age incidence of the disease, taking the acute and the chronic cases together.

TABLE showing Age and Sex of forty-nine cases since Lebert's paper.¹

	-5	-10	-15	-20	-25	-30	-35	-40	-45	-50	-60	-70	Over.	Unspeci- fied.	Totals.
Male, . . .	1	2	3	5	3	1	..	4	1	2	..	1	..	12	35
Female, . . .	2	1	2	1	1	2	2	..	1	2	14
Total, . . .	3	3	5	5	3	2	1	6	3	2	1	1	..	14	49

TABLE from Lebert's paper reckoning Acute and Chronic cases together.

Both Sexes,	2	8	3	7	4	1	4	2	4	2	..	1	6	44
Combining the totals of the two tables, }	3	5	13	8	10	6	2	10	5	6	3	1	1	20	93

This table shows that the affection is fairly evenly distributed throughout the whole of life, though on the whole it is rarest in old age. Lebert states that two-thirds of all the cases occur under 25, but the larger numbers of the above table do not confirm this statement.

In considering the age incidence it is important to take the age of the patient at the time of the first attack and not at the time of observation.

In Brik's² case the first attack developed at the age of 36, and the last at 60, and again in Kretschky's³ case the disease commenced at 41 and lasted 25 years, so that the patient was 66 when under observation.

The youngest case recorded, so far as I am aware, occurred at the age of 4 years,⁴ and the oldest in a woman of 72.⁵

The disease may attack more than one member of the same family. Thus two of Watson's cases were brothers, and Fuller⁶ describes the affection in two sisters.

SYMPTOMS.—The characteristic symptoms are paroxysms of cough and dyspnoea, which are relieved by the expectoration of the cast.

¹ West, *loc. cit.*

² *Loc. cit.*

³ *Loc. cit.*

⁴ *Loc. cit.*

⁵ Lebert, *loc. cit.*

⁶ *Trans. Path. Soc. Lond.*, v. 41.

The **dyspnœa** depends upon the obstruction produced by the casts, and varies with their number, their size, and the rapidity of their formation. It often develops gradually, keeping pace, as it were, with the growth of the cast, but it may arise quite suddenly. As a rule it is relieved at once by the expectoration of the cast, often completely, so that the breathing is normal until the next attack occurs; sometimes the breath remains short between the paroxysms, and this may be evidence either of the existence of other casts or of some organic disease of the lungs.

Spaeth¹ found the vital capacity reduced during the attack from 1675 c.c. to 1317 c.c.

However threatening and severe the dyspnœa may be, it is rarely fatal of itself.

Fagge² recorded a case in which a large cast, detached from the bronchus of one side, obstructed the bronchus of the opposite side, and led to immediate suffocation. This was and still is a unique case. When the cast is detached, it is normally expectorated at once without further difficulty.

In spite of the dyspnœa there is rarely any cyanosis, a fact which is explained by the circumscribed nature of the lesion.

The **cough** is paroxysmal and often of sufficient violence to produce vomiting, epistaxis, or hæmoptysis. It generally stands in direct relation with the dyspnœa, but it may be violent when the dyspnœa is slight, though the converse hardly ever occurs. Neither cough nor voice is hoarse or stridorous as in croup.

The **sputum** is generally mucopurulent and moderately abundant, but it may be scanty.

The **casts** usually appear in it as lumps or pellets, and their branching form is not visible until they have been floated out in water. In number and frequency they vary greatly, from one every few days to many daily.

In one case³ three to five spittoonfuls crowded with casts were brought up daily, and in another⁴ the patient stated that he expectorated "quarts" during an illness of two months' duration.

The number expectorated varies as a rule with the extent of the disease; with the rapidity with which the casts re-form; and with the ease with which they can be expelled, and this in turn depends on the looseness of their attachment to the walls of the bronchi and upon the strength of the patient. It is probable when the casts are numerous and the dyspnœa great that many tubes are involved.

The expectoration of the casts is, as a rule, attended with difficulty, but sometimes they are brought up without effort or serious discomfort. One odd fact is worthy of note, that they are not infrequently expectorated more abundantly at night.

In a case under my own observation,⁵ what appeared to be an asthmatic paroxysm occurred with perfect regularity about 2 a.m. every night, which, after lasting an hour or two, terminated with free expectoration of mucopurulent secretion in which the casts were found.

Hæmoptysis used to be regarded as a common symptom of the disease, but it does not appear to occur in more than one-third of all the cases. It is not rarely entirely absent, and often consists of nothing more than a few streaks of blood upon the outside of the cast. It may, however, be very profuse.

¹ *Würt. med. Corresp. Bl.*, 1857.

² *Sidlo, Allg. Wien med. Ztg.*, 1876, xxi. 259.

³ *Streets, Amer. J. Med. Sc., N.S.*, lxxix. 148.

⁴ *Path. Soc. Trans.*, xvi. 48.

⁵ *West, loc. cit.*

In Flint's¹ case it was estimated that about three pints of blood were brought up within twenty-four hours. In one of Chvostek's² cases the sputum consisted simply of clotted blood, in the midst of which the casts were found. Really profuse hæmoptysis was only recorded in 6 out of the 51 recent cases, and, in the only one of these that died, advanced phthisis was found *post-mortem*.

In most cases the hæmoptysis stands in direct relation to the expectoration of the cast, and ceases as soon as it has been got rid of.

Robinson³ records a case in which, with the hæmoptysis, such severe epistaxis set in as to require plugging of the nares.

The cases recorded are quite conclusive against the theory that the hæmoptysis is due to phthisis, for phthisis is found with plastic bronchitis where there has been no hæmoptysis, and hæmoptysis may occur, and even be profuse, where there is no phthisis. It is probable that in most cases the blood comes from vessels of the bronchi, which have been ruptured by the violence of coughing. There can hardly be any doubt that this is so in the lesser degrees of hæmoptysis, and for the more profuse no other source has been demonstrated.

Feelings of *oppression in the chest* and of *soreness beneath the sternum* are common as in other forms of bronchitis, but actual *pain* is unusual unless there be pleurisy, which, as stated, is a rare association. It does, however, sometimes happen that the patient will complain that each attack is ushered in and attended by pain, referred to the region of the chest in which the casts appear to be formed.

Fever.—The statements made as to the occurrence of fever vary, but it is the rule that the temperature is raised in the acute cases, and not in the chronic, and that too in the absence of any complication in the lung to which the rise of temperature could be referred.

Weil⁴ records a case in which the temperature rose to 104° on three consecutive days, and Escherich⁵ another in which the temperature rose repeatedly as the casts were forming, and fell at once as soon as they were expectorated. The accompanying chart is taken from the record of a case by Hall.⁶

In many of the chronic cases mention is made of fever at the commencement, though none was present in the subsequent stages.

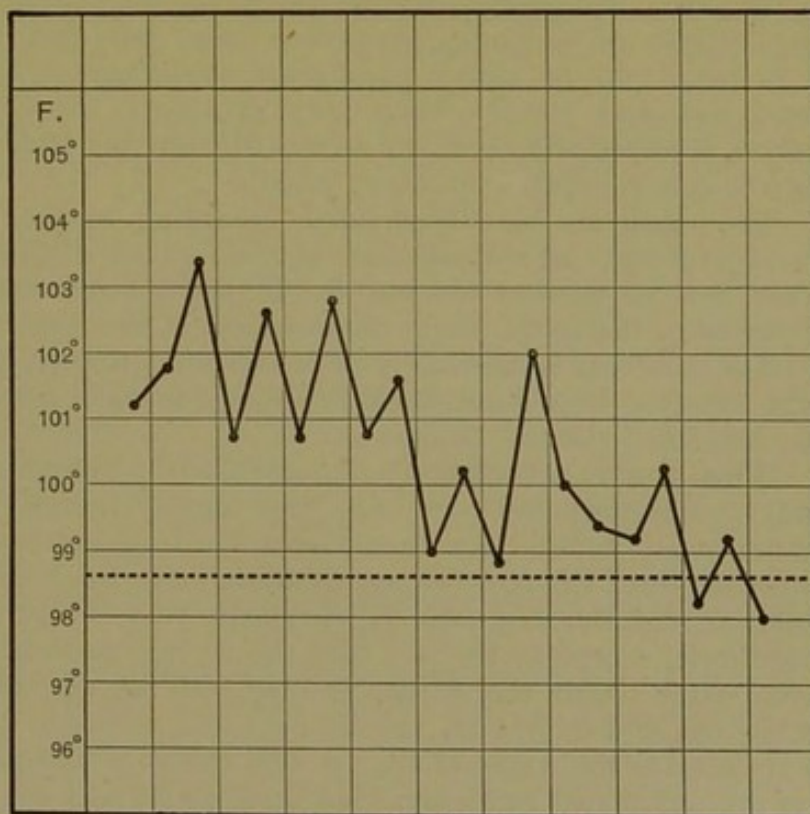


Fig. 37.

Temperature chart of fever accompanying a case of plastic bronchitis, recorded by Hall.

¹ *Med. Rec. N.Y.*, 1874, ix. 41.

² *Allg. Wien med. Ztg.*, 1876, p. 179.

³ *St. Louis, Med. and Surg. Jour.*, 1878, xxxv. 63.

⁴ Gerhard, *Kinderkr.*

⁵ *D. med. Woch.*, 1883, ix. 108.

⁶ *St. Barthol. Hosp. Rep.*, xiii. 125, 1877.

The fever is remittent in type, and may be associated with *sweating* and *loss of flesh*, so as to raise the suspicion of phthisis, a suspicion only set at rest by the course of the case. Sweating is, however, often the simple result of the violent coughing, and the general health even in long-standing cases is frequently well maintained.

Among the occasional accidents of the disease are mentioned *epistaxis*, probably the result of coughing, *diarrhœa* and *albuminuria*. The last affection, though recorded in several instances, is by no means common. It may, I think, in most cases be referred to the dyspnœa. At any rate its occasional occurrence is of no value as indicating any relation between plastic bronchitis and diphtheria.

The physical signs, in the absence of any pre-existent gross disease of the chest, are very indefinite, often no more than those of the chronic catarrh from which the patient suffers. They may be limited to the part of the lung in which the cast is formed; thus, for instance, localised rhonchus, sibilus, or even crepitation may be heard over the seat of disease. Diminution or absence of the breath sounds may give evidence of local obstruction to the tubes, or dulness to percussion, usually at the bases behind, indicate collapse of the lung or even pneumonia.

FORMS OF THE DISEASE.—The disease consists of a paroxysm of dyspnœa and cough, ending with the expectoration of casts. An attack may consist of a single paroxysm only, but this is very rare. As a rule it is made up of many paroxysms recurring at irregular intervals. Both paroxysms and attacks vary very greatly in severity and duration, but the disease is especially liable to recur, though the intervals between two attacks may be considerable, even many years.

The cases are usually divided, according to the severity and duration of the symptoms, into *acute* and *chronic*. This distinction, though convenient, must not be pressed too far, for in the chronic the paroxysms are often as severe as in the acute, while the duration of the acute is not rarely considerable.

The affection is essentially of a relapsing nature, and in the recent cases I have collected I have not found it possible to make any sharp distinction between the acute and chronic forms.

The acute attacks are characterised by greater severity, shorter duration, and higher mortality. In one form the symptoms are so severe that death occurs even before any casts have been expectorated, and the diagnosis is not made until the *post-mortem* examination. Such cases occur chiefly in young children, and the diagnosis is obviously very difficult to make from croup or diphtheria, except by the fact that the larynx has remained free from membrane. Even after death the diagnosis may still be doubtful, for the casts may be numerous, and occupy all the bronchi and even the trachœa. This excessively acute form is the rarest of all. A few cases are described by Fauvel,¹ and in all five are mentioned by Lebert,² but none, so far as I am aware, have been recorded distinct from croup or diphtheria during the last twenty years.

Of the other forms of acute cases, Lebert collected seventeen instances, of which four were fatal—three from suffocation, among which was included the memorable case of Fagge's, in which death was due to the impaction of the cast formed on one side in the main bronchus of the other.

When death does not occur, the case may either recover completely, or pass gradually into the chronic form.

¹ *Mén. de la Soc. d'observ.*, ii. 560.

² *Loc. cit.*

The chronic form of the disease is by far the most common. It consists of a succession of attacks of every degree of intensity separated by very irregular intervals of time. As a rule, as stated, the attacks are longer, the paroxysms less severe, and the mortality lower.

The access is often very sudden—that is to say, it commences without any definite preliminary symptoms. In most, however, there is a longer or shorter catarrhal stage, culminating in the paroxysm.

The access sometimes is marked by one or more rigors, severe enough even to suggest pneumonia, of which they may of course indicate the onset; but in the absence of pneumonia the rigors are rarely so severe, the temperature so high, or the patient so ill as with that disease.

THE DURATION.—In speaking of duration, distinction must be drawn between the duration of the paroxysm, the duration of the succession of paroxysms which constitute an attack, and the duration of the liability to the disease.

The paroxysm is usually short, though the symptoms may take some hours in gradually working up to it.

Both paroxysms and attacks sometimes show a strange periodicity, as in one of my own cases, where they recurred almost regularly at the same hour every night, or in Oppolzer's¹ and in Brik's² cases, where they recurred every month with the catamenia.

The attack thus constituted by a series of paroxysms may last very variable periods, from a few days to many weeks or months. Even in the same patient the different attacks may be of very different duration. In a few cases the attacks have extended over many years.

Thus Nicholl's³ patient brought up a cast almost every day for 7 years, and Gumoens'⁴ every 7–14 days for 8 years, while Waldenburg's⁵ patient suffered for 4 years, and Kischl's⁶ for 10 years continuously.

The liability may last, it would seem, almost all the life: thus Brik's⁷ case was first affected at the age of 36, and after suffering again at 41 and 42, remained free until 60, when the old attack returned. In Walshe's⁸ case the liability was known to have persisted for 14 years. The most remarkable instance of the kind is recorded by Kischl,⁹ whose patient suffered on and off with gradually decreasing intervals for 25 years, and for the last 10 years almost continuously. The intervals between the attacks, as already stated, vary from a few days to many years. The longest recorded interval is 18 years.¹⁰

PROGNOSIS.—Lebert states that of the acute cases 50 per cent. die, and of the chronic about 30 per cent. Of all the cases recorded, *i.e.*, 98, 26 died, yielding a percentage rate of 27. But it must be remembered that many of these died either of some complication produced by the plastic bronchitis or of the antecedent disease upon which the plastic bronchitis was grafted, as, for instance, phthisis, acute tuberculosis, or morbus cordis. Thus of the 51 recent cases, 10 died, but, if the various causes mentioned be excluded, the mortality is reduced to 4 in 51, *i.e.*, about 8 per cent.

The prognosis depends (1) upon the acuteness and extent of the affection, which may be judged by the severity of the symptoms; the amount of fever; and the number and size of the casts:

¹ *Loc. cit.*

² *Loc. cit.*

⁴ Schweizer, *Zeitsch. f. Med.*, 1854, p. 478.

⁶ *Prag. med. Woch.*, 1888, xiii. 69.

⁸ *Dis. of Lungs.*

⁹ *Loc. cit.*

³ *Lancet*, Feb. 1855.

⁵ *Loc. cit.*

⁷ *Loc. cit.*

¹⁰ Brik, *loc. cit.*

(2) Upon the strength of the patient, which is chiefly important as determining the ease with which the casts are expectorated :

(3) Upon the diseases with which it is associated, *e.g.*, phthisis, morbus cordis, tuberculosis, etc.

Age has little influence in modifying prognosis, for though in the young the power of resistance and the strength to combat the disease are small, still there are many cases of long duration recorded in children. As regards general health the prognosis is doubtful, but even in cases of long duration the health and strength are often well maintained.

Many cases get quite well and have no recurrence.

I saw, with Mr Batson, of Dorking, a young soldier 28 years of age, who had begun to expectorate casts some three months before, and had been discharged from the army as incurable. For five or six months he spat these casts. At the time I first saw him he brought up a cupful of casts, of considerable size, daily without much distress or hæmoptysis. From the defective breath sounds they seemed to come from the right base. His nutrition was maintained and his temperature normal. He was placed upon full doses of iodide of potassium, began to improve at once, and in the course of a few weeks got quite well. The whole duration of the attack was about six months. He remained under observation for many months, I think two or three years, and had no recurrence.

DIAGNOSIS.—The positive diagnosis can only be made by the expectoration of the characteristic casts, but it is necessary to exclude pneumonia and croup or diphtheria, to which the casts might be secondary.

1. From *pneumonia* the diagnosis is made by the absence of the characteristic fever symptoms and physical signs.

2. From *croup* or *diphtheria* by the absence of membrane on the fauces and of laryngeal symptoms.

The paroxysmal symptoms might further suggest *asthma*, *pneumothorax*, or *impaction of a foreign body* in the bronchus.

3. From *asthma* it may sometimes be difficult to make the diagnosis until the casts are discovered.

4. From *pneumothorax* the diagnosis is made by the absence of the displacement of organs and other physical signs of that affection.

5. From *foreign body in the bronchus*, with which of course the attacks are identical, by the history and relapsing nature of the case.

Where the casts are large enough to attract attention the diagnosis is easy, but in many cases of slight degree the patients complain of nothing but attacks of cough and shortness of breath of irregular recurrence, and the true nature of the case is not recognised until plastic bronchitis is thought of, and the patient made to expectorate into a vessel containing water. Then the characteristic branching casts are found and the diagnosis is clear.

CASES.

1. Plastic Bronchitis, very numerous casts, with hæmoptysis, lasting only a few days.

The patient, a man of 45 years of age, had lived for many years in Jamaica. He was a free liver, and came under his doctor's care for an attack bordering on delirium tremens. He became much better under treatment, when suddenly he was attacked with cough and hæmoptysis and said that "he was spitting up his lungs."

The hæmoptysis was slight and the amount of expectoration small. It consisted chiefly of casts, of which a considerable number were brought up. He suffered a little from dyspnoea or cough, but there was at no time any symptoms of suffocation.

The temperature was not raised, there were no signs of pneumonia or diphtheria, nor was the patient specially ill.

The casts were very numerous, of the normal composition and appearance, mostly of small size, the largest measuring about $1\frac{1}{2}$ inches long and being in the thicker part about $\frac{3}{16}$ inch in diameter.

The attack lasted only a few days and then subsided. The patient was seen frequently for the next two months, but had no return of the affection.

He left the country again shortly after, apparently in good health, and his subsequent history is not known.

2. Plastic Bronchitis of short duration and moderate intensity; probably a similar attack two years before.

A strong, robust man of 38, subject occasionally to rheumatism, was attacked while at Monte Carlo with pain in the chest beneath the sternum and with shortness of breath. A few days later, after a similar attack, he coughed up two lumps like macaroni, white, stringy, tough, and streaked with blood. It felt as if they came from the seat of pain, and their expectoration gave immediate relief.

Five days later he brought me two casts an inch and a half-inch long respectively. He could attribute the attacks to no cause, and had been feeling quite well during the interval. It is possible that he had a similar attack two years previously following influenza. He was placed upon iodide of potassium and had no return of his symptoms.

3. Plastic Bronchitis of twelve months' duration in a Child—Death from asthenia. *Post-mortem*.—Recent acute miliary tuberculosis of lungs.

Mary F., aged 9, was admitted into St. Bartholomew's Hospital on Jan. 19, 1897. Her history was as follows:—

She had been well till twelve months ago, when she began to cough, and brought up stringy sputum like pieces of flesh; the attacks came on with a good deal of dyspnoea and were relieved by the expectoration of these lumps.

The attacks occurred sometimes once a week and sometimes as often as two or three times in the day.

Nine months later she was admitted into the hospital, when the diagnosis was made of fibrinous bronchitis.

Three weeks ago she was attacked with a great deal of pain in the right side, and about this time she began to waste rapidly. Two weeks ago she had some hæmoptysis and had several attacks subsequently; but it had no very definite relation to the expectoration of the casts.

The child was very pale, feeble and ill, suffering a great deal from dyspnoea, which frequently compelled her to sit up in bed for hours together; the cough was very severe and wearing.

The child was pigeon-breasted, and very thin; the movements of the chest were poor but equal on both sides; fine crepitation was heard over the whole of both lungs, and at the right apex some bronchial breathing and bronchophony; the heart was natural, the sputum mucopurulent, the temperature normal, the pulse 108; the sputum was examined carefully, but no tubercle bacilli were found. On Feb. 11 she brought up some casts after an attack of cough and vomiting.

The child said she knew she had brought the casts up by the bitter taste in her mouth, with which she was familiar.

On Feb. 15 she brought up some more casts, and again on Feb. 23.

She failed rapidly in strength, and a day or two later died of exhaustion.

The *post-mortem* examination showed the following changes:—

The bronchial glands were enlarged but not caseated, and there were several enlarged glands at the bifurcation of the trachæa. The lungs were covered with fine adhesions which were most marked at the apices; they were studded throughout with recent miliary tubercles.

The bronchi were all somewhat dilated, and contained mucous secretion but no casts.

All the other organs were healthy.

4. Plastic Bronchitis for many months at irregular intervals.

A man aged 42, a carman, got a cold in August which he could not shake off; in October he was laid up by it for six weeks. For ten days in November, being then about and at work, he spat up several casts about the size of a crow quill. On December 31st he had a sudden attack of severe dyspnoea, lasting several hours, and similar attacks of moderate severity during the month following.

Between the attacks he was quite well and free from all shortness of breath. When the attack came on, the breath became gradually shorter, then the cough commenced and continued till a lump or two of blood, of the size of a small marble, was expectorated, after which the breathing got better.

Except the original bronchitis no cause for his attack could be traced.

In September I saw him again. The attacks had continued all the time, recurring about every three weeks, the longest interval being eight weeks.

The casts were for the most part of small size, about 1 inch long only, and a few only branched.

It was thought from the physical signs that these casts were formed in the lower part of the right lung.

Iodide of potassium was administered, but without any marked effect; the subsequent addition of Liq. hydrarg. perchlor. seemed to be most efficacious, but the case shortly afterwards disappeared from observation.

5. Plastic Bronchitis confused with Asthma—Periodical attacks daily at 2 a.m.

A woman, 40 years of age, stated that she had been in good health until attacked by what was called asthma two years before she came under observation. She had not been previously subject to bronchitis or to shortness of breath, and her symptoms developed all at once. Since that time they gradually became worse, until she was hardly twenty-four hours free from an attack. When under observation the paroxysms came on with almost perfect regularity at 2 a.m., the patient being awakened from sleep with great dyspnoea.

The attack was asthmatic in type, but there was no cyanosis. After lasting two or three hours expectoration became free and the paroxysms gradually passed off.

The attacks so strongly resembled asthma that the diagnosis of asthma had been made, but the history of the affection having developed only during the last two years and the remarkable periodicity of the attacks raised the suspicion of plastic bronchitis, and in consequence the sputum was carefully examined. This resulted in the discovery of small but numerous casts constantly present in the mucous sputum. They were small, grayish white, mucoid in appearance and much ramified. The largest were not thicker than a goose-quill, while many were much smaller. In length they varied, the longest being about 2½ inches. No account of hæmoptysis was given, and none occurred while the patient was under observation.

There were no physical signs in the chest, and the patient's general condition was fairly satisfactory, although the disturbed rest at night had made her somewhat feeble. The temperature was not raised, nor was there any sleep-sweating while under observation.

The various drugs employed produced no effect, and the patient left the hospital *in statu quo* after six or seven weeks' treatment.

The subsequent course of the case could not be traced, as the patient disappeared from observation.

6. A similar case occurred in a man of 55, who for three or four years was awakened almost every night by coughing, and coughed for ten to fifteen minutes violently, until he was relieved by the expectoration of a lump of viscid substance.

Except for this he was perfectly well and had not been laid up at any time. It was suspected to be plastic bronchitis from the history, and subsequently proved to be so.

Iodide of potassium gave him great relief, and when last seen he had been quite free from the paroxysms of coughing for two or three months.

TREATMENT.—The objects of treatment are—

1. To get rid of the casts.
2. To prevent their reformation.
3. To control the symptoms.
4. To deal with the complications as they arise.

To fulfil the first indication, emetics, *e.g.*, antimony and ipecacuanha have been administered, but they increase the prostration and do not appear to hasten the expulsion of the cast.

To loosen the clot, alkalis and alkaline waters have been recommended, but are of no advantage. Inhalations have been also used for the same purpose; of these lime water has been the favourite ever since. Sax¹ showed that membrane and casts dissolved in it most readily. But it is more than doubtful whether any inhalation reaches the seat of the disease at all, and if it did it could in most cases do no more than moisten the extremity of the cast. If inhalations do any

¹ *Wien med. Tr.*, 1886, xxvii.

good, their effect is most probably due to the steam or spray, and not to the actual drug employed.

With the object of controlling the morbid process, mercurials, *e.g.*, calomel or gray powder, have been freely administered, but they seem to have little if any real influence upon the disease.

There have been recommended besides, by one author and another, counter irritants, expectorants, sulphur, acetate of lead, sweet spirits of nitre, or even bleeding, but all alike with equal inefficiency.

There is one drug only as to the value of which most observers are agreed, *viz.*, *iodide of potassium*. This, exhibited in full doses, is held by some to have controlled, and by others to have entirely cured, the affection.

The only instances of successful treatment are those in which iodide of potassium has been freely administered. This is the line of treatment which I have always adopted, and, in the cases I have had under my own treatment, with invariable success.

The complications and various symptoms which present themselves in the course of the disease must be treated in the appropriate ways as they arise.

27. SECONDARY BRONCHITIS.

Of bronchitis secondary to the affections, little need be said, except to indicate the chief points of importance in each group.

I. Bronchitis due to congestion.—Congestion may be the result, as already stated, either of mechanical obstruction or of collateral hyperæmia. If congestion produce any physical signs at all, they will be those of bronchitis.

A. Bronchitis due to mechanical obstruction.—The obstruction is caused by the interference to the exit of blood from the pulmonary veins, and might therefore be due to the pressure upon them of a mediastinal tumour, aneurysm, or new-growth, but its commonest cause is disease of the heart. This is only to be expected, when it is remembered that the bronchial veins are in close connection with both sides of the heart—with the right through the vena azygos and vena cava, and with the left through the free anastomosis between the bronchial and pulmonary veins.

Among diseases of the heart, the obstruction might occur with any affection of the left side, *e.g.*, with the fatty heart of old people, with the later stages of aortic disease, with acute or chronic pericarditis, or with the muscular degeneration due to acute fevers and other general causes.

It is most frequently the result of **disease of the mitral valve**. Indeed, in mitral disease, the first symptoms which bring the patient to the doctor are often pulmonary rather than cardiac. In mitral stenosis especially the pulmonary symptoms may be so marked as to mask the cardiac, and even cause the mitral disease to be overlooked, and they may be brought on not only by exposure or cold, but even more certainly by over-exertion and fatigue.

The expectoration in these cases is moderate in amount. Hæmoptysis is not uncommon, the sputum being either simply streaked with bright blood, or containing dark lumps which sink in water; the former comes from ruptured vessels in the bronchi, the latter from the vesicles of the lung. The

dark lumps are altered blood, and come from lobules of the lung, into which the distended vessels have given way. If many vessels burst, the hæmorrhage is more abundant, and besides the lumps, the sputum contains bright blood, mingled with air bubbles. The corresponding pathological condition of the lung is that of infarct, and this may be of sufficient size to yield physical signs.

The **prognosis** with morbus cordis varies according as the bronchitis is an early or late complication; if late, and associated with general dropsy, the prognosis is bad; but if early, and without other signs of venous obstruction, the prognosis is good; nor does the expectoration of the small dark lumps modify it much, for such clots are often brought up at intervals for years without harm. More profuse hæmoptysis is of graver import, but I have seen a considerable amount of bright blood brought up for two or three days together, from time to time, without much distress or sufficient dyspnœa to keep the patient in bed. Even in the later stages of morbus cordis, both aortic and mitral, and in what seem at the time to be desperate cases, the hæmoptysis may subside, the signs of infarct disappear, and recovery from the lung-symptoms occur.

B. Bronchitis due to collateral hyperæmia.—Under this term are included two groups of cases.

In the one group the hyperæmia is of the nature of a reactive inflammation, caused by the irritation of some local lesion; the bronchitis will then be localized or diffuse according as the lesion is limited to one spot or disseminated through the lung; thus bronchitis localized to the apex may be the earliest, and perhaps for some time the only, sign of commencing phthisis, or again a general bronchitis may be the only localizing evidence of the disseminated lesions of general tuberculosis or of secondary cancerous growths in the lung.

In the other group of cases the collateral hyperæmia is more of the nature of a physiological than of a pathological process. When the respiratory function of any part of the lung is impaired or abolished, the rest of the lungs endeavours to supply the deficiency; the extra work thrown on these parts must, if they are to be equal to the demand made upon them, be associated with increased blood-supply—that is, with congestion. If the work demanded be more than they can do, the physical signs of overwork, that is, of bronchitis, will appear.

How far compensation can be carried depends upon the extent of the lesion and upon the rapidity of its development. If the lesion develop slowly, as in chronic phthisis, it is remarkable how large an amount of disease may exist without grave symptoms.

In acute pneumonia, on the other hand, where a large part of the lung is suddenly thrown out of gear, the respiration is greatly embarrassed and the signs of congestion or bronchitis often develop in the other parts of the lung. In young and healthy persons, fortunately, this does not often happen, but it is of frequent occurrence in the aged and in patients debilitated by disease or other causes.

When bronchitis develops under these conditions, as a complication of acute pneumonia, it is of the gravest possible import, and is usually the precursor of death, but if, on the other hand, pneumonia develop in the course of bronchitis, the prognosis is not necessarily so grave. In the former case the bronchitis means the breakdown of the lungs, or failing compensation; while in the latter case, though a serious complication, it does not have this significance.

Similarly very large effusions may be met with in the pleura, which having formed slowly have produced but slight symptoms, while with much smaller

effusions, which have developed rapidly, the respiration may be greatly embarrassed. If under these circumstances the signs of bronchitis arise in the opposite lung, they become the indication for immediate paracentesis.

In like manner, distension of the abdomen from any cause may lead to respiratory embarrassment. This it does by interfering with the movements of the diaphragm and lower ribs, by reducing the actual thoracic capacity from the displacement upwards of the diaphragm, and lastly by the collapse which occurs in the bases of the lungs. If in such cases bronchitis arise, it becomes the indication for the immediate relief of the abdominal distension if that be possible; for paracentesis where ascites is the cause, and for puncture of the bowel or other measures if there be tympanites. In the same way, bronchitis may arise in the course of an ovarian tumour, both with or without ascites, and it would rather be an indication than a contra-indication for operation.

I have also met with bronchitis in the last weeks of pregnancy, which I believe admits of the same explanation, for it resisted all the ordinary methods of treatment, and was immediately relieved by parturition.

2. Bronchitis in the course of fever.—In the specific fevers bronchitis is of frequent occurrence.

In *measles* it is so common as to form an almost essential part of the disease, and to constitute, with the complications to which it leads, in many cases the chief or only cause of anxiety.

Almost the same may be said of *influenza*.

In *typhoid fever*, again, most cases suffer at some time during their illness with bronchitis. It commonly develops in the second or third week of the fever, but it is not as a rule severe enough to modify much the course of the case. It may, however, be an early complication, and then lead to difficulty in the diagnosis.

For instance, a young married woman was taken suddenly ill with acute fever, and when seen two days later presented the signs of acute general bronchitis, but with a temperature of 104. The diagnosis of pneumonia naturally suggested itself, but no conclusive physical signs were ever found, and in a few days the characteristic signs of typhoid fever manifested themselves. The case ran its ordinary course and ended in recovery.

In another case, which was, it was thought, well advanced in convalescence from typhoid fever, acute bronchitis suddenly set in; the temperature rose and became of a hectic type with considerable daily remissions. The suspicion arose of acute pulmonary mischief, possibly of a tubercular nature, and for this reason I was asked to see it. I thought it to be simply a case of relapse, complicated with acute general bronchitis, and the subsequent course proved it to be so. As in the previous case, recovery took place.

A small number of cases of typhoid fever die of acute congestion of the lungs, with the signs of general bronchitis, and *post-mortem*, beyond œdema and collapse of the bases no other lesions than those of bronchitis are found.

Such a condition is common in all fevers with high temperature, and even as the result of high temperature alone, as in sunstroke and rheumatic hyperpyrexia. In typhus fever congestion of the lungs is one of the most frequent causes of death, and hardly any case escapes without suffering from it in some degree.

In all these cases of high temperature the question may fairly be raised whether the acute congestion does not depend in great measure upon the heart rather than the lungs. As an argument in favour of this view may be quoted the rapid improvement both in the physical signs in the chest and in the patient's general condition which follows the use of the cold bath. Thus, a patient deeply cyanosed and unconscious, with râles of all kinds over the chest, is placed in the cold bath, and in a few minutes the colour improves, the cyanosis passes off, consciousness returns, and the râles may completely vanish.

It is in this respect that an unduly rapid pulse, as well as an unduly rapid rate of respiration, is regarded as a sign of bad omen in severe fevers.

Although bronchitis may occur with any fever, it is most common with the acute exanthemata, and this association of bronchitis with acute skin eruptions is observed also in cases which do not belong to the group of fevers at all. Thus it has been met with in ordinary acute erythema, or with the roseola of syphilis; and like asthma it has been connected with the sudden disappearance of an eruption, acute or chronic, as, for instance, eczema, psoriasis, or urticaria. All these cases are, however, of interest rather than importance. They are by some regarded as vasomotor neuroses, and are placed in relation with those curious conditions in the skin which have been described as angeio-neurotic oedema. Some further reference to these cases will be found in the section dealing with Asthma.

The possible *causes of bronchitis in specific fevers* are three—

1. Acute failure of the heart, producing acute congestion of the lungs. This has already been spoken of.
2. Direct irritation of the bronchial mucous membrane by some toxic substance circulating in the blood. This view is only supported by the analogy of the irritative effects experimentally produced by the administration of cantharides in animals.
3. Infection of the bronchial mucous membrane by pathogenic organisms, either the specific organism of the fever itself, *e.g.*, Eberth's bacilli in typhoid fever, Löffler's bacilli in diphtheria, etc. (*homologous infection*), or some of the commoner non-specific organisms, like the streptococcus, or pneumococcus, introduced probably from the mouth or throat (*heterologous infection*). So far as observation goes at present the latter appears to be the more frequent occurrence. If this be so, nothing is more likely to reduce the liability both to bronchitis and pneumonia in fevers than the careful and frequent cleansing of the mouth and pharynx by antiseptic washes and sprays.

We may probably correctly apply to bronchitis Kanthack's¹ and Horton Smith's conclusions in relation to broncho-pneumonia, and regard the bronchitis occurring in the course of specific fevers as due in most cases to heterologous infection, *i.e.*, to some other than the specific organism of the fever.

Kanthack and Horton-Smith's results—

- | | | | |
|------------------|---|---|---|
| 1. Diphtheria | } | { | Diphtheritic (diphtheria bacillus, <i>heterologous infection</i>). |
| 2. Influenza | | | Pyococcal (rare— <i>heterologous infection</i>). |
| 3. Tuberculosis | | | Mixed—1. Diphtheritic and pyococcal—(<i>mixed infection</i>). |
| | | | 2. Influenzal " " |
| | | | 3. Tubercular " " |
| 4. Typhoid | | | pyococcal (<i>heterologous</i>). |
| 5. Scarlet fever | } | { | pyococcal (provisionally) <i>heterologous</i> . |
| 6. Measles | | | |
| 7. Pertussis | | | |

3. Bronchitis in the course of general diseases.

1. *Gout*.—The bronchitis which occurs with gout is of the chronic recurrent form, and affects the large tubes. It may precede or follow an acute attack, or develop in the course of it. In gouty patients a morning cough with the

¹ *St. Barthol. Hosp. Rep.*, 1896, p. 51.

expectoration of a small amount of yellow phlegm is common, and it is often at once relieved by an outburst of gout in the joints, or yields readily to the ordinary gout-remedies. Many patients recognise the association themselves, and by acting on the hint stave off the attack in the joints.

Crystals of uric acid have been described in the bronchial mucous membrane, but the observation has not been sufficiently confirmed. Urate of soda crystals in the smaller bronchi were recorded by Bence Jones, but this observation is, I think, unique.

Bronchitis stands, as already stated, in frequent relation with psoriasis and eczema, and they, as well as albuminuria, are not infrequently associated with gout, and may yield to the same remedies.

2. *Alcohol*.—Chronic bronchitis is very common in chronic alcoholism. It may be the direct effect of drink—as there is reason to consider the chronic pharyngitis and laryngitis of drunkards is—but in many cases it is the result of chills, incurred while drunk, to which, owing to the effect of alcohol upon the skin, drunkards are especially liable, aggravated by the general failure of health, and by the degeneration in the heart's vessels, to which alcoholism leads.

3. *Rheumatism and rheumatic fever*.—In rheumatic fever, bronchitis is a rare complication, but chronic bronchitis is not infrequently associated with chronic rheumatism as it is with gout.

4. *Syphilis*.—The association between the acute exanthemata and bronchitis has been already mentioned, but there is, it is stated, a special tendency to chronic bronchitis in the chronic forms of syphilis, and that independently of any lesion in the lungs. I cannot say that I can endorse this from my own observation.

5. *Ague*.—Bronchitis may occur in the course of severe ague in the same way as it arises in high fever, and from the same causes, but a form of intermittent bronchitis is described as occurring in ague-districts, and in persons who have suffered from ague, which yields to quinine and has been regarded as due to the malarial poison. I do not know of any examinations of the blood in these cases to show the presence or absence of the malarial parasite.

6. *Bright's Disease*.—With *parenchymatous nephritis* bronchitis is common. It is usually associated with general dropsy, and is in many cases rather an œdema of the bronchi and lung than an inflammation. If severe, it is a serious complication, and often the cause of death.

With *interstitial nephritis* or *granular kidney*, bronchitis is often chronic and affects the large tubes chiefly. It may be connected with the degeneration of the bronchial vessels as part of the general arterio-capillary changes. Where the heart fails, as it often does in the later stages of the disease, bronchitis will develop, as in other forms of heart failure. In the condition of chronic uræmia, it may very likely be of toxic origin.

7. *Pregnancy*.—Bronchitis is not an altogether uncommon complication in pregnancy, and may be due to the lungs being unable completely to meet the double demand made upon them by the requirements of the foetus as well as of the mother. In the later weeks especially, where there is great abdominal distension, it may be due, as already stated, to the mechanical results of compression. If there be morbus cordis, bronchitis is especially likely to develop, and it may be the first sign of the cardiac failure which is to ensue after parturition.

If severe bronchitis be present at the time of parturition, it may offer a serious impediment to delivery, owing to the difficulty of holding the breath so as to assist the movements of expulsion. If there be great cyanosis or very violent coughing, bronchitis may lead to abortion, but in milder cases it has no definite effect upon the course of the pregnancy.

8. *Asthma*.—In idiopathic asthma the bronchitis follows the paroxysm, and is caused by it, but paroxysmal dyspnoea, asthmatic in type, may arise in the course of any bronchitis. These attacks are no doubt largely dependent upon the obstruction of the tubes by secretion, and are relieved by free expectoration. In plastic bronchitis such attacks are the rule, and pass off as soon as the casts are expectorated.

Hay-asthma comes rather into the next category of bronchitis, viz., that due to direct irritation, unless both the asthma and bronchitis should prove to be of bacterial origin, as is not unlikely.

9. *Mechanical Irritation*.—The bronchitis which is so frequent in certain occupations is caused by the direct irritation of the mucous membrane by the substances employed. It leads sooner or later to organic changes in the lungs, and will be more fully discussed under the heading of pneumo-koniosis.

The **treatment** of the forms of secondary bronchitis is modified only by the causes upon which the affection depends, but it is often surprising, as, for instance, in gout, how quickly an attack, which has resisted the ordinary remedies, subsides so soon as the predisposing cause has been recognised and appropriately treated.

28. BRONCHIECTASIS.

Bronchiectasis is essentially a simple dilatation of a bronchial tube, and its structure is therefore the same as that of a bronchus. On the surface is the basement membrane covered with epithelium, often still retaining its cilia, while in the deeper substance the muscular and elastic tissue and cartilages are found, and though in old bronchiectases there may be no cilia and even no cartilage left, still the basement membrane persists, and may be traced as a continuous lining over the whole cavity. There ought, therefore, to be, one would suppose, but little difficulty in deciding what is and what is not dilatation of the bronchial tube; but great confusion has been introduced by the use of the term to include certain chronic cavities in the lung, lined with a smooth membrane without any trace of basement membrane, and though some of the cavities may, it is true, have taken origin from a dilated bronchus, most of them are due to other causes. It would tend greatly to clearness if such chronic cavities were described by some other name, and the term bronchiectasis reserved exclusively for those cavities the nature and origin of which is certain.

Bronchiectasis is generally divided into three varieties—the *cylindrical*, the *saccular*, and the *trabecular*. The cylindrical is nearly always a true bronchiectasis, the saccular frequently, the trabecular generally not.

If the trabecular variety—the false bronchiectasis of Grainger Stewart¹—be excluded, bronchiectasis is by no means a common affection. When met with it is most frequently associated with two conditions, viz., chronic bronchitis and fibrotic changes in the lung. It affects usually the medium and small tubes, rarely the large, and on the whole is most frequent in the lower lobes.

In the slighter forms it can only be detected by carefully splitting up the bronchi with scissors.

Bronchiectasis occurs also as an *acute* and a *chronic* affection, the acute being most frequent in children and the chronic in adults; and while in the former the walls are but little altered, in the latter the changes are considerable.

¹ *Edin. Med. Jour.*, xiii. 246.

According to the condition of the walls the cases of bronchiectasis have been placed in four groups (Biermer).¹

1. Ectasis with simple catarrhal swelling and relaxation of the bronchial walls, a form usually met with in children in connection with bronchitis and disappearing as the bronchitis passes off.

2. Ectasis with hypertrophied walls, the mucous membrane being thick, vascular, velvety, and occasionally villous, and the glands, cartilages, connective tissue, and even the elastic tissue increased. This form occurs usually in the medium tubes, and is generally cylindrical or spindle-shaped.

3. Ectasis with thin atrophied walls, most frequent in the saccular or ampullary form, but met with occasionally in the cylindrical ectasis of small tubes.

4. Ectasis with trabecular degeneration in which the essential structures of the walls atrophy and disappear, their place being taken by fibrous tissue.

For the purpose of description the ordinary classification into cylindrical, saccular, and trabecular bronchiectasis is the most convenient.

I. Cylindrical.—In the cylindrical form, as the name implies, the dilatation involves the whole circumference of the tube and extends some distance along it, having often, however, a fusiform rather than a strictly cylindrical shape. It may be widespread throughout the whole lung, commencing in the medium bronchi and extending even to the periphery, where it may terminate in a globular enlargement. The affected tube is often two or three times its normal calibre, and some of the smaller ones may measure even $\frac{1}{4}$ to $\frac{1}{2}$ an inch in diameter. The dilatation is, however, usually more limited, involving the tubes in a part only of one lung or it may be of both, but it is often restricted to the divisions of one tube or even to a single bronchus.

The dilatation may be uniform, but is not infrequently moniliform, *i.e.*, a successive series of fusiform dilatations are present, the tube between preserving its normal calibre.

The walls are usually thickened and the mucous membrane in a condition of acute or chronic catarrh, sometimes swollen, vascular, and spongy, at other times atrophied, as in some forms of chronic bronchitis, the strands of fibrous and muscular tissue being unduly prominent and forming a coarse meshwork between which the tube may be pouched. In other cases the walls may be thin and almost transparent, or they may appear but little altered from the normal.

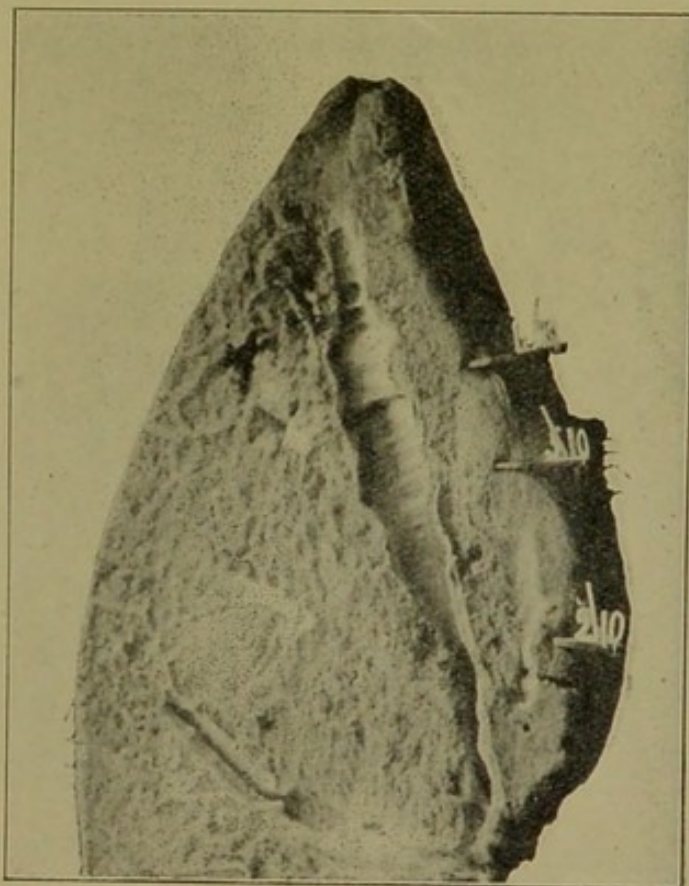


Fig. 38.—Acute cylindrical bronchiectasis in an adult.²

¹ *Virch. Arch.*, xix., 1860.

² Taken from a specimen (1679) in the museum of St. Bartholomew's Hospital, and was obtained from the body of a young man who died on the ninth day of an attack of acute pneumonia. There was nothing in the history or in the condition of the bronchi which suggested any previous disease of the lung. The lung is in a condition of gray hepatisation. The coats of the bronchi are thin.

The lung tissue around the dilatation presents, as a rule, the lesions associated with chronic bronchitis, viz., emphysema and partial collapse, but it may appear normal, or at other times be somewhat fibrotic.

In its acute form this variety has been most frequently described in children who have died during bronchitis or whooping cough. The greater frequency in children may be apparent only owing to the much higher mortality of bronchitis in them, but it is also probably real, on account of the smaller resistance which their bronchial tubes offer to disease.

It is also met with, though much more rarely in the adult, both with bronchitis and with inflammatory lung changes.

In chronic cases where there is much fibroid induration, the cylindrical and the saccular forms are often associated together in the same lung, and the distinction between them becomes of little importance.

In some cases the bronchiectasis is very extensive and present in almost all parts of the lung, so that the lung looks as if riddled with small cavities. These cavities prove in most instances to be dilated bronchioles, and the condition has accordingly been described as **Bronchiolectasis**. These cases are all very rare, and so far as is known are met with only in young children.

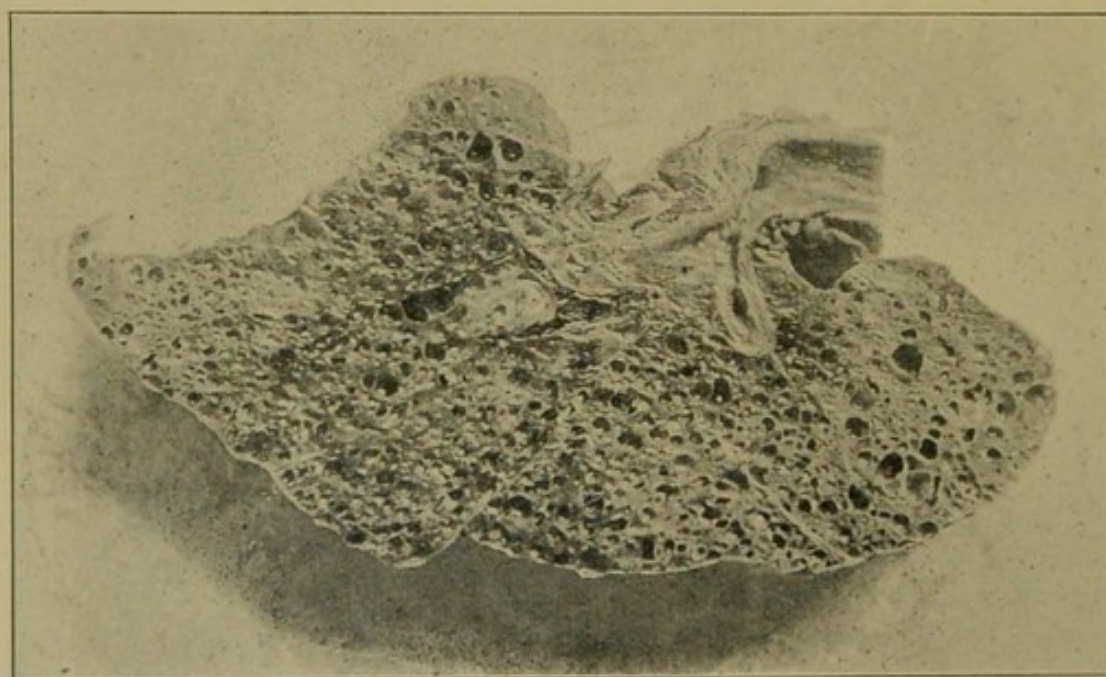


Fig. 39.—Acute bronchiolectasis (Dr. Morley Fletcher). The figure shows the honeycomb appearance of the whole lung. This was due to small cavities, which proved to be dilated bronchioles. On the surface some of these cavities projected and looked like bullæ. There was no evidence, even on careful microscopical examination, of tubercle or of broncho-pneumonia.

(This figure is better viewed sideways.)

An excellent example of this condition is recorded by Dr. Morley Fletcher.¹ It was met with in a child aged 3 years, who died of acute bronchitis.

Two good instances of the same condition are recorded by Dr. Sharkey.²

1. In a child of 2 years of age, who died twelve days after an attack of diphtheria. Small hard patches, somewhat pigmented, were disseminated through the lungs, the centre of each occupied by a dilated bronchus, the walls of the affected bronchi were infiltrated with small cells, and the infiltration had extended to the adjacent inter-alveolar tissue.

¹ *Path. Soc. Trans.*, 1901.

² *St. Thomas' Hosp. Rep.*, vol. xxii.

2. In a child of 4 years of age, who had suffered from cough for four months, the lungs were bulky and the surface showed numerous small, round, bladder-like elevations, which corresponded with small cavities. A similar condition to that on the surface existed throughout the whole of both lungs. The cavities were minute, the largest the size of a pea, with smooth walls, and containing either air only or frothy mucus. Microscopical examination proved these cavities to be dilated bronchioles, the walls of which were infiltrated with small cells. The larger bronchi were unaffected. The rest of the lungs contained numerous patches of broncho-pneumonia, with collateral emphysema.

In another case, recorded by Dr Tooth,¹ the honeycomb appearance of the lung, which was similar to that described in the other cases, proved on microscopical appearance to be due not to simple bronchiectasis, but to actual cavities of minute size, produced by the breaking down of small patches of broncho-pneumonia.

2. Saccular (Turtle-lung — Gruyère - cheese - lung).

—The saccular variety is more commonly met with in the form of globular pouches at the periphery of the lung or irregularly distributed through the whole or greater part of a lobe, especially the lower. Saccular dilatations do not, as a rule, exceed $\frac{1}{3}$ to $\frac{1}{2}$ an inch in diameter, but they are said sometimes to reach the size of a small Tangierine orange; the walls are formed of dense connective tissue, in which the constituents of the bronchial walls can be sometimes, but not by any means always, traced. The pulmonary tissue

around them is generally in a state of advanced fibrotic induration. Though generally numerous, there may be but a single such dilatation in the lung.

I have seen a case in which there were two small pear-shaped cavities in the lung, each about the size of the thumb nail, one in the apex and the other in the base, somewhat deep-seated and surrounded by what appeared to be normal lung tissue; the walls were slightly thickened but the fibrous tissue had not extended into the lung. In one of them, viz., that in the base, a small pulmonary aneurysm was found ruptured. The patient, a middle-aged woman, had been under treatment for slight hæmoptysis a fortnight before her death; no physical signs were detected, nor could they have been, and she died suddenly one day in the street from profuse hæmoptysis.

In some of the smaller saccules the communicating bronchus is not easy to find, and when they are filled with inspissated secretion they have been described as closed cysts,² but it is doubtful whether a saccular bronchiectasis ever becomes really closed.

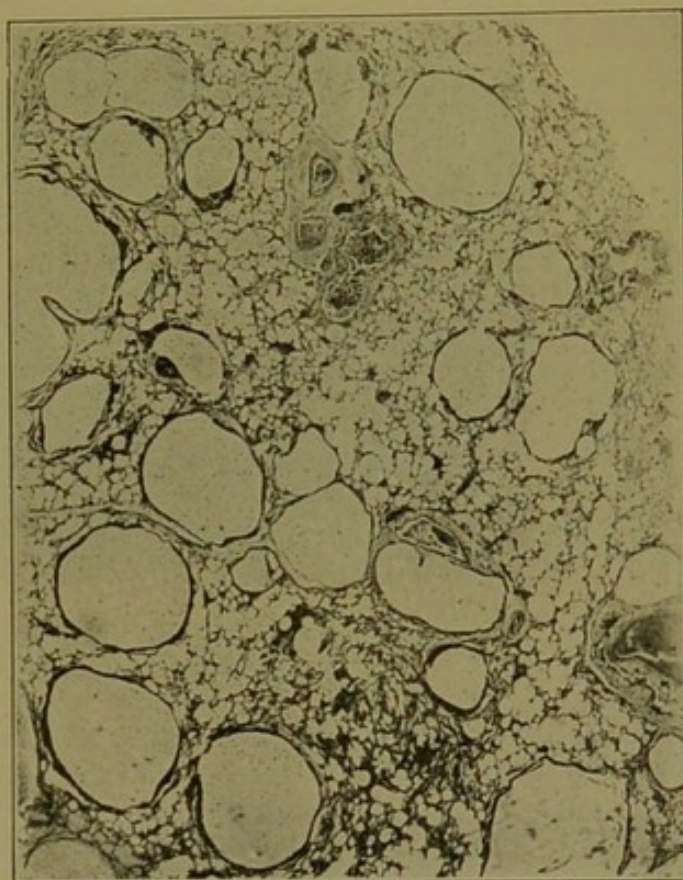


Fig. 40.

Section of same lung magnified. The smooth-walled cavities are well shown, with a definite lining. These are the dilated bronchioles, which were filled with fluid composed of mucus and desquamated epithelium. The tissues round them were infiltrated with small cells. A good deal of vesicular emphysema was present also, but it did not correspond in any way with the cavities. The darker portions of the figure represent the vesicles, which, except for the emphysema described, show no marked changes.

¹ *Path. Soc. Trans.*, 1897.

² Schröder, *D. Klinik.*, 1854.

By the irregular contraction of the strands of fibrous tissue connected with it, a saccular bronchiectasis may become extremely irregular in shape, and by being

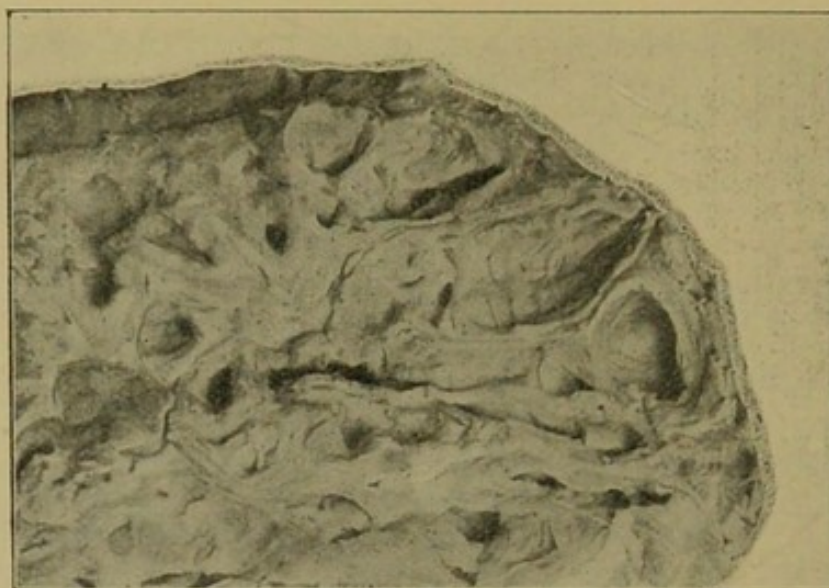


Fig. 41.—Bronchiectasis in a fibroid lung. Some of the cavities are saccular, some cylindrical, some quite irregular.

pinched in one place and pulled out in another, the walls may come to have a trabecular appearance, but their structure proves them to be still only dilated

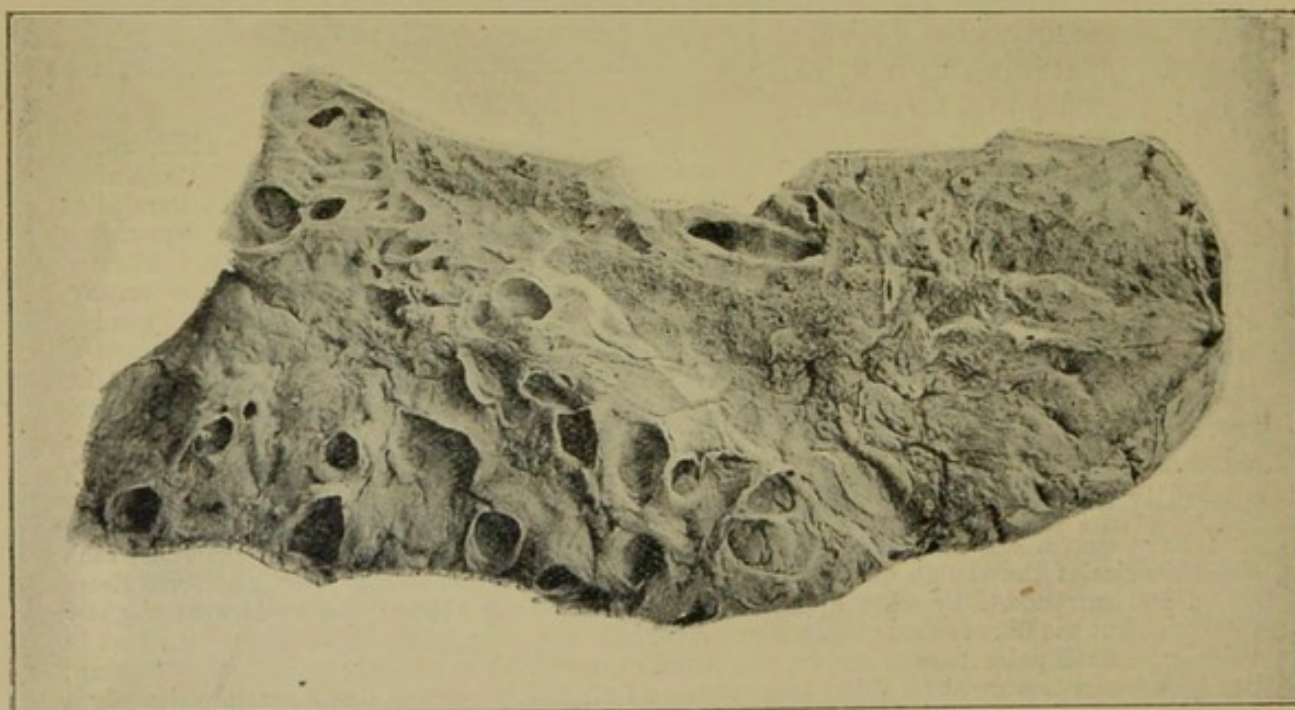


Fig. 42.—A specimen of saccular bronchiectasis. The whole lower lobe is converted into dense fibrous tissue, in which almost the only trace of the lung left consists in the saccular pouches formed by the dilated bronchi. (From the Royal College of Surgeons Museum, prep. 3361 B.)

(This and the preceding figure are best viewed sideways.)

bronchi (cf. fig. 80). On the other hand, not a few saccular pouches prove on microscopic examination to be no longer simple bronchiectases but chronic

cavities formed by ulceration. This is the case with many instances of so-called bronchiectasis after the impaction of a foreign body in the bronchus.

3. Trabecular.—What is called the trabecular form is, in most cases, really nothing more than an irregular chronic cavity having a smooth lining and very dense walls of connective tissue, in which no trace of bronchial structure remains, except in the trabeculae. These cavities usually communicate with two or more bronchi. There may be a single such cavity in the lung, but more often there are several, which, by opening into one another, form an irregular sacculated cavity of considerable size. The process is sometimes described as continuing until the whole or greater part of a lobe has been excavated.

But it is evident that to describe this as a bronchiectatic cavity is a misnomer.

The trabeculae, characteristic of such cavities, are of course the remains of the bronchi and vessels. They have been stated to form prominent ridges in bronchiectatic cavities, and to be short and stumpy in those of tubercular origin, but this distinction does not hold good.

It is these larger chronic trabecular cavities which figure most frequently in recent literature as bronchiectasis, and except for this form, which ought to be called by some other name, and which cannot be well dealt with apart from the other chronic cavities, bronchiectasis, though of pathological interest, has but little clinical importance.

The possible **causes of bronchiectasis** are clearly three:—

1. Distension of the tubes from within.
2. Diminished resistance in the walls.
3. Traction upon the tubes from without.

1. *Distension from within* might result either from violent expiratory efforts, such as prolonged coughing, or from the pressure of pent-up secretion. Accordingly we find bronchiectasis occurring in the course of whooping cough and bronchitis, especially in children, or associated with stenosis, and also with a foreign body in the bronchus.

It is very unlikely that a healthy bronchus can be dilated by simple expiratory pressure, and if distension were the prime cause, the dilatation should be uniform and widespread, instead of being localised, as it usually is. Even in extreme obstruction of a bronchial tube, dilatation may be absent, or when present may occur on the proximal as well as on the distal side of the obstruction.

It is clear, therefore, that some other, and that a local cause, must be sought for. This is found in the changes present in the walls and in the adjacent lung tissue.

2. *Weakness of the walls.*—The widespread and profound changes produced in the walls by bronchitis, acute as well as chronic, are sufficient explanation of the frequency with which bronchiectasis is associated with that affection. In acute bronchitis the change consists in an inflammatory infiltration, but in chronic partakes more of the nature of atrophy, the muscular and elastic tissue being replaced partly or entirely by fibrous tissue. Infiltration of the walls by tubercular, gummatous, or malignant growth may lead to the same result, and this result is still more likely to follow when actual ulceration from any of these causes has taken place.

It is possible, as Grainger Stewart suggested, that, without any actual anatomical change, the nutrition of the muscular and elastic tissues might be so far impaired as to permit of dilatation. This is the explanation given of bronchiectasis in some of the acute fevers, *e.g.*, *typhoid*, but as there is usually at the same time bronchitis enough to account for it, it seems unnecessary to fall back on a theory which does not admit of demonstration.

In all the acute cases where the cause is removable, the dilatation may rapidly disappear and recovery be complete.

3. *Traction from without.*—The great majority of cases of chronic bronchiectasis are found associated with chronic, indurative or cirrhotic changes in the lungs.

As the pathological fibrous tissue contracts, the effects will depend upon the direction in which the contraction takes place; if this be round a bronchus the result will be the constriction or obliteration of the tube; if between two bronchi, or between a bronchus and the pleura or root of the lung, the tube will be stretched out and dilated. In most cases of fibroid induration of the lung the pleural cavity is obliterated, as would be expected, but I do not think that it necessarily must be, as Hamilton asserts.

The fibrotic changes may start from the pleura, and the cases are not uncommon in which bronchiectasis is found in the collapsed and compressed portions of the lung corresponding with chronic thickening of the pleura.

But bronchiectasis is sometimes met with in the more recent collapse accompanying bronchitis and broncho-pneumonia. Here the dilated tubes are not in the collapsed lung but in parts adjacent. The mechanism is probably the same as that by which collateral emphysema is produced, aided by the changes in the walls consequent on the bronchitis. The dilatation in these cases is often referred to increased inspiratory pressure within the affected tubes, but it would often be more correctly regarded as resulting from abnormal traction from without, owing to the defective expansion of the collapsed lung.

Secretion.—This is mucopurulent in character, but varies greatly in amount according to the size and kind of cavity present. Even with numerous and fairly large cavities it may be scanty, but it is often very profuse, and in the trabecular form may be expectorated in gushes. As in other chronic cavities, it may, at any time, undergo fetid decomposition, but this is an accidental change and by no means pathognomonic of bronchiectasis.

The Seat.—Bronchiectasis involves most frequently the medium and small, and only rarely the large, bronchi. It occurs with equal frequency in either lung, and almost as commonly in both, though not to an equal degree. It may be found in any part of the lung, but its seat is largely determined by its cause; for instance, after bronchitis and broncho-pneumonia it is most common in the base, but in the course of phthisis in the apex, and though after pleurisy it may occur in any part, it is also most frequent in the lower lobes, because chronic pleurisy is most common there. If both lungs are affected, both bases are more likely to be involved than both apices.

TABLE showing seat of bronchiectasis (from Ziemmsen).

	Trojanowsky.	Lebert.
One lung,	39	28
Both lungs,	35	26
One lung, upper parts,	14	6
lower parts,	12	15
whole,	15
Both lungs, upper parts,	4	...
lower parts,	18	...

Sex.—There is no difference in liability between the two sexes, as Biermer showed, though it had been previously stated that men were more liable than women in the proportion of 2 or 3 to 1.

Age.—The affection is spread fairly uniformly over the different periods of life, but the acute forms are most common in the young and the chronic in the adult.

Lebert's statistics are as follows:—

Under 10	10-20	20-30	30-40	40-50	50-60	60-70
6	15	20	12	18	11	7

Biermer also found that 10 per cent. ($\frac{10}{100}$) of his cases occurred in children under 10 years of age.

Results.—The acute bronchiectases which occur in the bronchitis of children in all probability get quite well, as do also those bronchiectases in adults in which there is no permanent change in the bronchial walls. Some of the saccular forms may contract, and if very small, may perhaps become obliterated in whole or in part; or the contents may inspissate and the walls calcify, and thus, it is stated, a chronic bronchiectasis may closely resemble the so-called healed tubercle. Calcification is very rare, but instances are recorded by Rokitansky and Schroeder.

Inflammation not unfrequently arises in the walls, either excited by fetid decomposition in the secretion or by other causes, which may spread through them to the lung tissue adjacent.

In this way extensive pneumonia may develop and end in abscess or gangrene, or, if not fatal as it usually is, may lead to a very rapid extension of the cavity or sometimes to pneumothorax. Aspiration of the fetid secretions into the air-tubes may also give rise to patches of broncho-pneumonia, which in like manner may end in abscess or gangrene, and it is in this way that many patients die. Yet on the other hand it is remarkable how tolerant the lung becomes, and how long patients may continue to expectorate even the most fetid secretion without inflammatory lesions developing in other parts of the lung.

It has been stated that if the ulceration be tubercular, the resulting inflammation will spread rapidly, and if simple, slowly, but this statement is not correct.

The development and extension of the cavity is sometimes slow and insidious and unattended by symptoms, so that many of the large trabecular cavities described as bronchiectatic yield no history which will account for their origin.

Bronchiectasis is not uncommon in phthisis, especially in the chronic forms with much fibroid induration, but it may be that a bronchiectasis not originally tubercular may have become so later. This has been denied, and many of the old writers believed that there was an antagonism between tubercle and bronchiectasis. This opinion rested upon an erroneous view of the tubercular process, for caseous changes are frequently described but held to be distinct from tubercle.

Hæmoptysis is not uncommon in a slight degree, but it may be profuse and even fatal. It is then due to the common causes, viz., erosion of a vessel or the rupture of a pulmonary aneurysm.

As occasional complications, abscesses in the brain and suppurative meningitis have been recorded. These lesions are usually attributed to septicæmia, and are probably in most cases due to septic embolism, for they are most frequent where the contents of the cavity are fetid.

In the same way the subacute joint affections, rheumatic as they are called, which sometimes complicate these fetid cases, are also to be explained as the result of septic absorption.

The various other lesions described as found in fatal cases of bronchiectasis, such as fatty heart, fatty liver, amyloid disease, etc., are not peculiar to bronchiectasis, but are simply such lesions as occur in the cachexia consequent on any long-standing disease of the lung.

DIAGNOSIS AND SYMPTOMS.—For the diagnosis of bronchiectasis we require (1) evidence of the existence of a cavity, and (2) reasons for believing that this cavity is a dilated bronchus. The first may be matter of fact, the second of opinion, and thus the diagnosis of bronchiectasis must in the nature of things be nearly always uncertain and often impossible.

Cavities may exist and be of considerable size, and even placed in the midst of diseased lung, and yet yield no characteristic signs of their presence. This is still more frequent when they are small and the lung around them not grossly affected, and it is under these conditions that the cylindrical and saccular forms of bronchiectasis are commonly met with. The chief successes in diagnosis are made in children with chronic pneumonia at the base in which the signs of excavation have slowly developed, but even then the diagnosis is made rather

from general pathological experience than from the physical signs or clinical evidence in any particular case.

In the large trabecular form the signs are those of a chronic cavity, and chief among them is the expectoration, often in gushes at irregular intervals, of a large amount of secretion, the physical signs varying from time to time according as the cavity is more or less free of secretion.

The bronchiectatic origin of the cavity is a matter of opinion only, and it is difficult, as it so often is, after death, even with the help of the microscope, to decide the question, it must be frequently impossible during life.

Stokes laid down the rule that when the signs of cavity had existed long without change, and without the development of phthisis, especially if the cavity were in the base of the lung, the diagnosis of bronchiectasis was justified. It is at any rate upon these data that the diagnosis is usually made, but as no account is taken of the fact that similar cavities may develop as the result of suppuration or gangrene after pneumonia or in the course of chronic tuberculosis, the data are obviously insufficient. It is always misleading and often incorrect to speak of such cavities as bronchiectatic, and it would be more accurate to describe them by the term chronic excavation of the lung, which in fact involves no theory.

If inflammation occur, and be acute and progressive, the diagnosis will be difficult from pneumonia or phthisis; if there be much purulent secretion, from empyema or from other chronic cavities in the lung; and if the secretion be fetid, from putrid bronchitis, gangrene of the lung, or fetid empyema.

In their clinical history bronchiectatic cavities do not differ from other chronic cavities, so that the consideration of the symptoms, complications, and results of bronchiectasis will be most conveniently deferred until the general subject of chronic excavation of the lung is dealt with.

The **PROGNOSIS** depends greatly upon the complications which arise. A bronchiectasis may last for many years without change, and, indeed, even without symptoms; complete recovery, however, except in the acute or slighter forms, is impossible.

The chief dangers arise from the occurrence of subacute or chronic changes in the walls, or around them; from the putrescent decomposition of the secretion; or from hæmoptysis. When death occurs, it may be from any of these causes, from phthisis, or general septic infection.

TREATMENT.—The treatment will be that of the morbid conditions with which the bronchiectasis is associated and of the symptoms which arise in its course. It will be fully discussed when the general subject of chronic excavation of the lung is dealt with, and does not require special consideration in this place.

29. PNEUMO-KONIOSIS—INHALATION DISEASES OF THE LUNG.

These are the affections of the lung produced by the inhalation of dusts.

History.—The liability of the workers in certain trades to chest diseases was familiar to the writers of last century. Wepler¹ in 1729, and Le Blanc¹ in 1775, drew attention to the great mortality among the makers of French millstones, and Johnstone in this country to that among the needle-pointers of Worcestershire.

¹ Peacock, *Brit. and For. M. Chir. Rev.*, 1860.

Pearson¹ was the first, in 1813, to suggest that the dust particles gained access to the lung tissue, and he attributed the dark pigmentation often met with in the lung to the actual deposit in it of particles of carbon or soot derived from the air. This view was generally accepted from this time, and was adopted by Laennec and Andral. Gregory,² in 1831, was the first to describe a case of what was subsequently called miners' phthisis, and to connect the development of cavities and destructive changes in the lung with the coal dust inhaled, a view which was supported by the result of the chemical examination of the lungs by Christison. Similar observations followed, and, in 1837, Stratton³ suggested the name Anthracosis for this group of affections. So the subject remained until fresh interest was aroused in it by Virchow, who denied that the dark pigment of the lung was due to carbon inhaled, and attributed it to organic pigment derived from the blood. Active discussion of the question on both sides followed, but without deciding it, until Traube⁴ discovered in the lung of a charcoal burner the angular particles of the charcoal dust, thus proving that even coarse particles could penetrate into the lung tissue. This was followed by Zenker's⁵ classical case, in which iron dust was demonstrated by chemical tests, and the subject entered upon the experimental stage. Lewin⁶ showed that an iron solution in the form of spray could be carried by the breath into the alveoli and there detected by the ordinary tests, and Kauff⁷ showed the same for solid particles by causing dogs to breathe air laden with soot. Grawitz⁸ has shown that inspired dust may even travel through the lung into the pleura and thus reach the parietal pleura.

By these and other observations the penetration of the particles into the lungs was clearly shown, and Virchow,⁹ in 1866, himself acknowledged the proof complete.

The minute histology and the pathological sequence of events in the lung were soon exhaustively worked out by Zenker and others abroad, and by Peacock and Greenhow¹⁰ in this country, and the close relation between the inhalation of dusts and affections of the lung was clearly recognised. Pneumono-koniosis was the name by which Zenker proposed to designate the group, a convenient general term which has been adopted on the Continent but has not yet acclimatised itself in England.

The further question still remained open, whether in the cases in which destructive lesions of the lung followed the inhalation of dusts, the lesions were of a simple or of a tubercular character. The presence of the tubercle bacillus now proves the majority at any rate to be tubercular, so that what has been called miners' and grinders' phthisis can no longer be regarded as special forms of phthisis, but as ordinary phthisis occurring in a miner or a grinder.

The fact having been conclusively established that the prolonged inhalation of dusts could lead to grave diseases of the lung, the subject became of direct practical importance. Investigations followed one another in rapid succession into the causes of mortality in different dusty trades and occupations, and into the means of prevention, and dust diseases now form an important chapter in all treatises on Public Health and Preventive Medicine.

Morbid Anatomy.—The general pathological changes which result in the lung are of the same nature whatever the dust inhaled may be, but the different dusts vary greatly in their tendency to produce disease, and in the intensity of the changes excited, soot or carbon being almost inert, but iron and silica actively mischievous.

Dust deposits itself largely in the mouth, pharynx, and upper air-passages. What reaches the bronchi is for the most part taken up by the cells or entangled in the mucus, and is ultimately got rid of by expectoration. In time some may reach the alveoli, and be deposited on the walls, where it acts as a mild irritant, so that the vesicles come to contain a few swollen and detached epithelial cells with some small white cells, both containing the dust and forming the so-called dust-cells (*Staubzellen*). Many of these cells are also coughed up and got rid of.

Some of the dust particles pass through the alveolar walls and reach the lymphatics, where part is taken up by the lymph cells and by large branched cells, but part remains free. They are then carried in the course of the lymph stream towards the root of the lung, and are deposited in the lymphatic glands,

¹ *Phil. Trans.*, 1813.

² *Ibid.*, xlix.

³ *D. Arch. f. klin. Med.*, xiii. 63.

⁴ *Virch. Arch.*, xxxix.

⁵ *Op. cit.*, xxxv. 178.

⁶ *Ed. Med. and Surg. Journ.*, xxxvi. 389.

⁷ *D. Klin.*, 1860, No. 49.

⁸ *Beitr. z. Inhal. Ther.*, Berlin, 1863, 114.

⁹ *Berl. klin. Woch.*, 1897, No. 29.

¹⁰ *Path. Soc. Trans.*, xvi., xvii., xx. and xxi.

which become enlarged and pigmented. In the lung the pigment is most abundant in the perivascular rather than the peribronchial lymphatics.

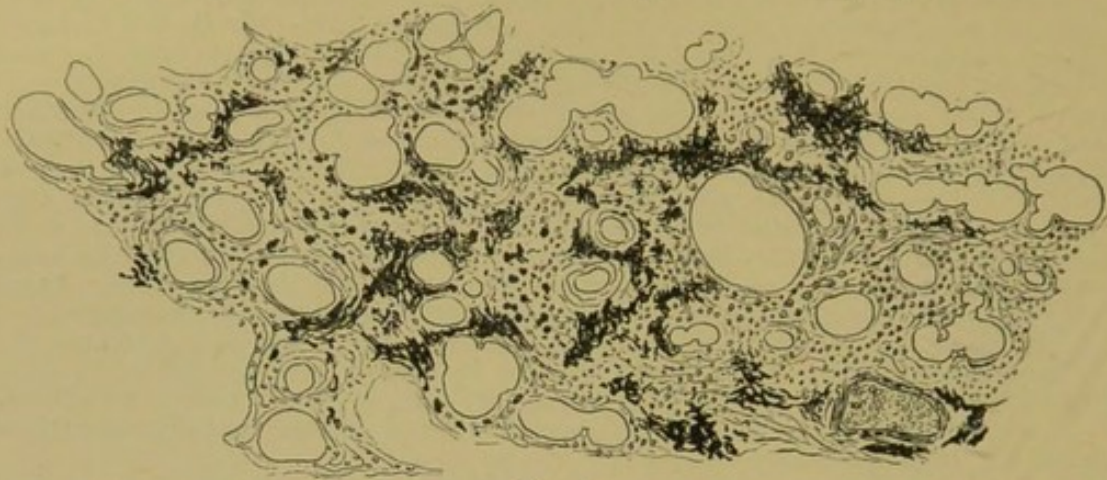


Fig. 43.

Section of a coal miner's lung, showing (1) the deposit of coal-dust round the bronchi and blood-vessels as well as on the walls of the vesicles; (2) general and considerable thickening of the connective tissue of the lung, due to the formation of fibrous tissue.

The dust in the lymphatics acts as an irritant throughout, and excites proliferative changes, which end in the production of dense connective tissue.

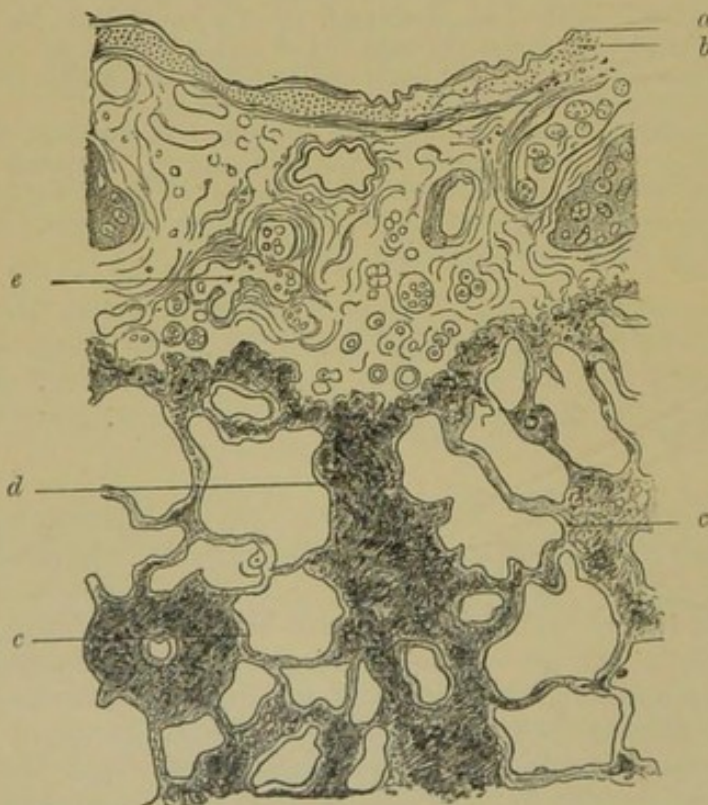


Fig. 44.

Bronchus from a coal-miner's lung (*Hamilton*). *a*, basement membrane; *b*, inner fibrous coat; *c*, pigmented nodules, surrounding arteries; *d*, pigmented lobular septum; *e*, empty mucus-gland.

In the early stage the lung presents on section numerous firm dark nodules, some minute, others as large as a pea, bean, or cherry, the parts between appearing normal.

The nodules consist of dense connective tissue, arranged in curiously concentric laminae, densest and firmest in the centre. On the outside of the nodule the vesicles are but little altered, except that they contain the pigmented dust-cells. A little deeper the alveolar walls grow fibrous and contract, the pigmented cells thus becoming enclosed in an irregular meshwork. As this meshwork contracts the cells disappear, and the pigment only remains, which in the centre of the nodule is scattered diffusely in very fine granules, and is thus less apparent there than at the periphery.

Where the nodules are discrete the alveoli between them remain unaltered. Where they are close together the intervening alveolar tissue becomes involved also, and thus larger nodules or masses are formed.

These still, however, retain the evidence of their composite formation in the numerous nodes with their concentric lamination, which gives to a section its very curious convoluted appearance. By the confluence of many such nodes larger and larger masses are formed, so that in the end the whole or greater part of a lobe may be converted into a dense fibrous mass, or several such masses may be found in the lung. Thus I have seen the whole upper lobe converted into a mass as large and hard as a cricket ball, and Ziegler gives a diagram of a lung with two masses in the centre, each as large as an orange.

Dusts are not all equally irritating. Charcoal may cause little change except pigmentation, while steel dust and especially silica is usually associated with advanced and widespread changes.

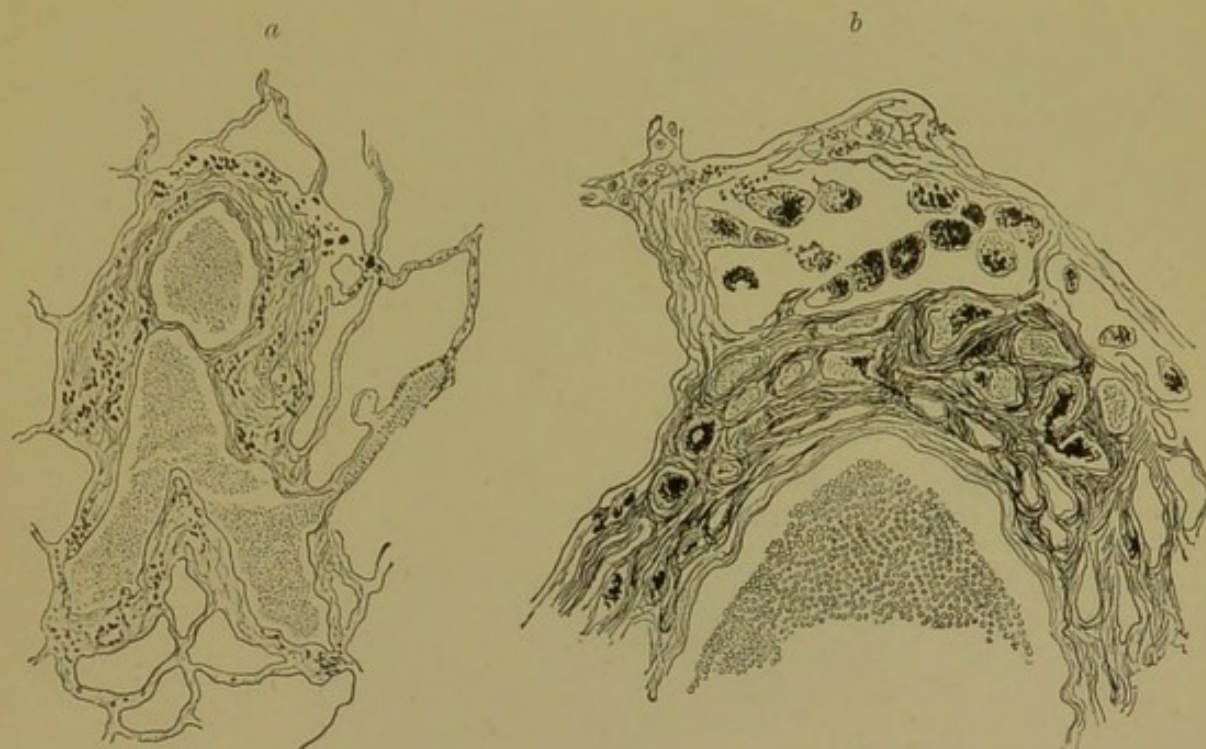


Fig. 45.

Sections of the lung from a case of chalicosis from Dr. F. W. Andrewes' collection. *a*, blood-vessels, showing the copious deposit of pigment in the lymphatics round them; the peribronchial lymphatics were almost free. *b*, portion of the same section more highly magnified, showing the pigmented cells in the alveoli at the periphery and their gradual inclusion and shrivelling in the connective tissue meshwork.

The **symptoms** are those of the different affections to which the dust inhalation has led. The earliest are those of irritation in the larynx and larger air-tubes, viz., troublesome cough with expectoration, the sputum containing the dust inhaled, both free and in cells, and being coloured by it.

The longer the irritation lasts the more difficult it will be to get rid of the dust by expectoration, in part owing to the shedding of the ciliated epithelium, and in part to the interference with the action of the ciliæ by the mucus and secretion covering them. Continued exposure to the dust leads to the symptoms of chronic bronchitis. The chronic fibroid induration in the lungs, with the emphysema which results from it as well as from the chronic bronchitis, manifests itself in shortness of breath. This, however, may not be for some time a marked symptom, until the changes become widespread or considerable, and then it may

be extreme. This is often spoken of loosely as asthma, but true spasmodic asthma is by no means common.

The **physical signs** are at first only those of chronic bronchitis and emphysema, but when induration has occurred and the lungs have shrunk, they will be those of cirrhosis of the lung. If the induration be massive, as it often is, especially at the apex, that part will be dull to percussion, and, when the tubes dilate, the signs of a cavity may be obtained. In the end, if phthisis develop, the case will present the ordinary signs and symptoms of that disease. Many affections often described as complications, *e.g.*, putrid bronchitis, gangrene, hæmoptysis, and others, are not connected with dust inhalation except as being the results of the diseases excited by it, and do not call for consideration.

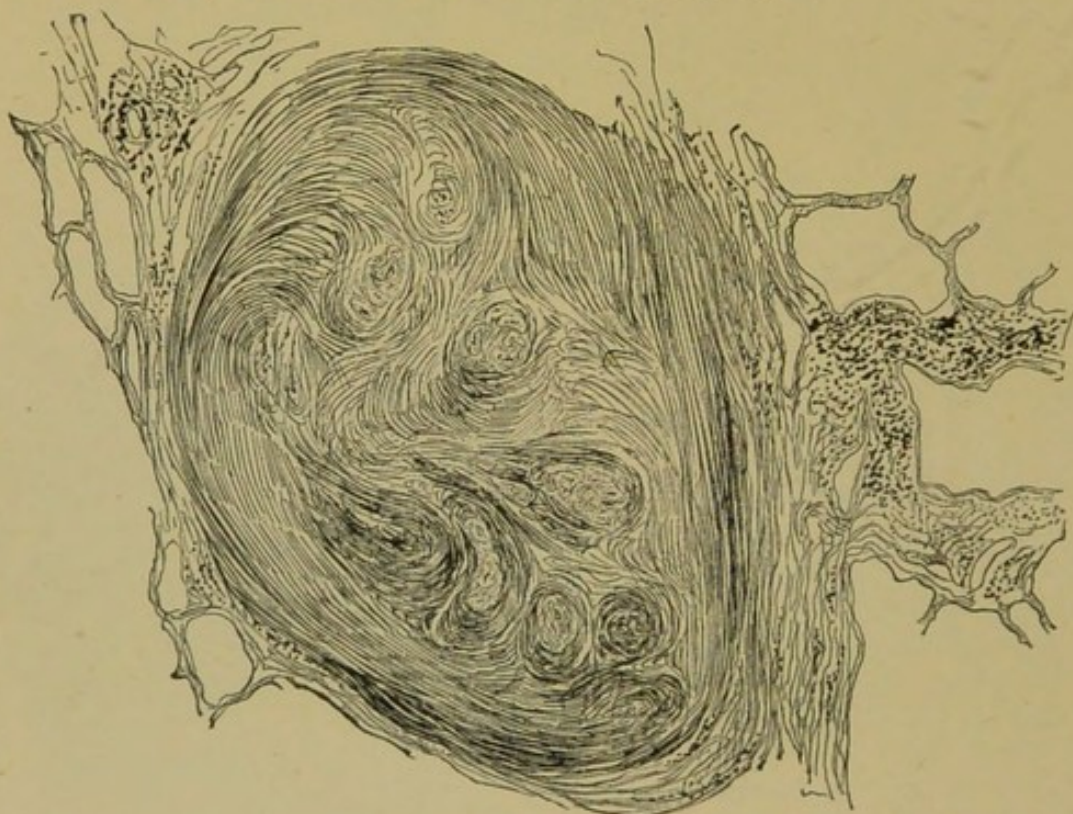


Fig. 46.

Section of a nodule from the same lung, showing the concentric lamination and convolution of the connective tissue bundles and its composite nature. The pigment at the periphery is well marked; here it is contained in cells. The gradual disappearance of the patches of pigment and the dissemination of the pigment throughout the nodule in fine granules as the central parts are approached are indicated.

The sequence of changes in the lungs are (1) chronic bronchitis; (2) chronic induration, local or general, with more or less of bronchiectasis; and (3) lastly, excavation and phthisis, each disease yielding its own signs and symptoms. Moreover, just as the victims of dust inhalation are liable to become phthisical, so are they also exposed to greater risk than others of acute pneumonia, the mortality from which affection is also in these cases much above the average.

As the children of the labouring classes often follow their parents' occupations, inherited predisposition comes into play, and when to this is added the weakness of constitution engendered by hard living and the want of sufficient food, and by the bad habits, especially in respect of drink, in which these classes so largely indulge, the high mortality and the short duration of life among them are fully explained.

The forms of Pneumono-koniosis.—The general liability to lung mischief, as well as the form which the disease is likely to take, and the course it will run, varies greatly with the nature of the dust and the amount of it inhaled. Hirt divides the dust into metallic, mineral, vegetable, animal, and mixed, and with this the pathological varieties of pneumo-koniosis fairly well correspond.

Different names have been given to the different groups, *e.g.*, *Anthracosis* to the vegetable group, *Siderosis* to the metallic, and *Aluminosis* and *Chalicosis* to the mineral. It is not, however, easy to make either classification tally with the trades or occupations of the victims. For instance, tool grinders belong to the group of metal workers, yet the dust which is most abundant in their lungs and does most harm is not the iron so much as the silica or sand derived from the grindstone. Roughly speaking, the following trades fall under each class of dust:—

Metallic.—Knife and tool grinders, polishers of metal, gravers, gilders, type-makers, glass polishers, etc.

Mineral.—Millstone makers, masons, potters, porcelain, china, and cement makers, etc.

Vegetable.—Coal miners, sweeps, charcoal burners, millers, bakers, and carriers of corn or other grain, ropemakers, jute spinners, sawyers, tobacco workers, etc.

Animal.—Workers in hair, wool, silk, or bristle, *e.g.*, weavers, carpet, brush or hat makers, dressers of cloth, sorters of wool, etc.

The dangers to health vary greatly in the different trades, and even in the same trade according to the part of the work engaged in.

Hirt gives a table showing for each group of dusts the relative liability in each to lung affections and their different forms.

TABLE showing liability to different forms of chest disease among the workers with the different dusts, calculated as a percentage, *i.e.*, for every 100 sick persons in each category.

	Metallic.	Mineral.	Vegetable.	Animal.	Mixed.	No dust.
Bronchitis, . . .	14.8	11.0	19.0	13.6	18.4	...
Emphysema, . . .	3.1	9.0	4.7	3.0	5.1	...
Pneumonia, . . .	7.4	5.9	9.4	7.7	6.0	4.6
Phthisis, . . .	28.0	25.2	13.3	20.8	22.6	11.1

What remains to complete the account given of the dust diseases will most conveniently be added when considering further the main pathological varieties which have been named.

Anthracosis.—Where the deposit consists, as it usually does, of finely divided carbon, the pigmentation is considerable, but the changes in the lung are often slight.

Carbon is the most inert of all the dusts. Even coal miners do not seem to be liable to phthisis in a degree much above the average, but among them the special nature of the work causes differences, for those who are engaged in blasting in the galleries, where the explosion produces dense clouds of very fine dust, are much more likely to suffer than the ordinary worker.

Anthracosis, owing to the part it played in the history of dust diseases, has acquired an importance which in a pathological sense it hardly deserves, for chalicosis and siderosis are much more active causes of disease.

Siderosis is a rare condition, and but few cases in all have been recorded, for in metal grinders' lungs the dusts are mixed, and consist more of silica or sand derived from the grindstone than of metal.

Zenker's first case occurred in a young woman, 31 years of age, who had been working for seven years in a factory, where a peculiar paper was made for the use of gold leaf makers. This

paper was prepared by rubbing into it a fine powder of oxide of iron. The patient developed phthisis and died. The lungs were found red in colour, and the secretion in the tubes as well as the lung tissue showed the presence of red granules similar to the powder employed, and this on chemical examination gave the iron reactions. Quantitative analysis of the lung made by Gorup-Besanez showed in every 1000 grammes of dried lung substance 14.5 grammes of Fe_2O_3 .

His second case was that of a man, 39 years of age, employed in polishing looking glasses for fourteen years. His lungs were found in the same state and contained abundance of iron.

The amount of iron in dried blood was determined for comparison. This amounted to 0.225 per cent., while in Zenker's first case the iron amounted to 1.415 per cent., in a later case of Merkel's to 0.827 per cent., and in other subsequent cases to smaller amounts.

The high mortality among the knife and tool grinders in the middle of last century was frequently referred to in literature.

Thus Hall¹ showed that while the average duration of life among the general population of Sheffield was 53 years, among the knife grinders it was only 32. Fox-Favell² also showed that the majority of the knife grinders died under 30. Similarly Oldendorff reported that in and around Solingen, while the general mortality was 19.6 per 1000, it was for the ordinary iron workers 22.9 and for grinders 30.4.

A report of the Amalgamated Society of Engineers, Machinists, Millwrights, Smiths, and Patternmakers for 1865 showed that out of 30,000 members 348 died in that year, and of these more than half died of some affection of the chest with an average age of only 36½ years.³

Since that time great improvements have been made in the processes of manufacture with great diminution in the mortality and in the amount of disease.

Chalicosis.—As already stated, Alison was one of the first to draw attention to the mortality among masons, who, he said in Edinburgh, rarely lived to the age of 50. The most startling statistics were published by Peacock in 1860.

In reference to the French millstone makers, he showed that all those engaged in this work suffered more or less severely, and that the majority died before 40, while of those who began to work before the age of 20 the average age at death was only 24½ years, and he demonstrated in several cases the presence of the silica dust in the lung.

Grinders' lungs, even where they also contain iron, contain more silica, and in some instances silica only. Potters also, especially those employed in the making of porcelain and china, suffer much from the siliceous dust and fine ground flint used in the manufacture.

Kussmaul⁴ made an extensive series of analyses of the ash of the lung at different ages in health and in different diseases. He found that infants' lungs contained no silica at all and children's lungs but little, but that the quantity increased as age advanced, while in quarrymen and those engaged in sandy work the amount might reach three times that of healthy persons of the same age, and that in these cases the silica might constitute 40 or 50 per cent. of the total ash.

Arlidge⁵ gives a similar analysis of the ash in a potter's lung. It contained 47.78 of silica, 18.63 of alum, and 5.35 Fe_2O_3 , the residue, 28.2, consisting of alkalies and other substances.

Pneumo-koniosis being not a disease but a cause of disease, **the course, duration, and prognosis** will vary with the nature and extent of the lesions produced, and the possibility or not of removing the sufferer from further exposure. Speaking generally, the course is slow and the duration long, but in all cases the life is materially shortened.

TREATMENT.—Where disease has been already excited, the treatment will be that in ordinary use for the affection produced, and the prevention of further mischief by the removal of the patient from the dusty work if that be possible.

¹ *Brit. Med. Journ.*, 1857.

² *Trans. Prov. Med. Surg. Assoc.*, xiv., 1846.

³ Report on Prevention of Disease, *Brit. Med. Journ.*, 1868, i. 79.

⁴ *D. Arch.*, 1867.

⁵ Cf. Milroy Lectures, *Lancet*, 1889, i. 615.

The obviously rational way of dealing with the pneumo-konioses is by prevention.

Dust should not be produced if it can be avoided. In some establishments this result has been obtained, for example, by the substitution of wet for dry grinding, to the great improvement of the health of those employed.

If dust cannot be avoided, it should be confined as far as possible to separate rooms or buildings, or kept damp.

For example, the grinding wheel might be partly or entirely enclosed, while the air, and with it the dust, might be extracted by means of a ventilating fan or other appliance. Every provision should be made for cleanliness in removing accumulations, and free ventilation. The use of a respirator of some kind or another is an obvious suggestion, and many forms have been devised, but the difficulty lies with the workpeople, who refuse to wear them. Something might be done in this direction by encouraging the wearing of a moustache.

The mortality of so many trades being due to dusts ought to be to a very great extent preventible. Much has been done already to reduce it, but much still remains to be done. The difficulties in each case can only be met by the desire to overcome them, and by application of common sense and suitable devices. Even where they seem to be insuperable the pressure of public opinion, and, if needs be, official inspection, would probably cause them quickly to vanish. At any rate the sacrifice of life in trade should not be tolerated when, as in most cases, it can be prevented, or at any rate reduced.

EMPHYSEMA.

The term emphysema as ordinarily used refers to a definite disease with characteristic symptoms and morbid anatomy. The essential pathological change consists in atrophy of the interalveolar septa, by which the neighbouring alveoli open into each other, and thus thin-walled, bladder-like bullæ containing air are produced. The necessary result of this disappearance of lung tissue is impairment of function, and this shows itself in dyspnœa and cyanosis.

Unfortunately the term emphysema is often used in a general sense to embrace all conditions in which the lungs are increased in size or the vesicles distended beyond their normal limits, but as in these cases there is no atrophy and often rather an increase than a diminution in functional power, it is clear that to describe such opposite states by the same term can only lead to confusion. Even when atrophy of the lung tissue is present, it does not follow that we have ordinary emphysema to deal with; for example, the so-called small-lunged or senile emphysema differs so markedly from the large-lunged ordinary emphysema as to constitute clinically an entirely different affection.

Lastly, the term interstitial emphysema is applied to a condition which has absolutely nothing to do with any of the affections previously mentioned, for the air is here in an utterly abnormal place, viz., in the interstitial tissue of the lung.

Thus in treating of emphysema it is necessary to make a sharp distinction between the different affections which have been described by the same name, but which are, for the most part, distinct and independent diseases. The want of this distinction has introduced great confusion into the statistics as well as into the theory of the disease, for it is often assumed that over-distension of the air vesicles not only may, but commonly does, lead to atrophic emphysema, though this has not yet been proved, nor is it, I believe, true.

After describing ordinary, true emphysema, I shall go on to describe the different affections separately, and shall discuss incidentally the relation, if any, which appears to exist between them.

30.—ORDINARY, GENUINE, TRUE, LARGE-LUNGED EMPHYSEMA.

True emphysema is not a condition of simple over-distension of the lung. The size of the lung is, it is true, increased, but in spite of this the weight is diminished, it may be even to the extent of one-third, and this loss of weight is due to an actual atrophy of the lung tissue. The term hypertrophous or large-lunged, as applied to this form of emphysema, is misleading, for though the latter is correct as referring to the size, the former is misleading, for the essence of the change is an atrophy and not a hypertrophy.

Morbid Anatomy.—On removing the sternum of a patient dead with this form of emphysema, the lungs do not retract as usual, but may even bulge forwards. They usually reach each other in the middle line, and may cover up the heart more or less completely. They rise abnormally high above the clavicle, and descend abnormally low, so that the arch of the diaphragm is flattened.

They may overlap in the middle line even to the extent of an inch and a quarter, and may pouch the pericardium, so that the heart lies on a cushion of lung (Fagge). The diaphragm may also, it is said, be convex towards the abdomen. Such extreme conditions as these are very rare.

These changes in the lung affect the conformation of the thorax, which is greatly distended above the normal, and occupies a position of deep inspiration.

The lungs themselves are pale or gray in colour, dry and bloodless, and markedly deficient in pigment. They feel soft and downy, crepitate but little when pinched between the fingers, and may pit upon pressure. The edges are rounded, especially above and at the margins, while the sides are often marked by prominences corresponding with the intercostal spaces. The most striking change beyond the increase of size is the presence of bladders. These are found chiefly at the apex, along the inner and anterior, and sometimes the inferior edges, or near the root of the lung. The bladders may be of considerable size, even as large as an orange, and are sometimes pedunculated. Their prominence is a *post-mortem* appearance; it cannot, of course, exist during life, or with the lungs *in situ*, but is due to the retraction of the parts around after removal from the body. On section the bladders collapse at once, their walls being thin and membrane-like. For this reason the changes cannot be well studied unless the lungs have been distended and dried.

The healthy lung when dried presents a fairly uniform spongy appearance on section, for although the air vesicles are not all of exactly the same size and not all cut across exactly in their largest diameter, still there is a fair uniformity which is wanting in the emphysematous lung.

The emphysematous lung on section exhibits a number of irregular spaces of varying size, even up to that of a walnut or small orange. These spaces are formed by the gradual disappearance of the septa, and the fusion of neighbouring alveoli or infundibula. Traces of the septa may be still detected in the smaller cavities, but in the larger are all lost. What is so remarkable in the emphysematous lung over and above these cavities is the absence of induration or thickening, and the thinness of the parts of the lung that are left.

The process commences by a gradual thinning and atrophy of the alveolar septa. A small hole forms in the septum and gradually enlarges, the capillaries round it become narrowed and ultimately break, but without hæmorrhage, and thus two or more neighbouring alveoli open into each other. The same atrophy involves the walls of the infundibula, and in this way the larger bullæ are formed.

The cells lining the alveoli seem to be passive throughout, and although they may be fatty or granular, still this is a secondary and not a constant or primary change.

The atrophy is often said to be preceded by dilatation of the alveoli. This may be so, but it is incapable of proof. Even if proved, it would not follow that the atrophy was the result of the dilatation; for the dilatation would be the natural consequence of the loss of elasticity which would necessarily precede the grave anatomical change manifested in the atrophy.

These changes in emphysema are not general or uniformly distributed through the lung. On the contrary, they are always more marked in certain parts, those most frequently and most markedly affected being the apex, the anterior margins, the roots, and often also the edges of the lower lobes, and it is in these positions that the bullæ are most frequent and of the largest size.

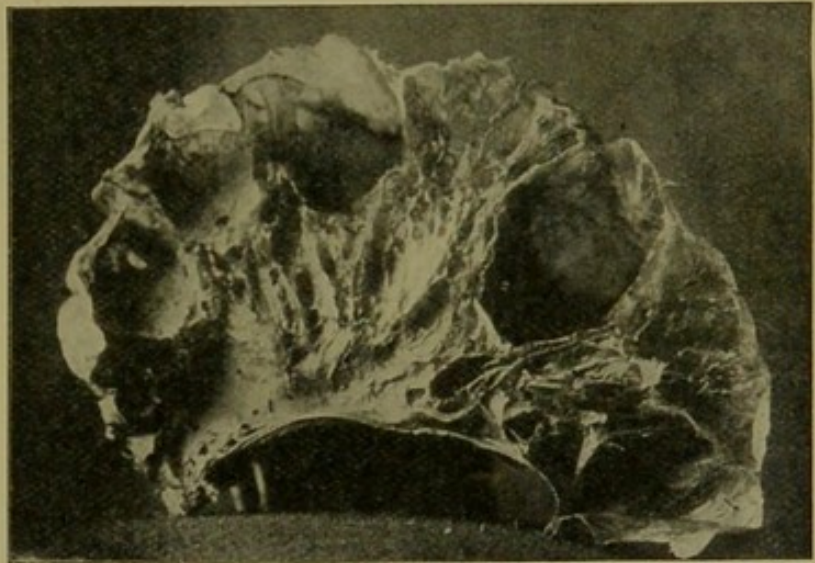


Fig. 47.

Photograph from a dried specimen of extreme emphysema (St. Barthol. Hosp. Mus., 1689) showing the big bullæ and spaces in the lung. Both lungs were in the same condition. It is difficult to understand how such extreme changes were so long compatible with life.

A case is described by Eppinger in which bladders as large as oranges were found throughout the whole lung, and another by Fraentzel,¹ where the lung formed a single large sac with dissepiments, but it may be questioned whether the last case at any rate was one of ordinary emphysema at all.

Results of the atrophic changes.—The atrophy of the septa and infundibula is attended, of course, with great destruction of the capillaries and small blood-vessels. The circulation is profoundly altered, and short circuits are formed through the anastomotic channels, which normally exist and are now greatly developed, between the pulmonary arteries and veins and the bronchial veins. With this altered circulation may be connected the congestion of the bronchi and the lesions of chronic bronchitis, which are almost invariably present in advanced emphysema. The obstruction of the circulation thus produced leads to distension of the pulmonary artery, the coats of which are usually thickened and affected with atheromatous change, while the increased tension in the pulmonary artery is met by hypertrophy of the right ventricle and auricle. As the disease progresses the time comes when the compensation of the right side

¹ *D. med. Woch.*, 1885, No. 11.

of the heart ceases to be adequate and dilatation occurs. As soon as the right side fails, venous congestion with its ordinary results develops. These are evident *post-mortem* in the distended veins, the congested liver and kidneys, and in the presence of ascites and general dropsy. The coronary circulation being affected by the venous congestion like the rest of the veins, the nutrition of the left ventricle and auricle suffers, and so the whole heart becomes dilated, often to an enormous extent. In these secondary changes there is nothing peculiar to emphysema, for they are all equally present in any other affection in which the right heart becomes unable to do the work required of it, as for instance in mitral disease.

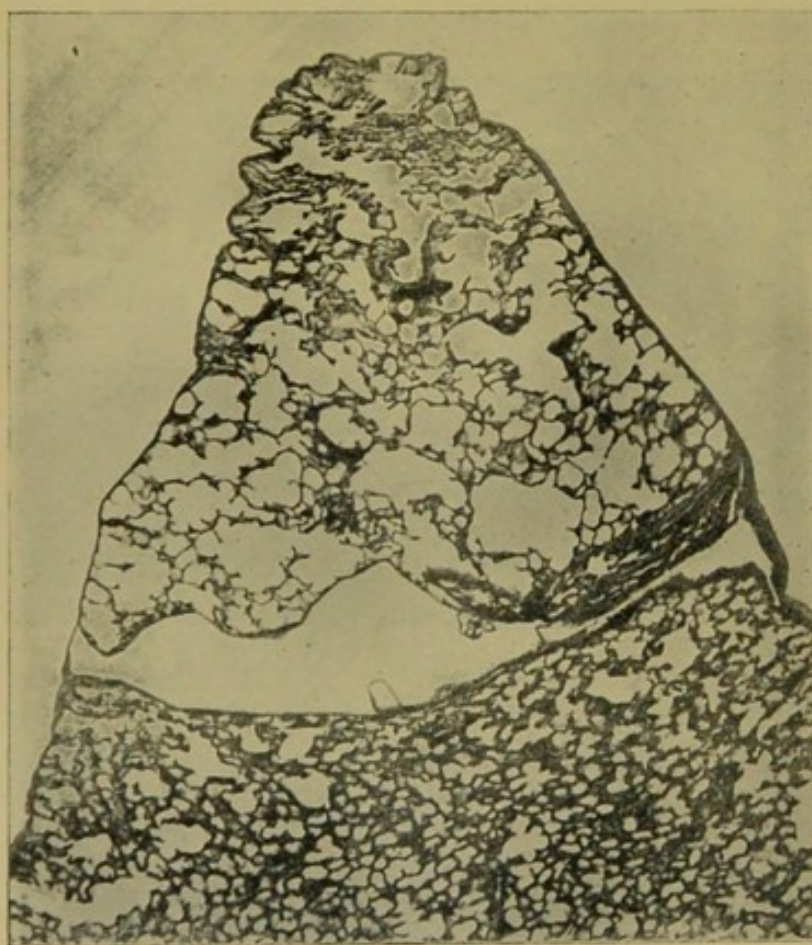


Fig. 48.

Photograph from a section of the upper lobe and part of the lower from a case of moderate emphysema. The vacuolation is most extensive in the upper lobe, but affects all parts shown in the section. Both lungs were in the same condition.

With the exception of the lesions of chronic bronchitis, which are almost constant, emphysema has no associated pathology. The pleura in most cases is affected but little, if at all. Bronchiectasis is rarely met with, and when present is to be attributed to the bronchitis.

Theories. — The vacuolation or rarefaction of the lung, as the dilatation of the air-vesicles may be called, and the atrophy of the septa being the essential conditions of emphysema, it is important to determine the relation in which these two changes stand to each other. On this question opinions have been and are greatly divided, the one school maintaining that the atrophy is the simple mechanical result of the stretching to which the alveoli are subject; the other, that

the atrophy is the primary change, and the dilatation the consequence of it. These two theories are called the *mechanical* and the *nutritive* respectively.

The **mechanical theory** implies that the dilatation of the vesicles is the necessary antecedent of atrophy and the cause of it. It assumes that the vicarious or transient emphysema and the ordinary or true emphysema are but different stages of the same process, an assumption which cannot be accepted without clear proof.

Granting for the sake of argument that over-distension of the vesicles might be the cause of atrophy of the septa, the distension required might be produced

in one of two ways, either as the result of inspiration, when some other part of the lung is prevented from expanding completely, as in vicarious or complementary emphysema, or as the consequence of forced expiratory efforts, where there is some obstruction to the exit of air from the lungs. Thus there are two mechanical theories of emphysema, the *inspiratory* and the *expiratory*.

The *inspiratory theory* has little in its favour, for vicarious emphysema is more allied to hypertrophy than to atrophy; it is attended with increase rather than with decrease of function, it may exist for long periods without the atrophy occurring which is characteristic of true emphysema, and there is no conclusive evidence at all that it does ever lead to such atrophy; indeed, all the evidence that exists is to the contrary.

In favour of the *expiratory theory* it may be urged that emphysema is frequently met with in persons suffering with long-standing cough and with chronic bronchitis, that it may often be found to follow these affections, and that it occurs in persons who are engaged in certain occupations which require forcible expiratory efforts, such as glass-blowing, and in those who are engaged constantly in public speaking and singing, or lifting heavy weights.

That all these conditions are capable of producing hyper-distension there is no doubt; the only question is as to the evidence that this hyper-distension can lead to the atrophy required. Assuming that it may, various suggestions have been made as to the way in which it acts.

(1) That the pressure within the alveoli causes the walls to burst and thus open into each other. Against such an explanation it must be urged first, that the pressure within neighbouring alveoli is the same on both sides of the septa, and if this be so, the walls might be compressed, but could not be burst, and secondly, that when rupture of air-vesicles actually does occur, the air finds its way not into neighbouring alveoli but into the interalveolar tissue, the lesion produced being not true emphysema but the interlobular or interstitial form.

(2) That the pressure squeezes the tissues in the walls, and thus causes nutritional changes in them which end in atrophy. The evidence of such nutritional change is seen according to some authorities in the cells and in others in the blood-vessels.

a. The cells have been described as undergoing fatty or granular degeneration (Rainey). This is now known to be not a constant or essential change, and the view now generally held is that the cells remain passive throughout, and that their degeneration is the result and not the cause.

b. The circulation through the small vessels and capillaries in the walls of the septa and infundibula is, it is suggested, interfered with by the compression or stretching. This theory, however, entirely lacks evidence.

As against the expiratory theory it may be fairly urged that if true emphysema were due to the increased expiratory pressure, it ought to be much more common than it is, and that account ought to be taken of those more frequent cases in which, the causes of hyper-distension being present, true emphysema does not follow; that true emphysema is known to occur, and that in early life, when none of the causes of hyper-distension can be shown to have existed; that the same person under the same conditions of life may develop true emphysema under special circumstances, as, for instance, after a severe illness, such as pneumonia or typhoid; lastly, that though bronchitis and true emphysema are very often associated, the bronchitis is in some cases known, and in others may be presumed, to be the result rather than the cause.

In the face of such objections it seems impossible to avoid coming to the conclusion that the mechanical theory is inadequate to explain the origin of true

emphysema, for the inspiratory theory does not apply at all, and that whatever influence increased expiratory pressure may have in aiding the development of true emphysema, it is not sufficient of itself; in other words, that a perfectly healthy lung cannot be made emphysematous in the true sense of the term by simple increase in expiratory pressure.

The nutritive theory.—This conclusion involves the acceptance of the nutritive theory, which maintains that the initial and essential change consists in a nutritive disturbance of the lung tissue, by which, in the first place, the elasticity is impaired, and in the second, wasting occurs. Endeavours have been made to find anatomical evidence of this change in the lung, but so far without success. The fatty and granular degeneration of the cells is, as stated, not constant. Eppinger has described changes in the elastic fibres, which end in their rupture, but this again is not constant.

It has been suggested that the atrophy is due to some interference in the circulation, as the result, for instance, of atheroma or thrombosis of the vessels, but there is no proof of this, and where such changes are known to have occurred, emphysema has not been produced.

In the absence of direct proof, the nutritive theory receives its strongest support from general considerations. All the arguments which go to show the inadequacy of the mechanical, support the nutritive theory. Beyond these it is known that emphysema may occur without any of the conditions leading to increased expiratory pressure, that many of the cases date back to early life, and that it is commoner in children than used to be thought. In this respect it is interesting to remember that Virchow¹ finds in the defective pigmentation of emphysematous lungs an argument in favour of referring the origin of the disease to early life.

The pigment of the lung, he points out, is chiefly carbon, which once deposited in the lung is never entirely removed. The absence of pigment in emphysematous lungs would imply, then, that the affected parts had never fully inspired during adult life; in other words, that they are in respect of pigment the lungs of children, and that the condition must have been present in childhood.

If the initial change may be congenital, or, at any rate, acquired early, it may also be acquired late in life, as in those interesting and important cases where emphysema has developed after an acute illness, such as pneumonia and typhoid fever.

Hertz describes the case of a cornet player who had been a healthy man until an attack of pneumonia, from which he recovered. He returned to his occupation, which, after a few months, he had to give up on account of shortness of breath. He was then found to have well-marked emphysema, which had certainly developed after the pneumonia, and presumably in consequence of it. In connection with this may be mentioned the great loss of elasticity observed by Perls in the lungs after typhoid fever.

Other writers, on account of the prevalence of emphysema in middle and late life, bring it into line with other diseases of a degenerative type, such as granular kidney and atheroma. Virchow² also draws attention to the analogy between the atrophy of emphysema and the fenestration of the omentum and other tissues, the causes of which are equally unknown. Thus the omentum may be converted into an open meshwork, and similar fenestration is seen in the falx cerebri and cerebelli, in the fringes of the cardiac valves, and also in the synovial membranes.

It is clear then that a nutritive change in the lung must be assumed as a primary and essential change in emphysema. Given this, expiratory pressure

¹ *Berl. klin. Woch.*, May 1888.

² *Loc. cit.*

would help by magnifying its effects and determining its peculiar distribution, those parts of the lungs dilating least, *e.g.*, the lower parts, which are subject to the greatest external compression by the muscles of expiration, and those dilating most which receive the least external support, *viz.*, the upper parts, the roots, and the anterior margins.

Two theories remain, but they deserve no more than passing mention, *viz.*, those of Lange and Freund. Lange referred emphysema to a neuro-muscular paralysis of the small bronchi, but if this could explain the dilatation it would not account for the atrophy. Freund attributed emphysema to a primary change in the ribs, which grew in length as well as thickness, and thus caused the expansion of the thorax. Why this theory is mentioned in every book it is hard to understand, for at the most it would account for the change in shape only, and this is neither constant nor essential.

Ætiology.—The actual frequency of emphysema is difficult to determine. Most of the statistics are unreliable, for no distinction is drawn between the various forms.

Lebert states that emphysema forms about 5 per cent. of all diseases, and as much as 12 per cent. of all affections of the lungs, while it is the cause of 2 or 3 per cent. of the total mortality from all causes. Virchow records that out of 196,458 patients admitted into the Charité at Berlin in the course of ten years, the percentage of emphysema was much smaller, *viz.*, 0·3, and of the fatal cases 0·6 or 0·7. This, too, is about the percentage of ten years at St. Bartholomew's Hospital, but of course allowance must be made for the fact that only the more severe cases are admitted into hospital.

Sex.—Roughly speaking, emphysema is from two to three times more frequent in men than in women.

3·4 to 1 (Hertz). 3 to 2 and 2 to 1 (Lebert).

This might be a natural sexual difference, but it is commonly referred to the difference in occupation and life of the two sexes, for given an equal liability to the initial change in the two sexes, the greater exposure of men to the exciting causes would be sufficient to explain the greater prevalence of emphysema in them.

Age.—Emphysema is especially a disease of middle life, or rather it becomes a marked disease at that period.

Lebert gives the following percentages :—

Age.	20 to 30	30 to 40	40 to 60	60 to 70	Above 70
	16 per cent.	28 per cent.	40 per cent.	11 per cent.	3·7 per cent.

In other words 68 per cent. of the cases occur between 30 and 60 years of age, 16 per cent. before 30, and 14·7 after 60. The last percentage is perhaps but another way of saying that emphysema is usually fatal before 60.

Before the age of 20 its frequency is represented by Lebert as only 1 per cent. Emphysema certainly occurs in the young, and of recent years the belief has gained ground that it is commoner in children than used to be supposed, and that the condition exists long before it makes itself evident by marked symptoms.

Barthez and Rilliet have watched it develop in children without asthma or bronchitis, and they believe that it is often present in those children in whom disproportionate dyspnoea is produced by trivial causes, and they are inclined to refer the emphysema of the adult very frequently to changes in the lungs which have developed in childhood.

Virchow, as already stated, owing to the absence of pigment, is inclined, on pathological grounds, to refer its development to early life.

Closely connected with this subject is the question of inheritance, which may be traced in a certain percentage of cases; according to Lebert, in as many as 12 per cent. (13 out of 108 cases).

Emphysema certainly runs sometimes in families.

Thus Schnitzler records an instance of three brothers who became emphysematous at the age of 30 without obvious cause, and Hertz another of four members of the same family.

Greenhow associated the inheritance of this affection with gout. What is transmitted is probably not the actual disease but the predisposition, so that the affection is easily excited by common causes.

Climate, occupation, and position in life are of importance only so far as they predispose to bronchitis and catarrh, which, given the initial change in the lungs, are such powerful exciting causes.

Excessive speaking and singing are often assigned as causes. There is, however, in my opinion no evidence to show that singing or speaking can produce any lesion in a healthy lung, but it is obvious that persons who have to sing or speak much will be the first to feel and to complain of the shortness of breathing which emphysema causes, and will therefore come earlier than others under medical observation. The same is true of those who have to play wind instruments, who live by glass blowing, or such occupations as involve great expiratory efforts. No doubt emphysema is met with in such cases, but account must be taken of the much greater number of those engaged in the same occupations in whom emphysema does not develop. I do not think it proved that any ordinary or extraordinary expiratory effort can produce emphysema in a healthy lung, but given the initial change, these and all other similar causes may determine or hasten its development.

Physical signs and symptoms.—Emphysema is recognised during life by the following signs:—

1. By the bilateral symmetrical increase in size of the lungs; shown by the liver standing at a lower level, by the diminution or absence of the cardiac dulness, and frequently by a characteristic change in the shape of the thorax.
2. By the defective respiratory movements, notably of expiration, and the alteration in the breathing sounds in consequence.
3. By the evidence of defective aëration of the blood made manifest by dyspnœa and cyanosis.

To these are frequently added, as an accidental but not necessary part of the disease, the signs of bronchitis.

The initial and essential change in emphysema is, as stated, the loss of elasticity and wasting of the lung, and this, long before it makes itself evident by physical signs, produces *difficulty in breathing*, at first experienced only on exertion, but as the disease progresses at rest also, and becoming in the end extreme. The diminution in the respiratory surface of the lung, with the consequent disappearance of blood vessels, leads to defective aëration of the blood, which shows itself in *cyanosis*. The obstruction to the circulation through the lungs causes the right side of the heart to hypertrophy, and subsequently to dilate. Partly as the result of its increase in size, and partly because, following the diaphragm, the heart stands lower in the thorax than normal, its movements become visible in the epigastrium and cause epigastric pulsation. In time the right side of the heart fails, and signs of venous congestion appear. The cyanosis deepens, the veins of the neck are distended and pulsate, the liver enlarges, œdema appears in the feet and slowly spreads upwards, ascites develops, and so the fluid rises in the body until it collects even in the pleura and pericardium. As these signs of cardiac failure develop the chest symptoms increase, and the cyanosis becomes extreme until the patient dies of suffocation and exhaustion.

The **symptoms** thus fall into three groups, which are sometimes spoken of as the three stages of the disease.

1. Those of respiratory difficulty.
2. Those of circulation obstruction, resulting in hypertrophy of the right side of the heart.
3. Those of defective compensation and heart failure.

The most striking feature in emphysema, especially in the later stages, is the amount of cyanosis which develops, and which is hardly met with to the same degree in any other chronic affection, unless it be in some forms of congenital morbus cordis.

Physical Signs.—In a well-marked case the patient has an anxious, careworn, suffering look, a bloated dusky face with dilated veins upon the cheeks and beneath the conjunctiva, thick lips and nostrils and prominent glassy eyes. The back is round, the shoulders high, and the neck short and thick. The chest is held stiffly and the head thrown rather forward.

The general nutrition is for some time well preserved, and the patient may even be fat. In the later stages the nutrition suffers and the patient becomes thin and emaciated, and there is then a marked contrast between the thick neck and rounded chest and the wasted legs and lower parts of the body.

According to Lebert, in not more than 30 per cent. is wasting a prominent feature of emphysema, and then only at the last.

Examination of the Chest. — *Inspection.* — The chest is generally expanded, and occupies an almost fixed position of deep inspiration, the shoulders are round, the sternum prominent, especially in the upper part, the ribs straightened and the intercostal spaces widened, the upper part of the abdomen constricted and pinched in, so as to give the appearance of a waist.

A girdle of dilated veins is often seen following the curve of the costal arch, but about an inch or so above it. It corresponds with the insertion of the diaphragm and abdominal muscles,

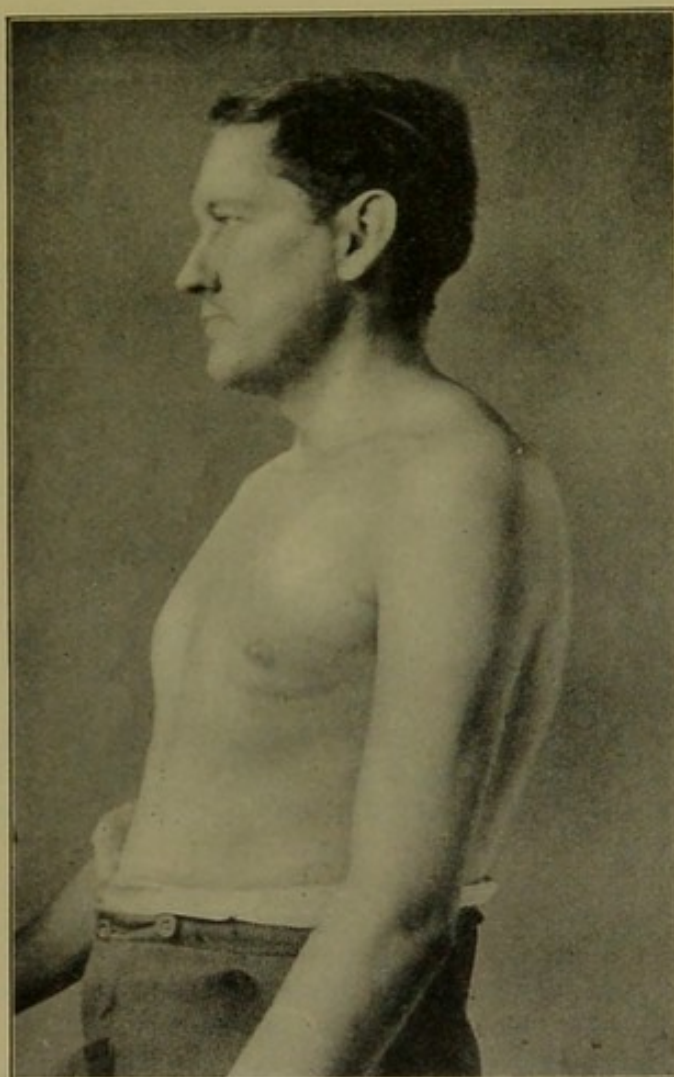


Fig. 49.

Photograph of a patient with well-marked emphysema, showing the high shoulders, rounded chest, and prominent sternum. The thorax gave an almost circular tracing, and the form of the chest was typically barrel-shaped.

and is due to their repeated contraction in coughing or straining. Accordingly, though common in emphysema, it is not peculiar to it, but may be found in most persons who have been subject to chronic cough, and even in healthy and very muscular men who have to lift heavy weights continually.

The dilatation of the thorax is chiefly in the upper parts, and especially in the antero-posterior diameter, which may be equal or nearly equal to the transverse, and thus yield an almost circular cyrtometer tracing.

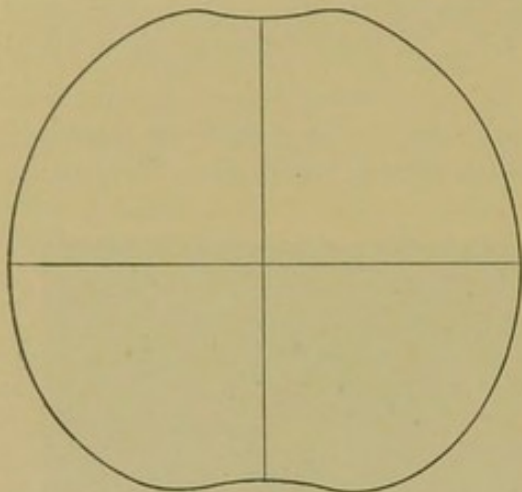


Fig. 50.

Cyrtometer tracing of barrel-shaped chest.

The shape of the chest.—The roundness of the thorax and the waist-like constriction below give to the chest the form of a barrel, so that the barrel-shaped chest has come to be regarded as characteristic of emphysema, and so it is when present, but it is often absent.

According to Lebert, in not more than 56 per cent. is the change of shape marked, and in 20 per cent. there is no obvious change at all.

Even when the chest is distinctly barrel-shaped it rarely yields, in the adult, a circular tracing, the difference between the transverse and sterno-vertebral diameters being still well marked.

The movements of the chest on respiration are very defective. The chest seems to move *en masse* and hardly to expand at all. It is expiration that is chiefly impaired owing to the loss of elasticity in the lung. To make up for this the expiratory muscles come into play, especially those which act upon the lower ribs. By the overaction of the abdominal muscles, and especially of the upper part of the transversalis abdominis which passes from one side of the costal arch to the other, there is caused the waist-like constriction described. These defective movements of expiration greatly diminish the amount of the air which passes in and out of the chest on each respiration. The inspiratory muscles attempt to remedy this by increased action, and thus the chest takes the position of deep or even deepest inspiration. To this excessive action of the inspiratory muscles, combined with the enfeeblement of their chief opponent, viz., the elastic retraction of the lung, the characteristic shape of the emphysematous thorax is to be referred. The overaction is attended with hypertrophy of the muscles concerned, especially of those at the root of the neck, viz., the scaleni, sterno-mastoid and the trapezii, and to this is due the fulness and thickness of the neck, while the trapezii and other muscles acting on the scapulæ produce the elevation and rounding of the shoulders.

The inspiratory muscles, however, do what they will, are unable to compensate completely for the deficiency of the expiratory movements, and the result is a great diminution in the tidal air even to the extent of from 20 to 60 per cent. (Wintrich). The reduction in the vital capacity is even greater still, from the normal average of 230 inches (3500 cm.) to 130 or even 65 inches (2000 or 1000 cm.). The great loss of expiratory power may be demonstrated by the pneumometer, the expiratory pressure being diminished until it is either the same as, or only slightly in excess of, the inspiratory pressure, whereas in health it is about half as great again.

In health, inspir. pressure = 70 to 80 mm. (3 in.) ;	expir. pressure = 110 to 130 mm. (4½ to 6 in.)
In emphysema, ,, ,, (,,)	= 77 to 100 mm. (3½ to 4½ in.)

The rate of respiration is usually not increased at all or not much ; it may even be reduced owing to the prolongation of expiration, but the type is changed, for the breathing is short and jerky, consisting of a short, sudden, laboured inspiration followed by an equally sudden collapse or recoil of the chest on the commencement of expiration, ending in what seems to be a pause, during which the expiration slowly completes itself.

These changes are well shown on stethographic tracings. In health inspiration is somewhat shorter than expiration, the curve is smooth and regular, save that the ascending limb corresponding with inspiration is steeper than the descending limb, which corresponds with expiration. In emphysema, on the other hand, the inspiratory limb is shorter, and so steep as to be nearly vertical, as is also the first part of the expiratory limb, and the duration of these two parts together is hardly as long as that of healthy inspiration alone. The remaining part of the expiratory limb is irregular, oscillatory and lengthened.

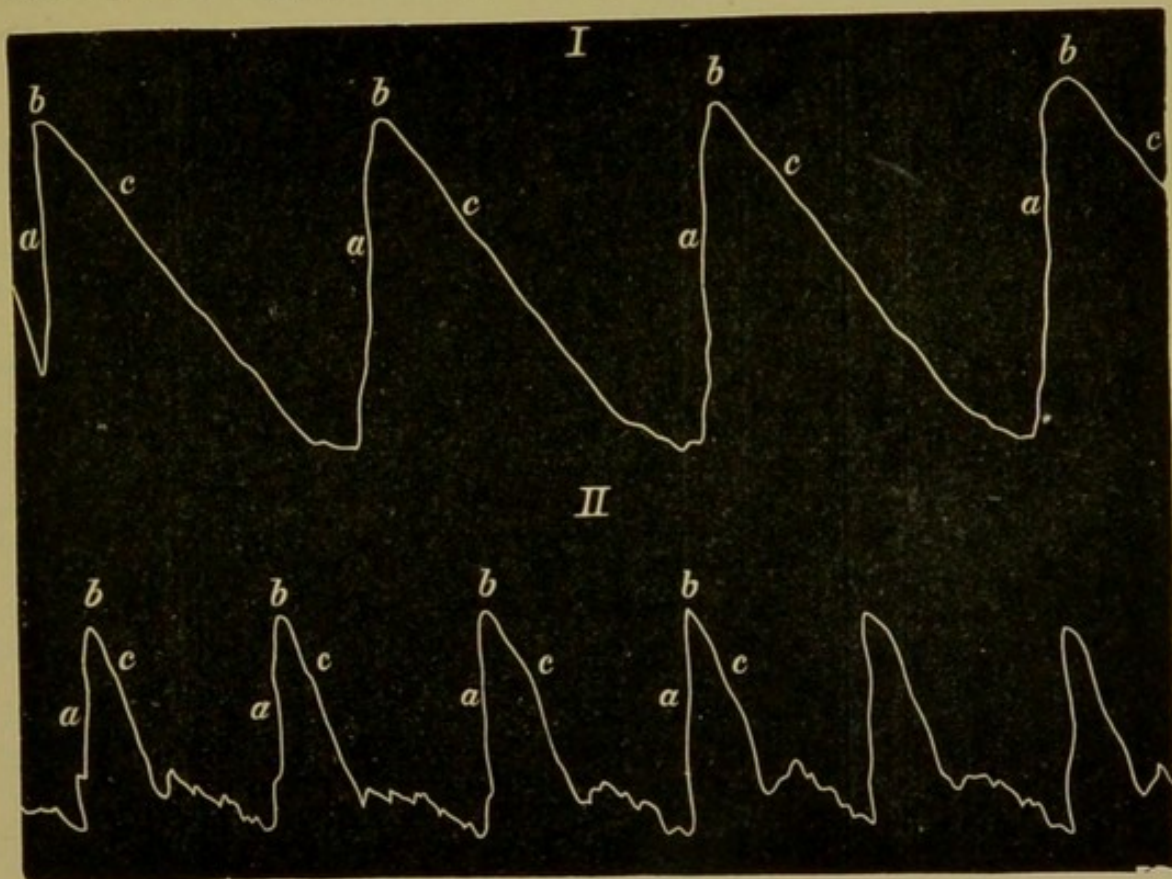


Fig. 51.

Pneumatograms. I. Normal. II. In Emphysema. *a*, inspiration ; *b*, apex of curve ; *c*, expiration. The small elevations are due to the cardiac impulse. (*Landois and Stirling.*)

The loss of elasticity in the lung affects the movements of the diaphragm as much as it does those of the ribs, for on expiration the diaphragm is not properly arched, and therefore contracts at a great disadvantage. Its defective action is probably after a time attended with an actual loss of contractile power, which may perhaps in the end amount almost to actual paralysis. At any rate the diaphragm is sometimes found completely relaxed ; it may even become, it is said, convex towards the abdomen, and as the muscle is assumed to be still active, its contraction then would make it a muscle of expiration, and produce what has been called the "typus inversus" of respiration. The inspiratory recession of the epigastrium which has been described in emphysema has been referred to this cause, but I should be more inclined to regard it as the result of paresis or paralysis of the diaphragm than to its inverted action.

The loss of elasticity and resistance in the lung shows itself also in the bulging of the supra-clavicular fossæ, and even sometimes of the intercostal spaces on expiration and their recession on inspiration.

Palpation yields but little additional information. The vocal vibrations may be diminished somewhat, but are often unchanged. The apex of the heart is generally not to be felt, but pulsation in the epigastrium and in the veins of the neck may in the later stages be felt as well as seen.

Percussion.—The most important evidence of emphysema is obtained by percussion, for by it the increase in the size of the lungs is ascertained, and that, too, when the characteristic change in the shape of the thorax is not present. The two cardinal signs are the absence of the cardiac dulness, and the displacement of the liver; to these may be added the general hyper-resonance of the whole thorax, and also, though to a less degree, of the supra-clavicular spaces.

The cardiac dulness is generally completely absent, but on deep percussion over the præcordium a difference may be sometimes detected, the note being less resonant than elsewhere, though not dull.

The diaphragm stands low, and so, of course, do the abdominal viscera in relation with it, viz., the liver on the right side and the stomach and spleen on the left. The position of the stomach and spleen in emphysema cannot usually be made out by percussion, on account of the hyper-resonance of the chest.

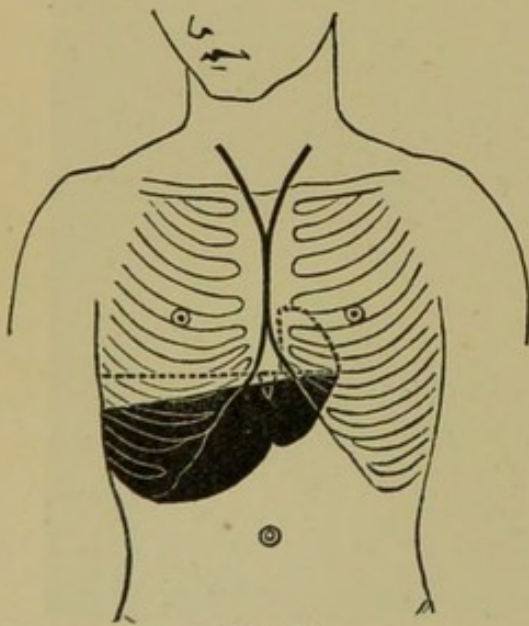


Fig. 52.

Diagram showing the percussion boundaries in a case of emphysema. The upper border of the liver stands at the level of the sixth rib on the right nipple-line—the cardiac dulness is absent.

The upper border of the liver is found in the nipple line at the 7th rib, or in the 7th space, but rarely lower than this even in the most extreme cases. This is a point which serves to distinguish emphysema from pneumothorax of the right side, a lesion which the hyper-resonance might suggest, for in pneumothorax the resonance extends as far as the costal arch.

Auscultation.—Owing to the feeble current of air in and out of the lung, the respiratory murmur is greatly diminished. Expiration is usually inaudible. At any rate it cannot be heard unless bronchitis is present. When audible it is prolonged, and may be even three or four times longer than inspiration.

A peculiar crepitation of a crackling or crumpling character, as of stiff parchment, is sometimes present, chiefly with inspiration, and especially at the apex,

about the nature of which there is much difference of opinion. It has been referred to irregular expansion of the lung, to the rubbing of the air-bladders against the costal pleura, or to actual pleurisy. It is certainly often heard when there is no pleurisy, and the first explanation seems the most probable. The "râles crepitants à grosses bullæ" of Laennec, is, I believe, due to secretion in the tubes.

Bronchitis when present, as it so often is, yields the usual physical signs in addition, viz., rhonchus, sibilus and crepitation. It, of course, greatly intensifies the symptoms.

Symptoms. — The symptoms fall conveniently into two groups, the respiratory and the circulatory.

1. *Respiratory.*—Of these shortness of breath is the earliest as well as the most constant. At first experienced only on exertion, it gradually increases throughout the disease, until at the last it becomes extreme. It varies greatly at different times, chiefly in relation with bronchitis, for a small amount of catarrh produces an entirely disproportionate amount of dyspnoea in emphysematous patients. This is especially the case in children, and may be the first indication of the existence of the disease.

The dyspnoea, though due in part to the diminished expiratory power which results from the loss of elasticity in the lung, is not due to this alone, but in great part also to the decreased aërating surface in the lung, consequent on the atrophy of the vesicular walls and the destruction of the capillaries contained in them.

The shortness of breath makes the voice weak and the speech short and jerky, for the breath cannot be held more than a few seconds at a time.

The dyspnoea is sometimes paroxysmal, and is then often called asthma. True asthma may, of course, be associated with emphysema, but in most cases the paroxysmal dyspnoea is produced by an attack of bronchitis, and by the accumulation of the secretion in the tubes.

The defective aëration of the blood shows itself in cyanosis, which is more marked in emphysema than in any other chronic affection except congenital disease of the heart. In the latter disease it is due to the actual mixing of the arterial and venous blood, either in the heart or in the great vessels near it. In emphysema also there is similar mixing of the two blood streams through the communications which exist between the pulmonary and bronchial vessels.

The cyanosis is at first most marked in the usual places, viz., the tips of the ears and nose, in the lips and beneath the finger nails. It may even be observed in the tongue. The small veins in the cheeks and on the neck and chest are dilated, varicose, and plainly visible. In the later stages the patient presents a deep purple colour, which though present over the whole body is deepest over the face, neck, and upper part of the trunk.

Like the dyspnoea the cyanosis depends not upon the condition of the lungs only, but also upon the heart, and is most marked in the later stages of the disease when the heart is failing.

As the result of venous obstruction hæmorrhages may occur, especially after violent coughing, in the skin or conjunctiva, or from the nose or bronchi. Hæmoptysis is no necessary part of emphysema, and when it happens is generally due to bronchitis. It rarely amounts to more than streaks in the sputum, unless infarct or embolism has occurred in the lung.

Fagge,¹ however, describes a case of fatal hæmoptysis in emphysema, for which no other cause could be found *post-mortem*.

Cough may be absent for some time, but in the end it is sure to develop. It varies with the bronchitis which causes it, and is, as is usual in that affection, worst at night and in the early morning.

The sputum in like manner depends upon the bronchitis, and is no part of emphysema. It has the ordinary bronchitic characters, and may now and then be blood-streaked. Owing to the defective expiratory power, the secretion in the tubes is difficult to get rid of, and the cough it excites is often paroxysmal and prolonged, and consists of a series of short expiratory jerks.

¹ *Loc. cit.*

Pain is often complained of, especially over the lower part of the chest and along the attachments of the diaphragm and abdominal muscle. It is generally the result of the muscular strains produced by the coughing.

2. *Circulatory*.—Emphysema throws a heavy tax upon the right side of the heart, inasmuch as the blood has to be driven through a greatly diminished number of small vessels in which the resistance is increased by the defective oxygenation of the blood. The circulation is still further impeded by the frequent paroxysms of coughing, and especially by the defective movements of the thorax, inspiration doing less than it should to suck the blood into the chest, and expiration less to drive it out. The right heart endeavours to compensate for these difficulties by hypertrophy, but rarely succeeds, at any rate for long, for the obstruction usually outstrips the compensation. In the end the general nutrition of the whole heart suffers, because the heart is supplied, like the rest of the body, with imperfectly oxygenated blood, and because the venous congestion, when it has once set in, affects the circulation through the coronary vessels and so leads to weakness of the whole heart, of the left side as well as of the right.

The dilatation affects the right side first and most, but in the end the left suffers also for the reasons just given, and the dilatation becomes general. The physical signs of dilatation are, however, frequently masked by the emphysema, for the apex beat may not be felt at all nor any increase of the cardiac area be made out by percussion, so that the heart is often found enormously distended after death though percussion failed to give proof of it during life.

The two most important signs of dilatation of the right side of the heart are epigastric pulsation and distension with pulsation of the veins of the neck. Epigastric pulsation is common in emphysema, even without much dilatation, owing to the diaphragm and the heart therewith standing lower in the thorax than normal, but it is not of itself conclusive, for it may be usually seen when the heart is beating violently, even in healthy persons, as after running or strong exertion.

The heart sounds are weak, especially at the apex, owing to their being conducted badly through the emphysematous lung. The pulmonary sounds are both accentuated, especially the second, which is often reduplicated. Systolic murmurs may be heard both at the apex and on the right side. They are commonly referred to regurgitation through the mitral and tricuspid valves respectively, but they are rarely propagated in the direction they should be if they were produced by incompetence of these valves; for instance, at the apex blowing murmurs are not uncommon when the heart is dilated, but they are rarely heard outside the apex, in the axilla or behind, and I therefore hesitate to refer them to regurgitation. Still regurgitation may certainly occur, though in my experience but rarely, both at the tricuspid and at the mitral orifices, but then the murmurs are propagated in the usual directions.

Out of a very considerable number of cases of dilatation of the heart which I have examined definitely for the purpose, I have only a very few times heard a murmur which I felt sure was tricuspid, and in one case the diagnosis was verified on *post-mortem* examination by finding an organic lesion there. I have seen also two cases of apex murmurs which were, like the ordinary organic mitral murmurs, propagated into the axilla and heard behind. In one, a man, the diagnosis was made on this account of mitral disease with secondary bronchitis. The bronchitis cleared up, the murmur disappeared, and the patient recovered. Twice later the same symptoms recurred, and the third time the patient died. On making the *post-mortem* examination no lesion was found in the heart except great dilatation. The second case occurred in a girl aged 12. The apex-murmur, transmitted into the axilla and behind like that of mitral disease, disappeared as the bronchitis got well, and when convalescence was established the heart seemed to be perfectly normal.

It seems thus impossible to avoid the conclusion that the murmurs associated with dilatation are, with but few exceptions, intraventricular and not due to regurgitation.

As soon as the heart has begun to fail the usual signs of venous congestion appear. The veins of the head and neck become greatly distended, the cyanosis deepens, pulsation in the epigastrium and cervical veins is more marked, and the liver becomes large and painful. As the stomach and intestines are also congested, their functions are affected, the digestion fails, and piles often develop and bleed. Soon dropsy sets in, first in the feet, then in the loins, and later in the abdomen and other serous cavities, the lungs grow more and more congested, the dyspnoea and cyanosis more extreme, and at last the patient dies of slow suffocation. As carbonic acid accumulates in the blood the patients become restless, then drowsy and wandering, and towards the end may suffer with twitchings or even fits, but more often they become unconscious or almost comatose, in which condition they die. In all this there is nothing peculiar to emphysema or different from what is met with in other cases of right-heart failure, as for example in the course of mitral disease, except so far as it is aggravated by the emphysema and bronchitis which have caused it.

Relation of emphysema to other diseases.—The list often given of diseases with which emphysema may be associated owes its length to the fact that the distinction is not clearly made between true emphysema and the other forms. When this is done the list reduces itself to a single affection, viz., bronchitis, which sooner or later is sure to develop, and the reason is to be sought in the congestion caused by the venous obstruction and by the anastomotic circulation set up. Given the primary condition of the lung, which predisposes to emphysema, bronchitis may become the determining cause of its development by the coughing to which it leads. Under all circumstances bronchitis aggravates the symptoms, and, when the secretion accumulates in the tubes, may cause those paroxysmal attacks of dyspnoea which are incorrectly described as asthma.

True spasmodic asthma occurs, of course, often in association with true emphysema, and in long-standing cases leads to it; but when asthma is spoken of as causing emphysema it is often the transient hyper-distension of the lung which accompanies the attack that is referred to rather than true emphysema.

A natural antagonism has been supposed to exist between emphysema and certain diseases, notably phthisis, croupous pneumonia, and morbus cordis. In all these conditions transient emphysema is common, but true emphysema is certainly rare.

Phthisis.—An emphysematous lung may become the seat of tuberculosis, either acute or chronic. Speaking generally, it is true that phthisis and emphysema are not commonly associated; but in estimating the real value of this fact allowance must be made for the different ages at which the two diseases are prone to develop, phthisis being especially an affection of early adult life and emphysema of middle age. It has been also stated that the development of emphysema may check the progress of tubercle. The fact is that when tubercle becomes stationary, fibroid contraction occurs, and the remaining parts of the lung undergo vicarious distension, but this distension takes place because the phthisis has become stationary—the phthisis does not become stationary because the distension has occurred. In any case it is vicarious emphysema and not the true emphysema which is found.

Croupous pneumonia at one time was said not to occur in emphysematous lungs, but it certainly does, and Lebert's figures show that it is the actual cause of death in 5 per cent. of the fatal cases. The two affections are, however, so

common that they would now and then in the ordinary course of events be accidentally associated, but there is no means of judging whether this occurs more or less frequently than it naturally should.

I think it may truly be said that emphysema does not increase the liability to croupous pneumonia, and that it cannot be proved to diminish it.

Morbus cordis.—The same is true of morbus cordis, viz., that it does not in any way affect the liability to emphysema, but the question may be fairly raised whether emphysema can be the cause of morbus cordis. In one sense, of course, it can and often is, for emphysema naturally ends in cardiac failure, and this may end in valvular incompetence, though not with the frequency that is often stated. If it be true that emphysema tends to cause atheroma of the vessels, the valves may become involved also, but except in this remote and indirect way emphysema does not appear to lead to organic disease of the heart.

Diagnosis.—The combination of the three cardinal signs—dyspnoea, bilateral increase in size of the lungs, and cyanosis—is sufficient to distinguish emphysema from all other diseases.

Of these the increase in size of the lungs is the most constant and reliable. The slighter degrees of dyspnoea and cyanosis may have many other causes, but in the later stages the dyspnoea is fairly characteristic, and the cyanosis is too deep to be confused with any other affection except that due to congenital morbus cordis.

The only other condition in which bilateral increase in size of the lungs is met with is in athletes, especially during training; but the free respiratory movements and the increase rather than diminution in functional power prove this condition to be a hypertrophy and not an atrophy.

Dyspnoea, and cyanosis too, might be associated with temporary distension of the lung, as, for instance, in asthma or acute bronchitis, but the course of the case would make its nature clear.

True emphysema affecting one lung only is unknown. If it were not so the hyper-resonance and distension of the side might suggest pneumothorax; but then the diagnosis would be given by the extension of the resonance to the costal arch and the displacement of the heart to the opposite side.

Forms of Emphysema.—Four kinds of emphysema are sometimes described. They would be better called four clinical stages of the disease.

1. Emphysema without complications and with little dyspnoea or distress, *i.e.*, early or slight emphysema.
2. Emphysema with transient attacks of severe dyspnoea and cyanosis, *i.e.*, in most cases emphysema complicated with intercurrent bronchitis.
3. Emphysema with constant dyspnoea and cyanosis, *i.e.*, advanced emphysema.
4. Emphysema with cardiac failure.

Prognosis.—True emphysema is an incurable disease, for no treatment can bring back the lung tissue which has atrophied. The disease may probably become stationary, and that too for long periods of time, but, as a rule, it steadily progresses, and though the rapidity with which the different stages follow one another varies much in different cases, still on the whole the affection is of long duration.

The prognosis therefore as regards complete cure is hopeless, and, as regards duration of life, varies, but as a rule the life is shortened by many years, for patients with emphysema rarely live to old age. The apparent prevalence of emphysema between the ages of 30 to 50 shown by statistics, chiefly drawn from hospital practice, is explained by the fact that the symptoms become marked in

middle life, and its less frequency after 60 by the fact that in most cases death occurs before that age.

The prognosis in respect of danger to life at any given time turns upon the stage of the disease, the amount of bronchitis and cyanosis present, the age, strength, and general nutrition of the patient, and especially upon the condition of the right side of the heart. There is no danger in the slighter degrees of emphysema, and the prognosis becomes that of the catarrh upon which the aggravation of symptoms depends. But where the circumstances are such that the recurring catarrhs cannot be properly treated or taken care of, the disease will become rapidly aggravated, and in this regard the position and habits of life become of prognostic importance. The older, feebler, and weaker the patient, the greater the amount of bronchitis and cyanosis, and above all things the more marked the cardiac embarrassment, the greater the risk at the time. Still, threatening as a case may seem, relief is often obtained by appropriate treatment, the alarming symptoms subside, and the patient returns to his condition prior to the exacerbation.

Of course in emphysema, as in other diseases, the occurrence of acute complications, for example pneumonia, or the existence of other general or local diseases, such as chronic pleurisy, chronic kidney mischief, cirrhosis, etc., greatly aggravate the danger.

Death is rarely sudden or without warning, and then is not due to the emphysema alone but to some complication. In most cases death is gradual, either as the result of bronchitis or of the failure of the right heart.

According to Lebert's statistics one-third of the fatal cases die with cardiac dropsy, and of the rest the majority die of pulmonary congestion and slow suffocation with the signs of acute or chronic bronchitis.

Treatment.—*Prophylactic.*—The primary and essential change in emphysema is the wasting of the lung, but the causes of this are not known and cannot therefore be counteracted, nor can the affection be recognised with certainty until it is far advanced.

Certain diseases and certain occupations, though not of themselves alone adequate to produce the disease, aggravate it when present, and must therefore be guarded against. For this reason emphysematous persons must be advised to give up all occupations which throw any unusual strain upon the lungs, such as those which involve violent muscular efforts, the playing of wind instruments, glass-blowing, etc. It will be wise also to forbid violent muscular efforts and occupations such as those described to all, even previously healthy patients, while recovering from acute pulmonary affections, as, for instance, from pneumonia or acute bronchitis, at any rate until convalescence is fully established, for instances are recorded in which true emphysema has developed rapidly under these circumstances.

It is against bronchitis that precautions are especially necessary, for this not only intensifies the symptoms but aggravates the disease. For this reason those suffering from emphysema must be greatly on their guard against catarrh; they must avoid all causes likely to excite it, and take the greatest care when it arises even in its slightest forms. They should be warmly clad, avoid all chills, and stay within doors in bad weather.

With this object, residence during the winter months either in the warmer winter resorts of England or in a good climate abroad is highly desirable.

The general health should be maintained at the highest level possible by means of well-regulated diet, exercise, and tonics, and this becomes of especial importance in the more advanced cases, when the disease is beginning to tell upon the heart.

In other respects the treatment of emphysema is symptomatic. The two prominent symptoms are dyspnœa and cyanosis, and these depend first upon the defective movements of the chest, and secondly upon the defective aëration of the blood.

The defective movements are chiefly expiratory, and they might be aided either by external compression of the thorax or by diminishing the resistance against which they have to act.

Many patients, during the exacerbations of dyspnœa which they suffer from, find relief by compressing with their hands the sides of the chest as they expire, and for this an elastic band some inches wide embracing the lower ribs may be substituted. For the upper parts of the thorax, which are those most affected in emphysema, a more elaborate apparatus has been constructed, but it is cumbersome and inconvenient. It consists of a double truss fixed on the spine behind, with the pads on the second spaces in front (Ferin).¹ Steinkoff's² apparatus is more elaborate still.

The only means of diminishing the resistance to expiration is by causing the patient to breathe into rarefied air, and for this purpose Waldenburg's apparatus is most used.

This consists of a small gasometer with weights and counterpoises by which the air it contains can be placed under varying pressures.³ It is fitted with a tube and mouth-piece containing valves, by which communication with the interior either during inspiration, or during expiration, can be made as desired. Another apparatus is like an accordion, which is expanded or compressed by the patient himself.

It is recommended that the rarefaction should not exceed $\frac{1}{30}$ inch of an atmosphere, and that the first administration should not exceed ten minutes in duration, but afterwards extend to half-an-hour two or three times a day, if found beneficial.

Expiration into rarefied air is said to give relief, not only during its application but for some little time afterwards. The cases of cure which have been recorded were obviously not instances of true emphysema.

Waldenburg also stated that even the inspiration of compressed air was useful, and he explained its usefulness on the ground that more oxygen was introduced into the lungs. Others do not confirm this, and it would seem *a priori* to be directly contra-indicated.

Patients have also been placed in the pneumatic cabinet under compressed air, but this has been generally found to be harmful.

On the whole the pneumatic treatment of emphysema is of little, if any, use in severe cases, and cumbersome and awkward to carry out in the slighter cases, so that it has fallen out of use.

The defective aëration of the blood might be met by the inhalation of oxygen, and in bad cases where the cyanosis is extreme and the signs of carbonic acid poisoning are beginning to appear it has given great relief, indeed, more in emphysema than in most other diseases of the lungs.

Of the complications, bronchitis is the most frequent and the most serious. It requires the most careful management, for it is upon it that the transient exacerbations of symptoms in great part depend. For this the usual remedies are indicated, except that they should be of the stimulating and not of the depressing class. Most of the remedies vaunted for emphysema are those which act upon the bronchitis, or upon the asthma if that affection be present. Opium must be given under the same conditions and with the same caution as in bronchitis. Where the secretions are not freely expectorated an emetic may do good, but

¹ *Bull. de l'Acad. de Méd.*, 1885, 888.

² *Berl. klin. Woch.*, 1890, No. 40.

³ Waldenburg, *Die pneumatisch. Behandlung*.

tartrate of antimony should be avoided on account of the depression which follows its use.

Cardiac failure is the greatest danger that has to be dreaded, and it may develop with very great rapidity in the course of one of those intercurrent attacks of bronchitis to which emphysema patients are so liable. In order to keep the heart as strong as possible every attention must be paid to the general health, the slightest sign of flagging noted and met, and the greatest care possible taken of the patient if convalescent from any intercurrent disease.

When cardiac failure has once set in, the right side of the heart must be relieved of strain as far as it can, by doing all that can be done to diminish the pulmonary obstruction, by placing the patient at rest, confining him, if needs be, absolutely to bed, and by the use of cardiac stimulants and tonics.

When the symptoms of cardiac failure become severe, and the right side of the heart is threatened with paralysis from over-distension, the question of relieving the over-distended cavities in some way becomes urgent. Dry-cupping between the shoulders is sometimes of advantage for the time, but more often fails, and nothing then is left but bleeding. The object of bleeding under these circumstances is simply to give mechanical relief to the over-distended cavities, and to effect this the blood must be withdrawn rapidly and in sufficient quantity. A large vein must be selected not too far away from the heart, and this is generally either the external jugular or one of the big veins of the arm.

The ventricle or auricle have even been tapped directly with a needle, but this operation introduces obvious risks of its own, and has, I believe, no advantage over venesection. Still, it has been done many times successfully.

The quantity of blood removed must be large, from 20 to 30 ounces in an adult of ordinary size, but this will depend in some measure upon the extent to which improvement in the symptoms follows. The relief given is often immediate and striking, so that the patient passes in a few minutes from a condition of urgent danger into one of comparative safety and comfort.

The objections to bleeding are obvious: (1) that it can at the best only give temporary relief; and (2) that the loss of so much blood will leave the patient in an anæmic and debilitated state which will in the end increase the weakness of the heart. This is true, but the object of bleeding in these cases is to meet a crisis, to give temporary relief, to save the life which is threatened. Where it is evident that the patient has not the blood to spare, or that the case is absolutely hopeless, then it would be folly to bleed; yet I have seen very desperate cases saved by a timely bleeding, which could not have lived without it more than an hour or two. The benefit may not only be obvious to those in charge, but the relief given to the distress may be so appreciated by the patient as to lead him afterwards to beg for its repetition, as in the following case.

A man of 45, deeply cyanosed and in the last stages of emphysema, was suddenly seized with severe symptoms; the dyspnoea became intense and the cyanosis grew deeper and deeper under observation. It was clear the man was dying, and he was bled to the extent of 30 ounces. His colour improved at once and the urgency of his symptoms rapidly passed away. A few days later a similar attack came on, and he begged to be bled again. This time 17 ounces were taken with the same relief. Once again, about ten days later, he was bled at his own request, but only 7 ounces were removed, and then also with great relief. The man died some days later, but there can be no doubt that he would not have survived the first attack had it not been for the bleeding, and how important the prolongation of life even for so long as this might be for the purpose of making a will or transacting other business needs not to be pointed out.

Free bleeding should not in emphysema, any more than in other diseases, be employed as a routine treatment, but in its place, and used with discretion, there can be no doubt that it may give great relief and for the time save life.

31. SENILE EMPHYSEMA — SMALL-LUNGED EMPHYSEMA—SENILE ATROPHY OF THE LUNG—ATROPHOUS EMPHYSEMA.

Although cavities are found in senile emphysema similar to those of ordinary emphysema, and produced in the same way by the opening into each other of neighbouring alveoli, still senile emphysema differs essentially from ordinary emphysema in the size of the lungs, which is greatly reduced below the normal instead of being increased. This diminution in size takes place in all directions, so that the thorax is contracted, the heart uncovered and the diaphragm raised.

As in ordinary emphysema, the vacuolation of the lung is most marked at the apex and edges, and the lung on section is dry and bloodless. On the other hand, the trabeculæ of the lung are frequently a good deal thickened and the tissue much pigmented. The bronchi are thinned and wasted, and often cylindrically dilated. These are changes like those which result from chronic bronchitis, and may probably in some cases be referred to that affection.

The right side of the heart is not hypertrophied or dilated; on the contrary, the whole organ is small and in the condition of brown atrophy.

The changes in the lung are associated with contraction changes in the thorax, which is shrunk in all directions, and presents the typical form of chest in the wasted old man. The back is rounded, probably from the atrophy of the intervertebral discs, the shoulders are thrown forward, the ribs have lost their curve, are flattened and sink downwards, so that the lower ones may almost reach the iliac crest. The sternum may appear prominent, but it is the flattening

of the ribs that makes it seem so. The cyrtometer tracing approaches the circular, as in ordinary emphysema, but for a different reason. In that affection both diameters are increased, but the antero-posterior most; while in senile emphysema both diameters are decreased, but the transverse most. It is to the flattening of the upper parts that the falling of the shoulders forward is due.

The intercostal spaces are narrowed and flattened, and the loss of elasticity in the lung may show itself in the upper spaces in front as well as in the supra-clavicular fossæ by the bulging on expiration and the recession on inspiration. The movements of the thorax on respiration are generally defective, for the chest moves as a whole with but little expansion; still the effect involves both inspiration and expiration, and is not, as in ordinary emphysema, chiefly expiratory.

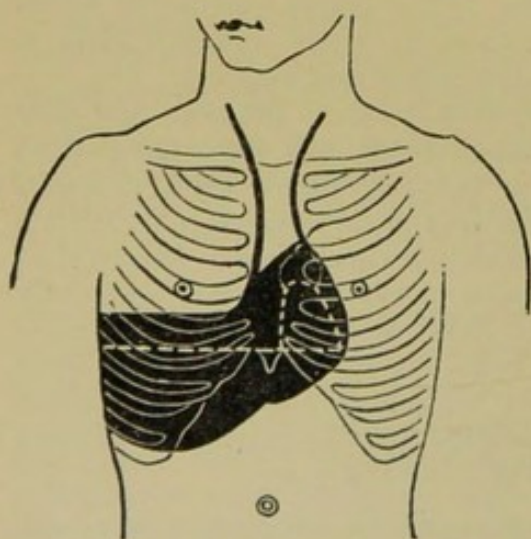


Fig. 53.

Diagram showing the boundaries of the lungs in a case of senile emphysema (bilateral contraction of the lungs). The white dotted line shows the normal boundaries.

Percussion demonstrates the great diminution in the size of the lungs, for the cardiac dulness is increased in all directions and especially upwards, the apex remaining in the normal place, the liver dulness commences at the fourth rib or space in the right nipple line on the left side, and the stomach resonance rises high into the thorax.

The percussion note is hyper-resonant over the whole pulmonary area, except sometimes at the apex, where it may be impaired owing to some fibroid induration there.

Auscultation yields little that is abnormal, unless it be slight prolongation of the expiration, or, if bronchitis be present, the usual signs of that affection. The vocal resonance is not much altered, but may be slightly increased, and the same is the case with the vocal vibrations.

The sputum is generally copious and purulent, and that even when auscultation yields little evidence of bronchitis. In other words, the bronchitis is often limited to the trachea and the larger tubes. If general bronchitis be present, the sputum has the ordinary bronchitic characters.

The breathing is short, but not markedly so; there is no cyanosis unless the bronchitis be considerable, nor are there any signs of cardiac dilatation or of venous obstruction.

Senile emphysema occurs in the wasting form of old age, *i.e.*, the patients are thin and generally wasted, not fat or well-nourished. The general signs of the disease are indefinite, *viz.*, those of increasing old age with a certain amount of bronchitis, but without any distressing dyspnoea or cyanosis.

The Diagnosis is for the most part easy. The age of the patient, the form of the chest, the alteration of the boundaries showing the decrease in the size of the lungs, these, together with the comparative absence of symptoms, are characteristic, and distinguish senile emphysema at once from the ordinary form. It is rather from other forms of contracted chests that the diagnosis has to be made, *e.g.*, from interstitial pneumonia, chronic phthisis, and old pleurisy. These affections, however, have different histories, develop at an earlier age, and are for the most part unsymmetrical.

Though a change peculiar to old people, senile emphysema is not found in all old people, not even in the most aged. If it stand in direct relation to any other disease it is probably to chronic bronchitis, and may be regarded in many cases as a chronic fibroid induration or contraction, with some secondary vacuolation of the lung in consequence.

No relation can be made out clinically between senile and ordinary emphysema, nor does the one pass into the other. The only points of resemblance are the vacuolation of the lung and the distribution of the lesion. In all other points senile emphysema stands in marked contrast with the ordinary emphysema; the size of the lungs, the shape of the chest, the character of the movements, the slightness of symptoms, the effect upon the heart, the age and period of development, and to these may be added the pigmentation of the lung and the frequent presence of indurative changes. In both the distribution of the vacuolation is the same and determined by similar causes. The primary change is an atrophy, this atrophy being in senile emphysema due to old age, and in ordinary emphysema to causes not yet known.

Prognosis.—The prognosis is fair. The disease is one of old age, but not otherwise of special gravity, except so far as it increases the danger of pulmonary complications when they arise, such as general bronchitis or pneumonia.

The duration of life does not appear to be specially affected by it; at any rate the condition with care may last for years without causing grave symptoms.

Treatment.—All that is required is the watchful tending of old age, protection against cold, and the careful management of bronchitis should it arise.

32. HYPERTROPHY OF THE LUNG — COMPLEMENTARY, COMPENSATORY, OR VICARIOUS EMPHYSEMA—RELAXED LUNG —PARALYSIS OF THE LUNG.

There are, no doubt, persons with large lungs as there are with large hands or feet. But by the term hypertrophy is not meant an increase in size which is a departure from the general average for the race, *i.e.*, a physiological variation, but one which is an abnormality for the particular individual, which has developed in that individual and was not always present in him, *i.e.*, it is a pathological condition. To be a true and not a spurious hypertrophy the increase in size must be unattended by any structural change in the organ, and must be accompanied with an increase in functional power.

In this sense we are familiar with hypertrophy of one lung in those cases in which the other has become shrunk and useless, a change which is strictly analogous to the hypertrophy of one kidney which takes place when the other is destroyed.

What occurs in one whole lung occurs doubtless also in parts of one or both lungs in order to compensate for local disease in other parts of them. This condition, whether unilateral or partial, is commonly described as complementary or compensatory emphysema, but it would be more correctly called hypertrophy.

Whether there is such a thing as general hypertrophy of the whole of both lungs is open to question. I believe it does occur, and that it is met with in those persons who have to use their lungs excessively, either habitually or from time to time. I have seen what I regard as this condition in athletes and in persons in training; the lungs in them were increased in size, the cardiac dulness covered up, and the diaphragm standing low, yet the respiratory movements were unusually free and the respiratory capacity and power greatly increased. There is an analogy for this in the hypertrophy which takes place under similar circumstances in the heart.

Such hypertrophy, interesting as it is, is a simple physiological adaptation of the lungs to the increased demands made upon them, and not being a morbid pathological condition requires no further consideration here.

UNILATERAL OR COMPLEMENTARY HYPERTROPHY OF THE LUNG.—This occurs when the opposite lung is contracted or shrunk, either as the result of disease in itself, as, for instance, of chronic tubercular excavation or fibroid induration, or in consequence of chronic pleurisy. In these cases the hypertrophied lung may increase even to nearly twice its normal size. On section no marked change from the normal is to be seen. The vesicles may possibly be a little larger than usual, but this is often hard to say, so that there must be considerable numerical as well as physiological hypertrophy. There is at any rate no true emphysema at all, for the vesicles do not open into each other and the functional power is increased; indeed the compensation may be so complete that there may be but little or no shortness of breath. In one instance of this kind the patient, a young man, was a first-rate short distance runner and the winner of many prizes.

Usually in such cases there is contraction in the diseased side, so that some deformity or want of symmetry is apparent in the thorax, but the hypertrophy may be so considerable as to completely make up for the contraction and

prevent any obvious external deformity. It has been asserted that in such cases as these the condition must be congenital, but it is not so generally; on the contrary, I believe that it is almost invariably acquired. I have more than once watched it develop, and I have seen it become well marked within a few weeks, though usually it takes a much longer time than this. All that is necessary is that the lung should be healthy, and thus its occurrence is a favourable sign. When the lung is not sound the hypertrophy does not take place, or at any rate not to the same extent.

Hertz states that this vicarious hypertrophy has not as yet been diagnosed during life. I am surprised at this statement, for I am so familiar with this condition that I should not regard it as at all rare. I have recognised it and demonstrated it many times during life, and in some cases have verified the diagnosis *post-mortem*.

It is quite easy to recognise, and is frequently missed only because it is not looked for in the right way. What is quite conclusive is the position of the middle boundary of the lung. This is easily made out by percussion; it extends across the sternum in a bold curve, and at the level of the fourth rib may reach $2\frac{1}{2}$ inches, as I measured it on one occasion from the middle line of the sternum. The heart is of course displaced, and the physical signs are, as would be expected, most marked when it is the left lung which is contracted and the right which is hypertrophied.

The displacement may indeed be as great as is met with in the most marked cases of pneumothorax. The level of the diaphragm is not altered, and the upper limit of hepatic dulness remains in its normal place at the upper border of the sixth rib in the right nipple line, showing clearly that the displacement is not a simple stretching of the lung by being drawn over to the contracted side. The breath sounds are not altered over the hypertrophied lung, except that as more air enters that side the noise of its passage in and out may be increased and the breathing become somewhat puerile, but it is everywhere vesicular.

Such a hypertrophied lung may remain for a long time, even for years, perfectly sound, but a one-lunged man is of course liable to greater risk from any pulmonary complication. Where the original disease has been tubercular, the remaining lung may ultimately also become tubercular, and the case will then run its course rapidly to its end, but even in these cases it is remarkable how long the implication of the second lung may be postponed.

What takes place in the whole of one lung when the other is contracted may also occur under similar circumstances in parts of a lung when some other part of it is obliterated by disease. Accordingly, it is not uncommon to find such hypertrophy in the upper parts when the lower have been bound down by chronic pleurisy, or in the lower parts when the upper have been the seat of chronic phthisis. These conditions have usually been spoken of as vicarious emphysema,

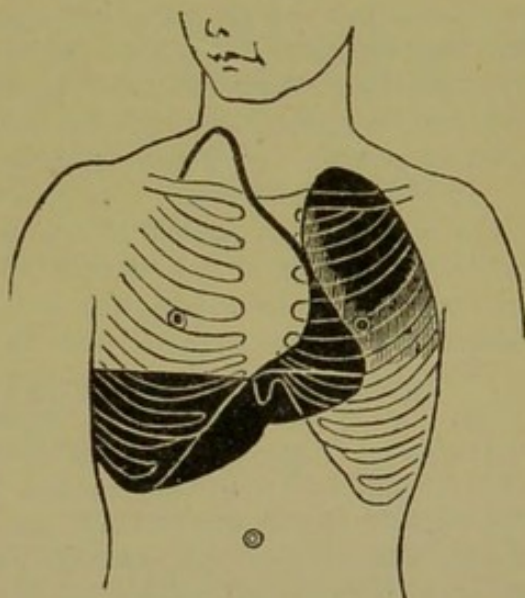


Fig. 54.

Diagram showing the displacement of the middle boundary of the lung in a case of fibroid contraction of the left lung, with complementary emphysema of the right.

but the vesicles here again are not much distended, there is no vacuolation of the lung, and the functional power is increased.

Hyper-distension of the lung.—From this local hypertrophy must be distinguished—at any rate theoretically, for the clinical distinction during life is often impossible—those conditions in which the vesicles are simply over-distended. In the great majority of cases this is a transient state, and depends upon the transient collapse of some neighbouring part such as so frequently occurs in the course of bronchitis in children. It passes off quickly as soon as the collapse upon which it depended has been obviated.

In the transient form, as well as in some of the more persistent forms, the increase in size of the parts is due to simple hyper-distension, and in neither are the changes characteristic of true emphysema found, so that to avoid confusion these conditions would be much better described as complementary, compensatory, or vicarious hyper-distension rather than emphysema. (*German Aufblähung. Volumen pulmonum auctum.*)

The mechanism of its production is no doubt chiefly inspiratory, *i.e.*, as the parts collapse the air inspired forces the parts around to dilate until the contraction is compensated. This collateral distension would obviously be greatly aided by expiratory efforts such as coughing. It is most marked and most common in children in connection with bronchitis and whooping cough, and often leads to marked prominence of the upper parts of the chest.

In the partial form of vicarious emphysema the hyper-resonance on percussion is the only sign by which it can be recognised. This is often sufficient to mask entirely the signs of the collapse which has caused it, so that the diagnosis of the collapse has to be made, not by positive physical signs, but indirectly and by inference from the hyper-resonance due to the vicarious emphysema.

The hyper-distension is sometimes general, and involves the whole of both lungs. It is then caused by violent coughing, or by some obstruction to the exit of air. Accordingly, it is met with in whooping cough, and occasionally with foreign bodies in the trachea or larynx. A similar transient condition is met with in spasmodic asthma, and must be distinguished from true emphysema, which is also frequently associated with that affection in its later stages, in some instances undoubtedly following the asthma and caused by it, but in others preceding and exciting it.

Such general hyper-distension is easily made out by the displacement of boundaries, like that of true emphysema, except that it is transient; the cardiac dulness is absent, the diaphragm low, and the hyper-resonance well marked.

RELAXED LUNG.—There remain finally for consideration those cases which resemble the last in the hyper-resonance to percussion, but differ from them all in the fact that the hyper-resonant parts are not increased but diminished in size. The common instances of this are met with in pneumonia or pleuritic effusion, or under similar conditions. Here the lungs have lost their tone or tension, and yield a note on percussion like that obtained from them after removal from the body.

In pleuritic effusion this relaxation is no doubt mechanical, and it is met with also when the diaphragm is pushed up into the thorax by ascites, an abdominal tumour, or distension of the bowels. In pneumonia a different explanation must be sought for, and is to be found, I believe, in the altered nutrition resulting from the adjacent inflammation.

This peculiarity of percussion is often extremely well marked and easily recognised. It is known as *skodaic resonance*. In the case of pneumonia it may be so well marked in front as to enable the consolidation behind to be correctly diagnosed, when it is impossible, on account of the patient's state, to examine the

back thoroughly. As the pneumonia subsides the hyper-resonance passes away, often before the consolidation has resolved, but it may continue even after resolution has commenced; in other words, the nutrition of the lung may take some time to recover itself. Usually in pneumonia the loss of tone occurs only in the remaining portion of the inflamed lung, but it sometimes involves the opposite lung as well, without there being in that lung any local cause.

The same loss of tone or tension in both lungs may also be met with in the course of general diseases in which the lungs are not otherwise affected. I have seen it several times, most frequently in typhoid fever. It is strictly comparable with tympanites, and is probably, like it, of neuro-muscular origin, and of very serious prognostic importance.

This condition has been described as paralysis of the lung, but the terms *pneumo-paresis*, or *paralysis*, have also been employed to indicate what has been believed to be a different condition, viz., one in which there is paresis or paralysis of the vasomotor nerves. If there really be such a condition at all it manifests itself by similar signs, and is of the same ominous significance.

To sum up what has preceded, there are, besides the truly atrophic forms of emphysema, viz., ordinary emphysema and senile emphysema, a variety of conditions which have been confused under the common term of emphysema.

1. The true hypertrophies, bilateral, unilateral, and partial.
2. The transient hyper-distensions, also bilateral, unilateral, and partial.
3. And lastly, the relaxed lung, sometimes produced by mechanical causes, sometimes by failure of nutrition, and this dependent upon local or upon general causes.

33. INTERSTITIAL OR INTERLOBULAR EMPHYSEMA.

In interstitial emphysema the air is found in the interalveolar or interlobular tissue of the lung and beneath the pleura. It presents itself *post-mortem* in the form of strings of beads or bubbles of air in the interstitial tissue, which a little pressure easily causes to travel from place to place, and of bladders of varying size beneath the pleura. It is easily produced experimentally by blowing the lung up with air. When the distension has reached a certain degree the lung bursts and air soon appears beneath the pleura, at first as small bladders, but it may rapidly extend and strip off the pleura from the lung for a considerable distance. With the lungs *in situ* this does not happen to the same extent, owing to the support which the pleura gets from the thoracic walls, and the air then travels along the interlobular septa and round the air tubes to the root of the lung and mediastinum, whence it may extend upwards to the root of the neck, producing surgical or subcutaneous emphysema. When the air has once reached the neck it may spread with rapidity over a great portion of the trunk.

Within the body the weakest spot in the pleura appears to be near the root of the lung, and here rupture may take place and pneumothorax be produced.

The pressures necessary to burst the lung and produce interstitial emphysema are considerable, but vary a good deal in different individuals.

Out of the body a pressure of from 2 to 4 inches of mercury are required, and in a case in which I performed the experiment with the lungs *in situ* rupture did not take place until the pressure reached 8 inches of mercury. These observations agree with those of Hutchinson, who stated that the healthy lung could resist a bursting pressure of from 3 to 9 inches of mercury.

In the infant a careful study of this question has been made by Dr Champneys.¹ He observed the same individual variations in his experiments, but the lowest pressure under which the lungs of the infant burst was 20 mm. of mercury (a little less than 1 inch), and the highest 80 mm. of mercury (= 3 inches), while it required from 30 to 100 mm. of mercury to burst the pleura. Again, while 20 mm. of mercury pressure were sufficient to burst the lung out of the body, 30 mm. were necessary with the lungs *in situ* and the front of the chest open, and from 80 to 100 with the chest intact.



Fig. 55.

Interstitial emphysema in the lung of an infant aged 9½ months, who died of whooping-cough. Large bubbles are seen beneath the pleura, and the course of the main superficial lymphatics running towards the root of the lung is marked out by them. (Photograph from a preparation in the St. Bartholomew's Hospital Museum 1693.)

root of the lung, and there lead to rupture of the pleura and pneumothorax.

Interstitial emphysema is a very rare affection at all times, and in the adult even rarer than in the child. In each it is most likely to occur where violent expiratory efforts have been made, and it is usually referred to them.

In the adult no expiratory efforts probably exceed those of the second stage of labour, and accordingly parturition is given as one of the commonest causes of the condition; yet, as a matter of fact, it is above all things rare.

¹ *Artif. Resp. in Stillborn Children*, 1887.

² Champneys, *loc. cit.* Wilks and Moxon, *Pathol.*, 2nd ed., 1875, p. 308. Angel Money, *Med. Chir. Trans.*, lxxvii. 101. For other refer. cf. Wilson Fox, *Lungs*, p. 183.

When rupture of the lung has once occurred, and air made its way into the interstitial lesion or beneath the pleura, it spreads under a much lower pressure, *e.g.*, 10 mm.

The trachea and bronchi require very much higher pressure to rupture them, *e.g.*, 100–150 mm. Hg.

Interstitial emphysema cannot be diagnosed during life, at any rate with certainty, until the air has reached the neck and become subcutaneous.

Subcutaneous emphysema of the neck has, however, many other causes besides rupture of the lung. Most frequently it arises as the result of rupture, either from disease or injury of the large air tubes or of the œsophagus. In another group of cases it follows tracheotomy, especially when artificial respiration has been performed and the same effect has been produced experimentally.

In these cases² the air follows the reverse direction, viz., from above downwards. It may spread widely over the neck and body, and also reach the mediastinum and

At the Dublin Hospital it was found to have occurred only 7 times in 13,748 cases, *i.e.*, in about once in 2000 cases. It may be noted in passing that pneumothorax was not met with once in this series.¹

Of the few instances recorded most have occurred in children in the course of whooping cough, or in new-born infants after insufflation. Even violent crying is stated to have caused it, but as against all these instances in which these causes have led to interstitial emphysema must be set that overwhelmingly greater number of instances in which they have not.

The necessary conclusion seems to be that the lungs, which have given way, have not been really healthy, but have been weakened by some past or present mischief. In some of the recorded cases definite lesions have been found, *e.g.*, broncho-pneumonia, lobar-pneumonia, and even phthisis.² When the lungs show no such lesions as these, collapse will hardly fail to be present. This is of course the condition in the lung of the new-born child that has not breathed, and it is a very frequent lesion in whooping cough. It is commonly stated that collapsed lung is easily distended, and it might be assumed that the first effect of over-distension, *e.g.*, of insufflation, would be to expand the collapsed portions. But this is not always so, and, as a matter of fact, if collapse has lasted some little time it may be difficult or even impossible to blow the lung out at all; indeed, it has happened to me in my experiments more than once to burst the lung without at all effecting the expansion of the collapsed parts.

It seems impossible then to avoid coming to the conclusion that, when interstitial emphysema does occur, as the result of coughing, violent expiratory efforts or insufflation in either child or adult, the lung is not really healthy, whatever it may appear to be, and it thus seems to be the case that interstitial emphysema cannot any more than pneumothorax be produced by any expiratory effort, ordinary or extraordinary, unless the lung be previously unsound.

With so rare an affection there is little to say in respect of **prognosis**. The slighter degrees are said to be recovered from, but considering the uncertainty of the diagnosis in slight cases, such a statement must be taken with much allowance. The graver cases, in which the air reaches the mediastinum or neck, are generally fatal, sometimes from the dyspnœa, sometimes from pneumothorax. In a few isolated cases recovery is stated to have occurred, but such facts are of no use in the prognosis of any given case.

Treatment there is none, except as far as possible to relieve the cough or control the violent respiratory efforts. The risk is that the child will die from dyspnœa or pneumothorax. If this does not occur the air itself need cause no special anxiety, for it will be absorbed and disappear, sometimes with great rapidity.

34. ATELECTASIS—COLLAPSE— APNEUMATOSIS.

Atelectasis is the name given to the condition in which the vesicles of the lung are collapsed and contain no air. It may be *congenital* or *acquired*; in the former case the vesicles have never expanded but remain in the foetal state, in the latter the air which the vesicles once contained has been removed either by being forced out by pressure from without (*Compression-Atelectasis*),

¹ Johnson and Sinclair, quoted Champney, *loc. cit.* Sir Wm. Turner, *Med. Chir. Trans.*, xl. 31.

² Damsch, *Deut. med. Woch.*, 1891, 618.

or by absorption from within, as the result of obstruction to the air tubes (*Obstruction- or Absorption-Atelectasis*).

Collapsed lung is airless, tough, firm and fleshy (carnified), does not crepitate on pressure or on section, sinks in water, is of a bluish-purple colour, and, if superficial, is shrunken and depressed below the surface.

The collapsed parts may at first be blown out again, but after a time this is not possible, owing to changes which have taken place in them, and led either to adhesion of the walls to each other, or to interstitial fibroid induration.

In the common obstruction form which results from bronchitis, the catarrhal inflammation is likely to spread to the collapsed vesicles, which then become distended again by the products of inflammation, a patch of broncho-pneumonia being the result.

The three forms of atelectasis are clinically sufficiently distinct from one another to call for separate consideration.

CONGENITAL ATELECTASIS.—In this form the vesicles have never expanded, but remain in the foetal condition, with the walls in contact and lined with cubical epithelium.

Complete atelectasis is, of course, incompatible with life, and is only met with in the stillborn. In infants who have breathed for a time, but have died shortly after birth, the whole of one lung or parts of both may be found collapsed, or smaller patches be scattered widely throughout the whole of both lungs. The parts most commonly affected are the bases behind and the lower margins and anterior edges in front. The apex is the part affected least of all.

When disseminated, the patches are sharply marked off by their purple colour from the rosy pink of the surrounding parts, and, if on the surface, are depressed below the general level; the pleura over them is smooth and has not lost its polish.

The collapse is usually due to defective respiratory movements, and is met with therefore in premature or in feeble, sickly infants, after difficult labour, or where the child has been stillborn, and subjected to artificial respiration or to insufflation. Other subsidiary causes may be found in undue weakness of the ribs from syphilis or rickets, and in obstruction of the air tubes by mucus or some of the discharges occurring during parturition.

Symptoms.—If the collapse is extensive, the condition is easily recognised by the general condition of the child. The respiratory movements are defective, the expansion imperfect, and the cyanosis marked and progressive.

In extreme cases life is rarely prolonged beyond a few hours. All that can be done to stave off death is to stimulate the child to breathe more deeply by slapping it on the back and buttocks, by plunging it into a bath of hot and cold water alternately, by artificial respiration or by insufflation. Of the methods of artificial respiration, the most effectual, according to Champneys' observations, is that of Sylvester. Little good can be done by these measures, except immediately after birth, and if they are without avail within a short time, the case is hopeless and the child will die.

In less extreme cases the child may live some days. Such infants are usually ill nourished and feeble, dull, and drowsy; the dyspnoea is evident, and the respiration short and shallow; the lower parts of the thorax recede with inspiration; the child has no voice to cry, but gives only a low whine or moan, and may refuse to suck.

The cyanosis is marked, especially in the face and fingers. The pulse is feeble and often irregular, and the fontanelles depressed. The skin feels cold and clammy, and the temperature is below normal.

In these cases, too, after a few days' struggle the symptoms become worse, the dyspnœa and cyanosis increase, twitchings or actual convulsions set in, the child passes into a state of unconsciousness, and dies.

When life is still further prolonged the child may rally, and after a time even the collapsed parts may re-expand and complete recovery occur. This can only occur where the collapse is slight and the general strength is well maintained. As a rule, if the child lives the affected parts of the lungs remain collapsed and ultimately become fibroid.

Instances of what has been regarded as congenital atelectasis have been recorded in adults, but most of them are very far from conclusive. In most cases the child continues weakly, and frequently succumbs to what seemed to be only a slight catarrh, the unexpected result of which is explained by the finding of the collapse after death.

It is rare in congenital atelectasis to obtain any physical signs, but sometimes dulness to percussion may be detected and be associated with defective breath sounds.

Associated Pathology.—If life be prolonged the affected parts may pass into a condition of fibroid induration. Broncho-pneumonia, which is so common in the obstructive form, is rare in the congenital form. The heart is, of course, frequently dilated and contains *ante-mortem* clot; thrombosis in the cerebral sinuses has also been described.

It has been suggested that atelectasis may be the cause of congenital morbus cordis, the obstruction to the pulmonary circulation checking the developmental changes which ought to complete themselves immediately after birth. This seems far-fetched, but deserves mention.

The **Prognosis** of congenital atelectasis is bad. Most of the cases end fatally in a few days after birth, or, when life is prolonged, the child, left with damaged lungs, will remain feeble and ill-developed, and be liable to be carried off by very slight causes.

Treatment.—For the extreme cases the treatment has been already spoken of; for the less extreme cases it consists mainly in supporting the strength by feeding and tonics, and by protecting the child against cold.

COMPRESSION-ATELECTASIS.—In this form the collapse is brought about by causes external to the lung, which either allow the lung to contract by virtue of its own elasticity or actually compress it.

All conditions which cause the lung to occupy less space within the thorax than normal may lead to this form of collapse, and among these, pleuritic effusions and pneumothorax are at the same time most effective and the most frequent.

Complete collapse can of necessity only be unilateral, and is met with where the pleura is completely filled with effusion or with air. The lung then gathers itself round its root and lies flattened like a pancake against the spine. If adhesions exist anywhere the lung may assume curious shapes.

In one case, in which there was an adhesion almost in the middle of the axillary region, a cord extended from the spot to the mass of completely collapsed lung at the side of the spine, and this cord proved to be composed of the corresponding portion of the completely collapsed lung.

At other times a considerable part of the lung may be kept expanded, and this occurs especially at the apex where lesions, as a rule tubercular, have led to obliteration of the pleural cavity.

Partial collapse may be unilateral or bilateral, according to the cause, upon which also its situation will greatly depend. Thus with ordinary pleuritic effusions the collapse is found in the lower lobes; with a large heart or pericardial effusion

at the anterior edges and in the left lower lobe; with rickets in the lower parts because of the flaccidity of the ribs; with curvature of the spine, posteriorly in angular and postero-laterally in lateral curvature; and similarly with other deformities of the thorax. When with distension of the abdomen the diaphragm is thrust up into the thorax, as, for example, with ascites, tympanites or abdominal tumour, it is, again, the lower parts that become collapsed. With intrathoracic tumours the parts collapsed vary with the seat of the tumour; but such tumours, especially when seated in the mediastinum, usually lead to more than simple collapse, and by compression of the air tubes and blood-vessels produce a mixed condition of collapse, œdema, inflammation and degeneration.

In all cases alike, though the change in the lungs is most marked in the immediate neighbourhood of the exciting cause, the tension of the lung is affected throughout, so that the parts at a distance lose tone to some extent; this loss of tone gives rise to the hyper-resonant note on percussion which is characteristic of relaxed lung, and in cases of large effusion this relaxation may be evident, even on the opposite side.

One of the best instances of cracked-pot sound I have ever met with occurred in a man with a healthy but relaxed and partly collapsed lung, lying over a dilated and hypertrophied heart.

For a time the collapsed parts may be easily blown up *post-mortem*, or if the cause of the compression be removed, will spontaneously recover, but after a time changes may take place in the lung or in the pleura, or in both, which fix the lung permanently in the collapsed position and render re-expansion impossible. The rest of the lung then dilates to compensate, and if healthy, undergoes complementary hypertrophy. Such changes, however, do not necessarily occur, even when the lungs have been collapsed for long periods. In speaking of pleuritic effusion and pneumothorax, instances will be given in which one lung had been completely collapsed for months, in two cases for as long as eighteen months, and yet on removal of the fluid expanded at once and completely.

The **symptoms, treatment, and prognosis** of compression-atelectasis depend upon the causes which produce it. If the cause be removable and too long time has not elapsed, the collapse will restore itself, and the affected parts become active again.

The gravity and urgency of the symptoms depend chiefly upon the rate at which the collapse develops; if, as in pneumothorax, its development has been sudden, the symptoms will be urgent, but if gradual, as in slowly-increasing effusions, the symptoms may be very slight, inasmuch as time has been given to the lungs to accommodate themselves to the gradually altering conditions.

OBSTRUCTION-ATELECTASIS.—In this form the collapse is due to the fact that the air tubes are obstructed, so that the air cannot enter as freely as it should.

When a bronchus is completely obstructed, the air disappears and the vesicles collapse.

As to the way in which the disappearance of the air is brought about, opinions have been divided. For a long time Gairdner's ball-valve theory was accepted, by which it was held that the removal of the air was mechanical, the obstruction acting as a valve, permitting the exit of air on expiration and preventing its entrance on inspiration. This view, which was, *a priori*, improbable, was disproved by Lichtheim's¹ experiment, in which the possibility of any valve action was excluded by the use of laminaria plugs, by which the occlusion of the tubes was made complete. It was thus proved that the air was removed by absorption. Lichtheim also showed that the gases which compose the air are removed, as was to be expected, at different rates, the oxygen most and the nitrogen least rapidly. One difficulty, however, presented itself: absorp-

¹ *Arch. f. exp. Pathol.*, 1879, p. 52.

tion would account for the disappearance of the air until the tension of the gases in the blood and in the vesicles of the lungs was the same, and then no further absorption would occur, whereas the fact was that the air was completely removed, and the vesicles became entirely collapsed.

To explain this, Lichtheim called in the elasticity of the lung, which he assumed was not fully satisfied until the vesicles were completely collapsed, and in support of this he appealed to the complete collapse of the foetal lung. He also stated that when a lung removed from the body was blown up and suspended in the air and kept moist, the air was gradually absorbed, and collapse took place at first in the external parts but ultimately in the whole lung.

On the other hand, it must be borne in mind that in the case of pneumothorax, the air is absorbed from the pleura quite as completely and as readily as from the lung, and in this case the absorption takes place with the elasticity of the lung acting in antagonism to it.

The subsidiary force required by the absorption theory, if the elasticity of the lung be inadequate, may be looked for in chemical combination. But this, which may be active in the case of oxygen and carbonic acid, will not so easily explain the complete removal of the nitrogen. It may be that the subsidiary force required is, after all, not physical but simply mechanical, and to be found in the increase of pressure produced by expiration, especially when increased as it is by the coughing which attends the affections in which collapse is usually met with.

Although the theory is not perfect in all its details, still Lichtheim's observations conclusively prove that the removal of the air is the result of absorption, and they also show that the absorption takes place chiefly by the blood-vessels; for whereas when the circulation was intact the absorption was complete in two to three hours, it required about double that time when the vessels were ligatured.

The question of the absorption of air from the lung cannot be separated from that of the absorption of air from the pleura, and looking at the question in a broad, comprehensive way, the conclusion can hardly be avoided that it is to the forces of chemical combination that we must look for a complete explanation of all the facts, though it must be admitted that at present full proof of the theory is not forthcoming.

If the collapse be of small extent, the diminution in bulk is made up for by the dilatation of the surrounding parts (*complementary emphysema*). Thus collapse and complementary emphysema go hand in hand. But this compensation is only possible up to a certain point. When this point is passed more extended collapse can only be provided for by the falling in of the soft parts, *i.e.*, of the intercostal spaces if the ribs be rigid, or of the thoracic walls if the ribs be soft. If the obstruction be in one of the large air-tubes, so as to involve the whole or greater part of a lobe, and if the ribs are rigid so that they cannot fall in much, complete collapse becomes impossible. As the air is removed, the blood-vessels and lymphatics of the affected parts dilate and effusion takes place into the alveoli; thus the resulting condition is not simple collapse, but a mixture of collapse, congestion, and oedema, *viz.*, the condition described as solid oedema. The same results follow if the obstruction affect the larynx or main air tubes, as in croup or with a foreign body, but as in these cases the whole of both lungs is affected, death cuts short the process before it has advanced very far.

In young children bronchitic affections are especially liable to lead to obstruction of the air-tubes, owing to the small size and slight resistance of the finer air-tubes, as well as to the fact that children have not learnt how to cough properly, *i.e.*, do not know how to use the expiratory muscles in the best way, so as to expel the offending secretion from the tubes. Moreover, the consequent collapse is likely to be extensive because of the softness and defective rigidity of the thoracic wall.

Hence it comes that in young children collapse is of greatest frequency and importance. Indeed, simple or pure collapse is hardly ever met with except in them.

The shrinking and non-expansibility of the collapsed lung is made more evident by the respiratory movements, so that the corresponding parts are drawn in during inspiration, *viz.*, the intercostal spaces if the ribs are rigid and the chest walls if the ribs be still soft.

Though obstruction-collapse may occur in any part of the lung, wherever an air-tube happens to be obstructed, still the common seat of the lesion is the lower lobes. This is due to two facts, viz., that the common cause is bronchitis, and that the secretion which causes the obstruction gravitates to the lowest parts. But even when the obstruction is in the trachea or larger bronchi, as in croup, the collapse chiefly occurs in the lower part. The reason is that the lower parts of the chest are the most pliant and movable, and therefore most easily sucked in; but even in these cases the collapse does not affect the lower parts of the lungs uniformly, but certain parts especially, viz., the margins all round, for with marked inspiratory recession it is these parts which are the most compressed between the thoracic walls and the viscera beneath the diaphragm, chiefly, of course, the liver on the right side.

In little children, no doubt, the feeble muscular development plays its part in the mechanism of collapse; but this has not really, I think, the importance which is often attached to it, for however strong the respiratory muscles might be, they could not act to full advantage if their attachment lacked rigidity, as the thoracic walls do in little children.

If the thoracic walls be rigid, as in the adult, I fail to see how mere inspiratory deficiency can cause collapse. It may lead to defective expansion of the chest, but of itself it cannot lead to collapse. The thoracic walls are kept in the mean position between inspiration and expiration by the action of mutually opposing forces, and the respiratory movements depend upon the alternate action of these forces. The forces which expand the chest are muscular, and if the inspiratory muscles are weak or paralysed, not only will the respiratory movements be defective, but some contraction of the side will occur, although not of itself sufficient to cause collapse as it is ordinarily understood.

If, however, the diaphragm be paralysed, collapse of the lower parts of the lungs will necessarily follow, not so much because the diaphragm does not act, as because it ceases to offer its normal resistance to the abdominal muscles, the unopposed action of which would then force the diaphragm up and cause collapse of the parts in relation with it by compression, in the same way that abdominal distension does.

Long-standing obstruction to the air passages, or its frequent recurrence during childhood, *i.e.*, at the time when the ribs are soft, is the explanation of the common chest deformities, viz., *Harrison's furrow* and the various forms of *pigeon breast*. These may occur with obstruction in the nasopharynx, *e.g.*, with large tonsils and adenoids, but they more frequently develop in rickety children, who, besides having bones which are abnormally soft, are specially subject to recurrent bronchitis.

1. *In Children*.—In infants with capillary bronchitis, collapse is the complication which is most frequent and most to be feared. The patches may be numerous and widely scattered, but being masked by the complementary emphysema, may yield no physical signs. Thus the diagnosis of collapse becomes generally a matter of inference and not of direct evidence. Collapse may be inferred when the amount of dyspnoea and cyanosis exceeds that which the bronchitis alone seems sufficient to explain. At the bases behind, it is sometimes extensive enough to give a dull percussion note; the voice and breath sounds are then as a rule absent there, so that the diagnosis from pleuritic effusion may not be easy. If the collapsed parts pass into a state of broncho-pneumonia, the voice sounds may become increased and the breathing bronchial, and the crepitation may acquire the peculiar sharp ringing character which is almost pathognomonic.

Inspiratory recession is often considerable, especially in infants, and it may be almost as extreme as in croup. The transient constriction thus produced across the lower part of the thorax used to be described as the peripneumonic furrow, and when persistent it is the same thing as Harrison's furrow.

Inspiratory recession in bronchitis is a grave sign, though not necessarily of fatal omen.

Perhaps the most remarkable fact about atelectasis in children is the rapidity with which it may disappear. Thus I have seen the base dull one day, and the next resonant again. This rapid change is, of course, not met with in pleuritic effusion or in broncho-pneumonia, and therefore in doubtful cases helps to establish the diagnosis.

Usually when the bronchitis subsides the collapse which it has caused quickly disappears, and the lungs become quite normal again.

Permanent changes in the lung as the result of simple collapse are by no means common.

The **treatment** of collapse consists in exciting the child to breathe deeply, to cough or to vomit, in the hope that the secretion which has plugged the tubes may be dislodged. For this purpose the child may be put into a hot mustard bath, or may have an emetic given it—sulphate of zinc, gr. i., or sulphate of copper, gr. $\frac{1}{4}$, in a teaspoonful of water, may be safely given to an infant, or subcutaneous injection of apomorphia (gr. $\frac{1}{30}$ to $\frac{1}{40}$), but the nauseating and depressing emetics are contra-indicated. Collapse is generally an indication of failing powers, and therefore stimulants will be necessary, and all depressing treatment must be avoided.

2. *In Adults.*—In adults collapse plays a very subordinate part in bronchitis compared with its frequency and gravity in children, and when it occurs it is, as stated, rarely pure, but a mixture of collapse, congestion and inflammation.

History.—The literature of atelectasis is copious, but the important facts in its history are few.

Until the early part of this century, the condition, though recognised in infants, was attributed to congenital pneumonia, and it is to Jörg, in 1832, that the credit is given of referring it first to imperfect respiration. Legendre and Bailly, about 1846, distinguished it from pneumonia, and also described the condition in older children. About the same time West and Gairdner gave it the name of Collapse, by which it is best known in this country. A little later Bartels and Ziemssen, among others, showed the relation in which collapse stands to lobular pneumonia. Gairdner's theory of its mechanical production was accepted by most writers until recently, though Bartels pointed out the inadequacy of this theory, and suggested the absorption of the air by the blood as the real explanation. This view was not finally adopted until Lichtheim's writings in 1879.

35. HYPERÆMIA—CONGESTION—HYPOSTATIC CONGESTION AND ŒDEMA.

The circulation of blood through the lungs varies with the physiological requirements of the body. Hyperæmia is the natural result of increased physiological activity, and then not only does an increased amount of blood pass through the lungs, but owing to the dilatation of the vessels, they also contain at any given time a much larger amount of blood than normal. Such physiological hyperæmia is an indication of health, but similar hyperæmia which is not in response to physiological demands constitutes a pathological condition to which the name congestion is given.

Hyperæmia or congestion is the initial stage in inflammation, and it is quickly followed by exudation; so again venous congestion is likely to end in effusion in the lungs as elsewhere. Hence it comes that congestion of the lungs cannot be marked off by any definite line from inflammation on the one hand, and from œdema on the other.

Symptoms and Physical Signs.—The line between simple congestion and congestion with exudation is difficult to draw except in theory.

Simple congestion, from its interference with the circulation, and therefore with the aëration of the blood, leads to more or less dyspnœa, as the result of which the breathing is somewhat accelerated, but no physical signs are produced unless it be that the increased movement of air causes the ordinary breath sounds to be somewhat exaggerated and noisy.

When exudation occurs the signs are those of bronchitis; suffocative bronchitis if the fluid effused be abundant; of hypostatic congestion or œdema if it be effused slowly and gravitate to the lower parts.

The signs of congestion are practically those of bronchitis, and, as a matter of fact, simple congestion, *i.e.*, congestion without exudation, is hardly recognised clinically as a pathological condition except in connection with mitral disease; but in what close relation congestion stands to exudation even in these cases is shown by the fact that it is the tendency to recurrent bronchitis from slight causes that, in many early cases of mitral disease, first brings the patient to the doctor. Profuse expectoration of watery fluid is often taken as the pathognomonic sign of œdema, but this is incorrect, for the most serious cases of œdema are those in which fluid is exuded copiously into the tubes, but not being expectorated suffocates the patient. Indeed, profuse expectoration is an evidence either that the œdema is not very acute or that it does not involve both lungs.

When hypostatic œdema occurs the fluid exuded drives the air out of the vesicles, and so the part becomes dull on percussion.

If the tubes are also choked, as usually happens, the voice and breath sounds are diminished or absent, and the diagnosis becomes difficult from collapse or pleuritic effusion.

Speaking generally, then, the signs of congestion of the lungs are those of bronchitis, and the symptoms are varying degrees of dyspnœa up to the most intense form of it, such as is met with in suffocative bronchitis. In acute cases the symptoms may develop with very great rapidity and the patient die asphyxiated in the course of a few hours. Such cases are described as acute congestion or acute œdema, indiscriminately.

Results.—Congestion, as stated, naturally leads on to *exudation*. If the congestion be extreme, the small vessels in the alveoli may rupture here and there and *hæmoptysis* be the result. If the bleeding is slight, the blood may lie in the vesicles some time before expectoration, and become dark in colour; the small solid black lumps so commonly expectorated in the course of mitral disease, which sink in water, are instances of this. If the bleeding be considerable, the blood is in part expectorated at once and is of brighter colour. The condition in the lung will then be one of *infarct*, and will yield the usual physical signs.

Broncho-pneumonia may arise in consequence of the exudation in the tubes in the same way as it does in the course of bronchitis. Lastly, the lung in the condition of hypostatic congestion is specially liable to *acute pneumonia*, but this often develops insidiously, and with little evidence of its occurrence except the rise of temperature.

Though the diagnosis of congestion of the lungs is not on the whole difficult, the cases vary widely in respect of prognosis and treatment according to the cause and the conditions under which they develop.

Ætiology.—The forces which are concerned in the circulation through the lungs are—(1) the right side of the heart, which drives the blood into the lungs; (2) the respiratory movements, inspiration aiding the blood in its entry into the thorax, and expiration in its exit; (3) the chemical changes which effect the aëration of the blood; and (4) the left side of the heart which drives the blood from the lungs into the arteries. So long as the normal relation is preserved between these several agencies, whether the lungs be worked much or little, congestion cannot occur. But so soon as the balance is destroyed, or, in other words, so soon as the forces which assist the entry of blood into the lungs are increased, or those which aid its exit diminished, congestion will occur. It will of course be most marked where both causes are combined; for example, where there is on the one side obstruction to the entry of blood into the left ventricle, and on the other hypertrophy of the right heart. This is what occurs in mitral disease, and the chronic congestion which results leads to the characteristic pathological condition known as *brown induration of the lung*.

In many instances the circulation is affected throughout the whole of both lungs, so that the congestion is general. Local congestion, however, might be produced without any increase in the total amount of blood contained in the lungs as a whole, by irregularities in its distribution. For example, when one lung or a part of one lung is prevented, either by external compression or by some intrinsic lesion, from performing its functions adequately, the remaining parts will contain the blood which ought to have been distributed to the whole and thus become congested. This is called *Collateral Hyperæmia or Fluxion*.

So far the causes of congestion mentioned have been chiefly, if not entirely, mechanical. Of the rest the most important group is that in which the congestion is associated with inflammation. Acute congestion is, as already stated, the initial stage of inflammation, while in the later stages the interference with the circulation and with the function of the inflamed parts leads to collateral fluxion in the rest of the lung. Owing to the rapidity with which the congestion develops in these cases, and the urgency of the symptoms which it produces, inflammatory congestion forms a very well-marked and important clinical group. For this reason the causes of congestion could be conveniently classified as *inflammatory* and *non-inflammatory*. This division would correspond closely, though not exactly, with the old-fashioned classification into *active or arterial* and *passive or venous congestion*, the former being also called *acute* and the latter *chronic*.

Of these terms, acute and chronic are the best, if used strictly in a clinical sense, as applying to the symptoms, and not in a pathological sense with reference to the lesion. They would then indicate what is the most striking difference between cases as they are met with at the bedside, for where congestion develops rapidly the symptoms are acute or urgent, and where it develops gradually they are not urgent and may be but slight. Though it is true that in the inflammatory group the symptoms are almost always acute, still it does not follow that in the non-inflammatory group they are always chronic; on the contrary, it not infrequently happens that in such cases where for some time the symptoms have been slight, or chronic, they undergo sudden exacerbation and become acute. Sharply contrasted as the terms acute and chronic are in respect of symptoms, they are not as sharply contrasted in respect of cause, and they are not therefore as useful as a basis for an ætiological classification.

Forms of Congestion.—For the purposes of classification it is best simply to arrange the cases as far as possible in clinical groups. In one group will be placed all those cases in which the symptoms are associated with inflammation, in another those which are more or less obviously the result of mechanical

causes, and in a third group the somewhat miscellaneous remainder which cannot be referred definitely either to inflammation or to any mechanical cause.

I. ACUTE INFLAMMATORY CONGESTION.—The first and most important form of acute inflammatory congestion is that which occurs in connection with *acute pneumonia*. Pneumonia may set in with the signs of general pulmonary congestion. Usually, in the course of a few hours, local signs develop which make the nature of the case clear, and even when they do not, the acuteness of the symptoms and the clinical features of the case are sufficient to establish the diagnosis. In these acute cases the process rarely stops with congestion only, but goes on to exudation, and the physical signs are those of acute bronchitis or even of œdema. In rare cases the exudation is so extreme and the expectoration so profuse that they were placed by Trousseau in the category of œdema of the lung, and were named by him *Pneumonia Serosa*.

It is sometimes observed in cases which set in with signs of acute general congestion, that as the local manifestations appear, the general congestion diminishes. This is analogous to what is seen in the skin, where a widespread initial erythema often rapidly diminishes when a localised lesion develops.

In the later stages, when the local consolidation is fully developed, the general congestion is mainly due to collateral fluxion, *i.e.*, to the derivation of the blood from the parts which are no longer functioning to those which are still competent. It is obvious that collateral fluxion must occur in almost every case of pneumonia, but only in a few does it produce symptoms, unless the characteristic acceleration of the respiration be in part referred to it. When definite symptoms occur they are those of bronchitis or œdema.

In connection with this may be considered a fact of general importance, *viz.*, that congestion and œdema of the lungs is often evidence of their physiological or functional breakdown.

When from any cause extra work is thrown upon the lungs they become hyperæmic, but, so long as both heart and lungs are healthy and the work not more than they can deal with, the blood is sufficiently aerated and the circulation properly carried on, so that in spite of the hyperæmia pathological congestion does not occur. So soon as, from any cause in either heart or lungs, the extra work becomes overwork, *i.e.*, so soon as the demands made upon the lungs are more than they are able to meet, compensation becomes defective, pathological congestion results, and symptoms arise; in other words, the occurrence, in an extra worked lung, of bronchitis or œdema is evidence of overwork, *i.e.*, of physiological inadequacy or breakdown.

The importance of this is recognised clinically and affects both treatment and prognosis. Thus in the case of effusion into the pleura or of ascites the occurrence of the signs of congestion of the lungs becomes an indication for immediate paracentesis. If, as in pneumonia, the cause is irremovable, the prognosis becomes very grave, and a fatal issue is almost certain.

Acute congestion in pneumonia has three possible explanations—(1) Simple general initial congestion, which may subside as the inflammation localises itself; (2) collateral fluxion, which is essentially conservative and compensatory, and (3) functional inadequacy or pulmonary breakdown. The last two stand, as stated, in very close pathological relation to one another.

The second form of acute inflammatory congestion of the lungs is that which accompanies *acute bronchitis*, especially when it involves the small tubes, *i.e.*, acute capillary or suffocative bronchitis. This is, no doubt, the explanation of most of those cases in which acute congestion of the lungs is said to have followed exposure to hot and cold air, to irritating vapours, dusts, etc.

The inflammatory symptoms in those cases are not so marked as in pneumonia, but they are not likely to be entirely absent.

Beyond these two forms of acute inflammatory congestion, it is doubtful if there be any other.

Closely allied with the inflammatory group are those cases in which acute congestion occurs in the course of specific fevers, in some of which it is not an infrequent cause of death. It may be that the high temperature or the poisons generated by the fever affect the lung directly, but without doubt they seriously affect the heart as well, and it is not unlikely that the symptoms of acute pulmonary congestion which arise under these conditions are really due to rapid heart-failure. It is with fevers of a malignant type, and especially when associated with a very high temperature, that death with acute pulmonary symptoms is most frequent, *e.g.*, typhus, malignant smallpox, or malignant scarlet fever, and it is the common mode of death in cases of hyperpyrexia whether in connection with a fever, sunstroke, or acute rheumatism.

II. MECHANICAL CONGESTION.—In this group the congestion is the result of some more or less mechanical interference with the passage of blood through the lungs. It may be acute or chronic, according as the causes which produce it are of rapid development or of long standing. The obstruction may be seated in the left side of the heart, in the pulmonary veins, or in the region of the pulmonary capillaries; if there be no actual obstruction in the heart or vessels, the blood may be kept back in the thorax by abnormal pressure conditions during respiration; for example, by inspiratory obstruction, as in croup. Lastly, all the forms of collateral fluxion are to a great extent mechanical, and the result of the diversion of the blood from the diseased parts of the lung to those still in functional activity.

1. Cardiac Congestion.—This has its origin in the left side of the heart, either in consequence of some impediment to the entrance of the blood into the left ventricle or of some weakness of the left ventricle itself.

There can, of course, be no obstruction equal to that of a left ventricle which does not do its work, and it is in connection with the sudden failure of the left ventricle that the most alarming and acute cases of pulmonary congestion arise. It is not, however, in this group that the changes most characteristic of congestion are found. These occur in the other group, where the obstruction exists to the entrance of the blood into the left ventricle, and the most common and typical instance is mitral disease.

a. Mitral Disease.—Mitral disease in either of its forms is an affection of long standing. To overcome the obstruction the right ventricle hypertrophies and may completely compensate for it. But this compensation takes place at the expense of the lungs, which are placed between two opposing forces, both of which tend to the same result, *viz.*, congestion; for on the one hand the vessels are distended by the blood which is dammed up in them owing to the obstruction in front, and on the other by the blood which is driven into them with increased force by the hypertrophied right ventricle. This leads to the well-known pathological condition of brown induration of the lung or the heart-lung as it is commonly called.

Brown Induration of the Lung—the Heart-Lung.

The two prominent characters of the heart-lung are its colour and the dilatation of its vessels.

The lungs are firm and compact to the touch, heavier than normal and less elastic. The colour is of a dull reddish brown or rusty hue, and is due partly to the abnormal amount of blood it contains and partly to the deposit of pigment. The pigment is of two kinds: first, the common pigment of the lung, which is carbon, and derived from the air; and secondly, a yellowish brown pigment yielding different chemical reactions, and derived from the blood. Both alike

are found partly free in the tissues and partly contained in cells in the interstitial tissue, and also in the epithelium lining the alveoli.

The vessels are dilated, tortuous, and varicose, as is easily seen in the bronchi with the naked eye. With the microscope the most striking changes are observed in the alveoli.

Here the capillaries form prominent loops projecting into the lumen, and are two or three times their normal size.

Traube taught that the lumen of the alveoli, being thus encroached upon, their respiratory capacity was diminished, and thus explained the dyspnoea, but Grossmann¹ has shown that the alveoli walls are stretched by the tense capillaries, and the respiratory capacity thus increased, but at the same time the respiratory excursion is diminished owing to the diminished elasticity or greater stiffness produced, the dyspnoea being due to this cause.

Both in the bronchi and in the alveoli these varicose vessels may burst. After death the signs of hæmorrhage may be visible both in the bronchi and in the vesicles, and no doubt some of the pigment in their walls is derived



Fig. 56.

Section of heart-lung, showing the distension and varicose condition of the capillaries. The vessels have been injected with Berlin blue. Their large size is evident when compared with the alveoli.

from this source. The interstitial tissues are sometimes thicker than normal, but not always, and it may be a question whether fibroid induration is really an essential part of the process at all. Doubtless many of the earlier descriptions of the heart-lung included other conditions which did not belong to simple congestion, *e.g.*, œdema, infarct, and even sub-acute or chronic pneumonia. Certain it is that in many simple cases there is little, if any, interstitial change, which it may therefore be concluded is, if not a secondary and independent, at any rate a subordinate, change.

The congestion of the lung may easily lead to other conditions, *e.g.*, œdema, hæmorrhage, infarct, hypostatic congestion, and even to sub-acute or chronic pneumonia.

The *symptoms* of cardiac congestion of the lungs are for a long time little more than some shortness of breath on exertion. There is a tendency to bronchitis without definite cause, and an attack is more often determined by fatigue than by chill. The expectoration of the dark solid lumps of altered

¹ *Zeitsch. f. klin. Med.*, xvi. 161, 270.

blood which sink in water is almost pathognomonic, but not otherwise of much clinical importance. When the right ventricle gives way and general œdema sets in, the pulmonary symptoms are often relieved and do not become severe again until the end, when chronic œdema and hypostatic congestion develop. If the right heart recover itself under treatment and the general œdema disappears, the pulmonary symptoms may again become marked, and it is under these circumstances at times that fairly large hæmorrhages occur, and cause infarction. I have seen several instances of such infarction of the lungs, when the general signs of right ventricle failure were passing off. Serious as such a complication is, its gravity is not as great as might be thought. I can recall many cases in which, in spite of extensive infarction, recovery progressed favourably.

b. Weak Left Ventricle.—When the cause of the obstruction lies in a weak left ventricle the changes in the lungs are hardly ever so marked as those just described. Congestion must be of long standing to develop the heart-lung, and the duration of these left ventricle cases is comparatively short, to be measured by weeks rather than years. Besides all this, the nutrition of the whole heart suffers, so that there is not the hypertrophy and overaction of the right heart to intensify the congestion. No attempt being thus made to compensate for the obstruction, the congestion makes itself felt at once in the right auricle and systemic veins, to the relief of the lungs.

When the congestion travels beyond the right ventricle, one of the first sets of veins to feel its effects is the coronary. In this way the circulation of the whole heart is affected, and its nutrition suffers still more. The weakness of the left ventricle is thus further increased, and therefore the congestion produced by it, and so a vicious circle is established which, if not broken, quickly leads to a fatal issue. The same thing happens with mitral disease when the right ventricle begins to fail, so that symptoms which have been for a long time chronic may then rapidly become urgent, and upon the signs of chronic congestion be grafted those of acute congestion with all its consequences.

The most severe cases of acute congestion, which are not of inflammatory origin, are met with in connection with sudden failure of the left ventricle, and of this the three commonest causes are high temperature, acute specific fevers, and some poisons. Acute congestion of the lungs is the common mode of death in hyperpyrexia, whether the result of rheumatic fever, sunstroke, or other cause. In malignant specific fevers, though congestion of the lungs is a common cause of death, the symptoms do not depend upon the temperature alone, for they may arise where the temperature is not very high, as, for example, in diphtheria. The cause must then be looked for in the direct toxic effect upon the heart or heart centres of some poison produced by the fever, and although it cannot be proved that these poisons do not affect the lungs directly, still the most simple explanation seems to be that the symptoms depend upon the failure of the heart.

The same causes which, if intense, produce peracute symptoms, if less acute cause acute or subacute, sometimes only chronic symptoms. Thus in typhoid fever the general exhaustion may express itself as a gradually increasing bronchitis, which often yields readily to stimulants and digitalis, thus showing the symptoms to be really cardiac.

In all cases where the left ventricle is weak, and especially if the signs of sub-acute or chronic congestion be present, the symptoms may at any moment become urgent, and often from very slight causes. The best instances of this are

met with in cases of organic disease of the left ventricle—for example, in aortic disease, of which the following is a good instance.

A man aged 40 was admitted into the hospital with double aortic disease. He was ill and the heart was dilated, but he presented no alarming symptoms at the time of admission. He was put to bed, and remained comfortable until the evening, when, about 9 o'clock, without obvious cause, he was seized with severe dyspnœa, the heart became greatly embarrassed in action and still further dilated. The patient began to spit up some frothy fluid mixed with blood; this rapidly increased in amount and became more blood-stained, until in a very short time he was expectorating pure bright blood in considerable amount. The dyspnœa and distress became extreme, the patient grew rapidly more and more cyanosed, and it was evident that he was dying. The view was taken that the symptoms depended upon the sudden failure of the left ventricle, and acting on that opinion I bled the patient from the arm, taking about 25 ounces of blood. As the blood flowed the colour improved and the urgency of the symptoms subsided, so that in an hour the patient was out of danger.

He lived for more than three months without any similar attack, and then died gradually of exhaustion.

2. Obstruction in the pulmonary veins could only be produced by pressure upon them from without or by thrombosis within. *Thrombosis of the pulmonary veins* is hardly ever primary, but almost invariably the result of the direct extension into the veins of an old clot formed in the auricle. It is not a disease of itself but the end stage of many, especially of those in which death has been the result of slowly-increasing and long-continued asthenia, *e.g.*, malignant disease or phthisis. In such cases the auricle and ventricle are often found to contain a firm decolourised clot flattened so as to form a cast, as it were, of those cavities at the end of their systole. A narrow band of clot often connects together the auricular and the ventricular portions, and from the auricle prolongation it may extend into the pulmonary veins for a considerable distance. This is not a rare condition *post-mortem*, but there are no means by which it can be recognised with certainty during life or distinguished from the congestion due to a failing heart; in fact, the two conditions are usually associated together.

3. Obstruction in the pulmonary capillaries in order to produce symptoms must be widespread or general. Again the mechanical interference with the capillary circulation in the lungs is the explanation of the chronic congestion in the bronchi met with in emphysema and in some of the fibroid affections of the lung, while acute congestion may be caused by capillary embolisms if they be sufficiently numerous. Of this the best instance is the fatty embolism which is sometimes the cause of death after fracture of bones.

4. Inspiratory Obstruction.—When there is serious obstruction to the entrance of air into the air tubes the violent inspiratory efforts made to overcome it, if they fail to expand the air vesicles, distend the blood-vessels, for the blood is sucked into them and retained there.

In this way are produced the collapse and congestion of the lower parts of the lung in laryngeal obstruction, and of the irregularly scattered patches in bronchitis.

5. Collateral fluxion is in most cases, no doubt, mechanical in the main, the blood which ought to have been sent to the whole lungs passing only through a part of them. Of course many cases of collateral fluxion are instances of physiological and compensatory hyperæmia rather than of pathological congestion, but the one easily passes into the other.

Collateral fluxion may be *acute* or *chronic*, as the causes which have produced it are sudden or of gradual development. Thus in pneumothorax the severity of the symptoms is, no doubt, in part due to the sudden diversion of the blood to

the one side. If life be preserved, and the lungs have time to adjust themselves to the altered conditions, the symptoms lose their urgency. The same holds good also with extensive pneumonia or a rapidly developing pleuritic effusion.

With embolism of the main branches of the pulmonary artery the symptoms that result are not due to congestion but to the absence of blood, and the patient dies rapidly of suffocation, not for want of air but want of blood in the lungs. Where the embolisms are small some of the symptoms are doubtless due to collateral fluxion in the remaining parts.

III. CONGESTION DUE TO MISCELLANEOUS CAUSES.

—In this group are placed a number of miscellaneous cases, the causes of which are indefinite, *e.g.*, rapid cooling of the skin, vicarious menstruation, the action of certain poisons, hysteria and emotion, puberty, etc.

The pulmonary congestion caused by a *sudden chill* is nothing more than acute bronchitis.

Of *vicarious menstruation* we know little or nothing. The very existence of such an affection is denied by many authorities, and doubted by many more.

Certain drugs, like nitrite of amyl and nitroglycerine, produce general vascular dilatation, in the lungs of course as in the rest of the body, but they rarely cause any pulmonary symptoms.

In individuals with special idiosyncrasies certain drugs or articles of diet lead to attacks of dyspnoea, which, on the analogy of the erythemas produced by similar causes in the skin, are referred to pulmonary congestion, but without any proof or demonstration.

Acute fevers and *high temperature*, and probably *uræmia*, act rather through the heart than upon the lungs direct, and this explanation Cohnheim gives of the congestion caused by *carbonic acid poisoning*, for he regards carbonic acid as a powerful cardiac poison.

The difficulties of respiration met with in *hysteria* and under *emotion* (puberty, if it has any effect at all, must act also through the emotions) are referred by some writers to congestion of the lungs, but it is more likely that they are primarily of cardiac origin.

Acute pulmonary symptoms may develop without definite cause during grave illness, and sometimes in persons who are fairly well at the time. They have been described as acute *pulmonary paresis or paralysis*. It has been suggested that the symptoms are due to sudden loss of pulmonary tone or tension, and that the condition is neuro-muscular in origin, and strictly analogous to the acute paralytic distension of the intestines, described as acute tympanites.

It is, of course, theoretically possible that congestion of the lungs could be produced by vasomotor inhibition, excited by various reflex stimuli, and perhaps some of the causes lately mentioned act in this way, but of the vasomotor nerves of the lung we know little, and we have no proof that any reflex paralysis of them could give rise to the symptoms of pulmonary congestion.

The blood pressure in the pulmonary artery is independent of that in the carotid, and the causes, direct and indirect, which affect it in the carotid fail to affect it in the pulmonary. The only positive observations forthcoming are the following:—

1. Direct irritation of the medulla raises the pressure in both vessels.
2. Suffocation raises the pressure in the pulmonary artery, but leaves that in the carotid unchanged, or causes it to fall.
3. Stimulation of the distal parts of the divided vagi is without effect.
4. If the spinal cord be divided at the level of the seventh dorsal vertebra, irritation of the proximal portion as well as of the medulla causes the pressure to rise in the pulmonary artery, while irritation of the distal portion has no effect upon it.

From these observations it has been concluded that the lungs have vasomotor nerves, and that they travel from the medulla down the cervical cord and gain access to the lungs through the upper seven dorsal nerves.¹

It is clear that so long as the theory of the vasomotor innervation of the pulmonary arteries rests upon no more conclusive evidence than that so far given, it would be rash to attempt to explain the symptoms of disease by it.

HYPOSTATIC CONGESTION.—Where congestion lasts some time, or is due to some chronic cause, the action of gravity comes into play, and the results are to be most marked in the lowest parts of the lungs, *i.e.*, in patients lying in bed, at the bases behind. To this condition the term hypostatic congestion is applied. As in the legs under similar circumstances, so in the lung, exudation takes place from the vessels, and hypostatic congestion passes into *hypostatic œdema*. The exudation slowly drives out the air and takes its place, so that the parts become airless and sink in water, and when the condition has persisted for some time the parts become so firm in consistence as to deserve the name *solid œdema*, which has been given it. A lung in these conditions offers a very suitable soil for the development of any of the germs of inflammation if they gain access to it, and so it is not uncommon for inflammation to arise in the hypostatic parts, giving rise to the so-called *hypostatic pneumonia* (*q.v.*).

In the earliest stage there is little to be seen in the affected parts of the lung except dilatation of the vessels in the alveolar walls. Then follows later the exudation of serous fluid into the air vesicles and small tubes, rendering the parts firmer and less crepitant. The section is much more juicy than natural, and the air-tubes contain some frothy clear fluid. If the exudation continue, the air is in the end driven out of the air vesicles entirely, so that the parts become doughy and pit on pressure, cease to be crepitant, and sink in water on section. Blood-stained serum runs freely away from the surface, and the bronchial tubes are filled with similar frothy fluid. Microscopical examination shows that the alveolar epithelium has been shed, and the swollen cells are seen lying in the alveoli, and with them a few blood cells, both red and white. In this stage the parts have a grayish translucent colour and look, as they are, œdematous. If more blood cells are exuded the parts have a redder colour, and from its resemblance to a section of spleen the name "*splenisation*" has been given to this condition. Sometimes actual hæmorrhage may occur, usually only in small patches, but if of greater extent it may resemble and indeed amount to an infarct. Extensive hæmorrhage is rare except in connection with morbus cordis, but some blood is found in almost every case, and the difference is one only of degree. Œdema of the lung in the strict sense is not present, for the interstitial tissue, though it may be a little swollen, is not otherwise altered, and the exudation is intra-alveolar and not interstitial.

Hypostatic congestion is as a rule a bilateral affection, *i.e.*, both bases are involved, but not necessarily to the same extent. Indeed, as Piorry showed, the right is usually more affected than the left, but something will depend upon the position in which the patient has been accustomed to lie, and if for any reason the position has been entirely upon one side, the congestion may be also unilateral. Hypostatic congestion varies greatly both in extent and degree from simple vascular congestion up to solid œdema. Commencing at the bases behind it slowly spreads upwards, and may involve the lower half, or even more, of both lungs.

The **physical signs** vary according to the amount of the lung involved and the stage of the lesion. So long as there is nothing but vascular congestion physical

¹ Cf. Lichtheim, *Die Störungen des Lungen Kreislaufes*, 1876; Badoud, *Würzburg Verhandl.*, 1874; Frey, *Die pathol. Veränd. d. Lungen nach Lähmung der N. Vagi*, 1877; Bradford, *Journ. of Physiol.*, vol. x.

signs will be absent. When exudation has occurred the signs will be those of bronchitis, viz., rhonchus, sibilus and crepitation, but so long as the vesicles still contain air the percussion will not be dull. So soon as the air is all driven out from a sufficiently large patch the percussion becomes dull, and as the tubes are filled like the vesicles with fluid, the breath and voice sounds will be diminished or absent. The diagnosis from effusion into the pleura then becomes difficult, and may be impossible without the use of the exploring needle, but the two conditions are not infrequently associated. In an advanced case all the stages may be found at the same time; at the base, solid œdema with the signs described; above, the signs of bronchitis with crepitation; and still higher, congestion only.

If pneumonia develop, the signs of consolidation may appear, but, owing to the obstruction of the tubes, the characteristic signs are more frequently absent, and the physical signs are so misleading that the diagnosis of pneumonia is by no means easy. Even the temperature is often no help, for it is often raised but little, and sometimes not at all.

The symptoms vary greatly, chiefly in relation to the rate at which the lesion has developed. If, as is usually the case, the development be slow and gradual, the symptoms may be slight and indefinite, nothing more perhaps than a slight acceleration of the respiration. For this reason it should be the rule, in all asthenic cases in which hypostatic congestion is likely to arise, to examine the bases of the lungs at regular intervals, otherwise extensive mischief may be found when not in the least anticipated. If the development be more rapid, as in the case of acute fevers, the attention may be drawn to the lungs by the shortness and rapidity of breathing, and especially by the dusky colour of the complexion. The temperature is not raised unless inflammation develop, but even when acute pneumonia sets in, the access and fever are rarely so marked as under ordinary circumstances.

The Prognosis of hypostatic congestion varies with the conditions under which it has arisen and the cause upon which it depends, and with the means we have of relieving the one and removing the other.

The Treatment also varies in the same way. The chief immediate cause is weakness of the heart, and that must be dealt with in the ordinary ways, by good feeding, tonics, and cardiac stimulants. Of these, digitalis and strophanthus are very useful, but I think that citrate of caffeine is better than either, especially when administered *sub cutem*.

The other prime cause is gravitation, and a great deal may be done in patients confined to bed to obviate this by frequently changing their position, so that the same part of the lung should not constantly be the lowest, shifting them first on to one side and then on to the other. This change of pressure, which is recognised as so necessary to avoid bedsores, is equally important to avoid congestion of the lungs.

Grave as the prospects of hypostatic congestion are when the affection has reached a high degree, and the causes are irremediable, still in the lesser degrees much may be done to relieve or to remove it; but prevention is in all cases better than cure, and treatment of the conditions under which it is so likely to arise, may often, by anticipation, prevent its occurrence.

36. ŒDEMA—SEROUS INFILTRATION (Laennec) —TRANSUDATION—INUNDATION (Rokitansky).

Œdema of the lung is generally the consequence of congestion, and differs from it only in the exudation which is present, *i.e.*, in the tissue proper of the lungs or outside the lung tissues in the vesicles, and accordingly œdema has been divided into interstitial and alveolar, but such a distinction is pathological rather than clinical, and is without practical importance. Like congestion, œdema may be acute or chronic, inflammatory or not.

It is an extremely common pathological condition in the *post-mortem* room, having developed during the agony. In these cases, as Cohnheim said, the patients do not die because of the œdema, but the œdema develops because the patient is dying.

Chronic Œdema.—Chronic œdema arises under the same conditions as chronic congestion, of which it is only a later stage. When fluid is once effused into the lung it tends to gravitate to the lowest parts, and the disease is then called *hypostatic œdema*.

Hypostatic congestion is the term frequently applied to both congestion and œdema; in other words, hypostatic congestion is hardly ever simple, but is a mixture of congestion and œdema. Yet clinically the conditions should be kept distinct, for, as we often see in mitral disease, congestion may exist for a long time without œdema, and then with no very obvious cause œdema may rapidly develop.

The explanation of this is not clear. Some writers maintain that the exudation is due to a change in the vessels, by which they are rendered more porous and leak, and that it is not until this change has taken place that œdema occurs. If this were so the transition from congestion to œdema should be more constantly gradual than it is, and not so rapid and acute as it frequently happens to be. Another explanation seems to me much more probable, *viz.*, that the œdema is brought about by an increase in the obstruction offered to the circulation, and in the great majority of cases to an increased feebleness of the left of the heart. Distended vessels, we know, allow of the passage of fluid from them, and it has been shown experimentally that congestion leads to a much increased flow of lymph from the lymphatics at the root of the lung.¹ It is therefore probable that, so long as the flow does not exceed certain limits, the lymphatics are competent to deal with it and carry it off as fast as it is effused. When the exudation exceeds what the lymphatics can deal with in the given time, it passes readily out of them into the vesicles or bronchi, partly by direct transudation and partly through the stomata. When the lymphatics are working up to their maximum a very slight cause may be sufficient to lead to their incompetency, and thus determine exudation or œdema.

Conversely, if the œdema be only slight a slight change in the general or local conditions may lead to the disappearance of the œdema which had developed. This theory harmonises best with experience at the bedside, for there is no doubt that many cases of slight œdema are recoverable.

Acute Œdema.²—All the causes which produce chronic œdema when they act slowly or operate for a long time, may produce acute œdema if they act rapidly; but when œdema develops gradually, gravity comes into play and leads to hypostatic œdema; when it develops rapidly gravity has not the same effect, and the œdema is more often general.

The causes of acute œdema may be roughly classified in the following way:—

- | | |
|--|---------------------------|
| 1. Acute inflammation. | 5. Certain poisons. |
| 2. Acute collateral fluxion. | 6. Hyperpyrexia. |
| 3. Sudden mechanical obstruction to the vessels. | 7. Acute nephritis. |
| 4. Acute fevers. | 8. Paracentesis thoracis. |

¹ Welch, *Virch. Arch.*, lxxii.

² Acute œdema has been called *apoplexia pulmonum serosa* (Stickfluss), in contradistinction to *apoplexia pulmonum vascularis*, which is the name suggested for acute congestion.

Most of these causes have been spoken of as producing acute congestion, but, as stated, acute congestion is always attended with exudation if it lasts long enough. Indeed, clinically the term acute congestion is applied to cases in which some exudation has occurred, but without copious expectoration, and œdema is often reserved for those cases in which there is profuse expectoration. It is obvious that the distinction is purely artificial.

Taking profuse expectoration of a watery fluid as the clinical criterium of acute œdema (the pathological criterium it certainly cannot be), the affection is very rare, chiefly, no doubt, because the patients die before expectoration occurs; in other words, because the patients have not the strength to cough the fluid up, and die suffocated by it.

Acute inflammatory œdema may be local or general, primary or secondary.

If general, it cannot be distinguished from acute capillary or suffocative bronchitis, and, like it, is often rapidly fatal. If local and primary, it cannot be distinguished from the inflammation of which it is only the intermediate stage.

Acute œdema may develop as a secondary affection in the course of acute inflammation of the lungs as the consequence of collateral fluxion. The signs are those of acute bronchitis in the rest of the lung. They develop rapidly and are of serious import. It is a grave condition at all times, even if it involve only the rest of the affected lung, but when it attacks the opposite or sound lung as well it is almost invariably fatal. The gravity of the signs of bronchitis when associated with pneumonia varies according as it precedes or follows the pneumonia. In the former case it aggravates the pneumonia only, but in the latter it indicates functional breakdown of the lungs, and is, with hardly an exception, fatal.

Another form of acute collateral fluxion occurs as the result of sudden mechanical obstruction to the vessels, as in thrombosis of the main pulmonary veins, or of embolism or thrombosis in many vessels of the lung.

Acute fever, hyperpyrexia, and certain poisons, act, no doubt, chiefly by causing rapid failure of the heart. Their action has already been considered when speaking of the causes of acute congestion.

In acute nephritis, again, it is probable that many cases are due to cardiac failure, but others may be referred to toxic causes (uræmia) or to the same causes which produce general anasarca. Acute œdema is a rare complication in any form of Bright's disease, although chronic œdema is common enough in the later stages.

During the administration of æther as an anæsthetic, profuse discharge of muco-serous fluid occasionally gives rise to alarming symptoms and seems to threaten life from suffocation. It is not often, however, that the patients die of it, and it is probable that this discharge comes rather from the air-tubes than from the vesicle.

One of the most remarkable and interesting forms of œdema is met with occasionally, during or immediately after paracentesis thoracis (*bronchorrhœa serosa*). It is a rare event, and hardly occurs in more than a fraction per cent. of paracentesis cases.

Though there is an extremely copious discharge of clear fluid, it is attended with much less grave symptoms than its amount would lead one to expect, and is hardly ever fatal. This is due to the fact that it comes entirely from one lung and not from both. It is usually attributed to the rapid expansion of the lung which has been long compressed, the blood-vessels becoming suddenly distended with blood and exudation resulting. If this be the explanation, it is difficult to understand why it does not more frequently occur. I have performed paracentesis, and seen it performed, under all sorts of conditions and in all sorts

of ways ; the fluid being removed very rapidly or very slowly ; under considerable suction or with hardly any suction at all ; in cases where the lungs have been bound down by extensive adhesions, or where they were free to expand without hindrance ; yet I have seen this profuse discharge in one or two cases only, and in these I could find no satisfactory explanation of its occurrence.

In my last case the patient was a fairly healthy young man with a large effusion of no long standing. The paracentesis was performed in the usual way with very little suction. The operation was completed without the occurrence of any special symptoms, but within a few minutes the discharge began and lasted for two hours, during which time about two and a half pints of clear watery fluid were expectorated without any difficulty, and without any grave symptoms or urgent dyspnoea. It came entirely from the recently expanded lung, and ceased almost as suddenly as it began. The patient made a rapid and good recovery.

An interesting case, which, however, comes into a different category, has been recently published by Dr Calvert, in which nearly a fortnight after paracentesis a serous discharge commenced and lasted for nearly four months, in the end ceasing gradually and the patient making a good recovery. The fluid came from the upper parts of the lung on the affected side. Calvert suggests, as seems most probable, that it was due to adhesions, which, as the lung expanded, compressed the pulmonary veins coming from the upper lobe.

Treatment.—The general treatment of acute œdema, as of acute congestion, will vary with the cause. But whatever this may be, where the symptoms are urgent the heart shows signs of rapid failure, and thus both cardiac and general stimulants will be required, while the rapidly-increasing cyanosis suggests the mechanical relief of the congestion either by dry-cupping or venæsection. If dry cups to the back of the chest prove, as they probably will, ineffectual, the question of bleeding arises. Bleeding with the object of relieving the lungs and the distended right side of the heart is not a method of treatment to be played with. It must be free or not at all. Twenty or thirty ounces of blood at least must be rapidly removed, and the advisability of such active treatment will have to be determined by the consideration whether the general strength of the patient is such as to stand the loss of so large an amount of blood. In some cases bleeding is, however, obviously the only resource, and its risks must be faced. I can recall several cases in which timely bleeding in these desperate cases has had fortunate results, even when success seemed hardly to be hoped for.

ACUTE INFLAMMATION OF THE LUNG.

The acute inflammatory consolidations of the lung called pneumonia fall into two broad groups—in the one the consolidation is of a considerable extent individually, but localised ; in the other the consolidations are numerous, small, and widely disseminated.

The former, because it involves usually the whole of a lobe, or more, is called lobar pneumonia ; the latter, from its irregular dissemination, is called patchy, and because it usually involves the terminal bronchi and the corresponding groups of vesicles, lobular.

As these two groups stand in correlation with other marked pathological and clinical peculiarities, they form a useful and natural classification ; yet it is obvious that the classification must not be pressed too far, as there will be many cases which it will be difficult to assign to their proper category.

A more scientific and accurate classification would be based upon the pathogenic organisms which have excited the inflammation, and thus we might have pneumonia caused by pneumococci, streptococci, staphylococci, Friedländer's bacillus, the bacilli of tubercle, anthrax, and those of putrefactive processes, etc., and it may be that we shall in time be able to recognise these different

forms by their clinical peculiarities, but at present science has not advanced so far.

Accepting the classification into lobar and lobular as useful, so far as it goes, we must recognise at the outset that either of these forms of pneumonia may be produced by various pathogenic organisms, and, again, that the same pathogenic organism under certain circumstances may produce either form of pneumonia; in other words, that lobar pneumonia, though commonly caused by a pneumococcus, may occasionally be due to other pathogenic organisms, and that pneumococci, though commonly producing lobar pneumonia, may under certain circumstances produce the lobular form.

37. ACUTE PNEUMONIA, CROUPOUS PNEUMONIA, LOBAR PNEUMONIA, PLEURO-PNEUMONIA.

This is the common form of acute idiopathic inflammation of the lung in the adult.

ÆTIOLOGY.—Lobar pneumonia is one of the most widely extended as well as one of the most fatal diseases. It is distributed over the face of the whole globe, and there is no climate or race which is free from it.

Frequency.—Its frequency is very great. It is said to constitute 3 per cent. of all diseases and 6 per cent. of all medical diseases, and to account for 6·6 per cent. of all the deaths in the one case and 12·7 per cent. in the other. Hirsch states that the average mortality from pneumonia is not less than 1·5 to 2·3 per 1000 persons living.

Copenhagen¹ statistics give the incidence rate for pneumonia as 6·2 per 1000 living, and the death-rate as 1·2. The number of cases occurring in the Austrian army for ten years averaged 1·14 per cent. per annum; in the Prussian army for seven years 1·15 per cent., but the morbidity varies much in different years. Thus it was 0·93 per cent. in 1878–9 and 1·55 in 1874.

The statistics of St. Bartholomew's Hospital² show that in the six years 1881–6 pneumonia formed 5 per cent. of all the medical cases admitted into the wards, and caused 6 per cent. of the total medical deaths, while of the respiratory diseases alone it constituted 26 per cent. and caused 21 per cent. of the deaths.

These years were selected as being before the prevalence of influenza.

Seasons.—The frequency varies a good deal in different places, and in the same place at different times, but in all places alike it is most prevalent at the breaks of the year, and in this respect runs to some extent parallel with bronchitis. In this country and in the western parts of Europe the winter and spring are the seasons of greatest prevalence, about two-thirds of all the cases occurring in this half of the year, the remaining third being nearly equally divided between the other two quarters.

The cases of pneumonia³ occurring in London in the year 1893 were thus grouped—

January to March,	30	per fortnight.
April, May, and June,	62	„
July to October 21st,	27½	„
Remaining ten weeks,	16	„

¹ *Virch. Jahrb.*, 1888, i. 332.

³ Herringham, *B. M. J.*, 12th May 1894.

² *Ibid.*, 1884, 562.

The following statistics are given by Seitz for Munich, by Jürgensen¹ for six large German towns, and by Sturges² for Westminster Hospital:—

	Seitz.	Jürgensen.	Sturges.
Winter, . . .	32.2 per cent.	33.1 per cent.	30 per cent.
Spring, . . .	36.8 „	33.1 „	34 „
Summer, . . .	15.3 „	16.1 „	14 „
Autumn, . . .	15.7 „	17.7 „	22 „

It is impossible to avoid connecting the prevalence of pneumonia at these seasons with the violent changes of temperature so common then, and this

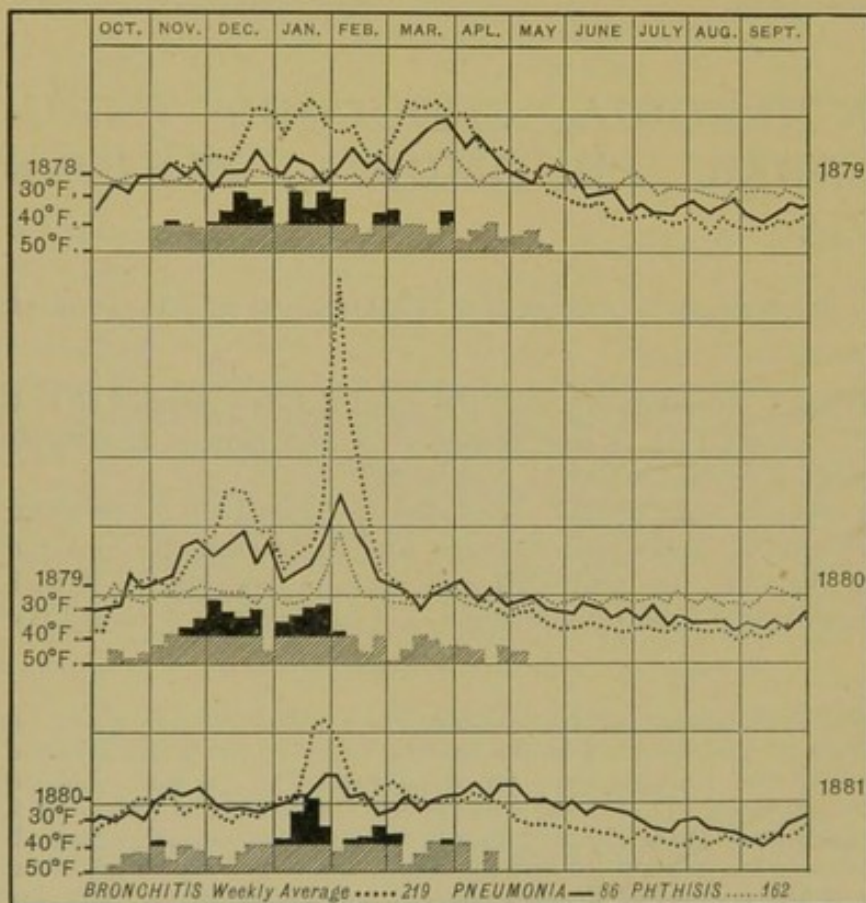


Fig. 57.

Weekly deaths from bronchitis and pneumonia in London (Longstaff, statistical enquiries). The grey shaded areas indicate mean weekly temperatures below 50° F. The black indicate mean temperatures below 40° F.

Beyond the cold winds, dampness and changes peculiar to this season, other meteorological conditions are without influence.

It has been suggested that pneumonia stands in relation to the level of the subsoil water, becoming prevalent when the level is low and thus following a period of diminished rainfall. The evidence is not conclusive, but we may perhaps bring into this connection the instances in which outbreaks of pneumonia have followed the drying up of marshes in the spring and summer.³

Simple exposure to cold or even severe weather does not of itself increase the liability to pneumonia, for those suffer least who live most in the open air. Thus sailors are said to be less liable at sea than at home, and soldiers in the field

conclusion is supported by the close relation which exists in respect of the influence of season between pneumonia and bronchitis; but, as Longstaff has shown, although the mortality curves of bronchitis and pneumonia establish the general relationship between these two diseases, there are minor variations in them which indicate variations in their causation, and this is confirmed by the fact that the greatest prevalence of pneumonia falls at a rather later period than that of bronchitis, bronchitis being most frequent in January and February and pneumonia in March, April, or May.

¹ *Bayr. Ärtzl. Intell. Bl.*, No. 33, 1884.

² *Tham, Virch. Jahrb.*, 1886, ii. 120.

³ *Loc. cit.*

than in garrison, and pneumonia is undoubtedly more prevalent among the urban than among the rural population. These facts tend to disprove the statement frequently made that pneumonia is quite as likely to attack the robust as the weakly. It is true that the robust often succumb, but it is also generally true that even the robust have not been in their usual health just before their attack. Thus pneumonia often follows a period of overwork, fatigue, or worry, and is not uncommon in the depression which follows an accident or confinement within doors, and this without any other assignable cause than the general ailing produced in these ways.

Sex.—Men are attacked more frequently than women in the proportion of at least 2 to 1.

The statistics of St. Bartholomew's Hospital for nine years show 660 men to 265 women, a ratio of 2.5 to 1, and the Collective Investigation Records give a ratio of 2.1 to 1, but Bary,¹ analysing 23,000 cases, obtains a still higher ratio, viz., 3.6 to 1. Jürgensen states that with advancing age the difference between the sexes becomes less marked and finally reversed, but the statistics at St. Bartholomew's Hospital do not bear this out:

Age.—No age escapes, but the general impression is no doubt correct that pneumonia is most frequent in early middle life. In little children, before 2 or even up to 5 years of age, pneumonia is most commonly of the catarrhal form, but the difficulties of diagnosis makes the statistics for these ages somewhat unreliable, and it not rarely happens that a case which has been diagnosed as croupous pneumonia during life turns out after death to be catarrhal.

The following figures are obtained from an analysis of 925 cases at St. Bartholomew's Hospital. For simplicity they are calculated out in percentages:—

Under 5 years	=11.6	From 30 to 40 years	=13.7
From 5 to 10 "	=11.5	" 40 " 50 "	=10.0
" 10 " 15 "	=10.0	" 50 " 60 "	=0.3
" 15 " 20 "	=15.6	Over 60 "	=0.1
" 20 " 30 "	=23.5		

These figures show that pneumonia rises rapidly in frequency up to the age of 30, and then declines rapidly again, but it must be borne in mind that the decline is not so great as it seems, for the number of persons living at these ages becomes also rapidly less.

The specific fevers, notably measles, diphtheria, and enteric, are often said to increase the liability to pneumonia, but it is really doubtful whether pneumonia is more frequent after these fevers than after ill-health in any way produced. With influenza the case is different, for there is no other disease which has anything like the same effect upon the frequency and fatality of pneumonia. This will be referred to again later.

Of the exciting causes that most popularly given is chill, but patients often do not distinguish the rigor and chills with which the attack begins from the chilliness due to exposure to cold. According to Jürgensen, pneumonia does not follow a chill due to exposure to cold in more than about 12 per cent. of all cases.

Where the attack is referred to emotion, fright, strain, and other indefinite causes, the association is clearly accidental.

The relation in which pneumonia stands to inflammation adjacent to the lungs, to injury, to pulmonary congestion, embolism and infarcts, to tuberculosis and other general diseases, will be considered in other places.

¹ *Viert. Jahrbr. f. gerichtl. Med.*, N.F., xxxix. 104.

THE BACTERIOLOGY OF PNEUMONIA.

Historical Review.—To Klebs¹ belongs the credit of initiating research in this direction. He described in 1877 a round mobile micrococcus as present in the lungs, sputum, and blood of pneumonic patients. In 1881 Eberth² described another similar but different micrococcus, and in 1882 Koch discovered still another which had an oval form. It was Friedländer's³ investigations, however, in 1882 that fixed attention on the subject. He discovered the peculiar microbe which goes by his name, and was successful in its cultivation and inoculation, though the pneumonia produced was disseminated in patches and not lobar. His observations were for the time regarded as conclusive, until the appearance in 1884 of the work of Fraenkel⁴ and Weichselbaum.⁵ Fraenkel described another microbe which proved to be much more frequent in pneumonia than that of Friedländer. Of this also successful cultivations and inoculations were made. Lastly, Klein,⁶ in 1890, in a peculiar outbreak of pneumonia found a new and entirely different bacillus from those hitherto described.⁷

The investigations of Weichselbaum and others showed that though the pneumococcus was by far the commonest microbe, yet it was not the only one.

Thus, out of 124 cases the pneumococcus was found in 94, the pneumo-bacillus in 9, and in the remaining 21 some other germ, *e.g.*, streptococcus, staphylococcus, etc. Wolf⁸ found the pneumococcus in 66 out of 70 cases, and Netter in 75 per cent.

Since then Netter and Klemperer,⁹ examining the juice obtained by a needle puncture from the lung during life, in 82 and 21 cases respectively found the pneumococcus in every one.

∴ We may conclude, therefore, that though the pneumococcus is the pathogenic organism of the ordinary or idiopathic croupous pneumonia, many other organisms also can excite an acute inflammatory infection of the lung, which so far we have no clinical means of distinguishing from pneumococcus pneumonia, *e.g.*, the pneumo-bacillus, *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, *Bacillus tuberculosis*, *Bacillus anthracis*, and possibly many others.

Perhaps the peculiar course which many cases of pneumonia run may depend upon the nature of the infecting germ or upon some mixed infection, and some day we shall be able to recognise these different forms of pneumonia by their clinical signs, but at present science has not progressed so far.

In 76 cases¹⁰ of pneumonia the pneumococcus was found associated with other germs with the following frequency:—

With <i>Staphylococcus aureus</i>	25 times, alone in 20.
	With <i>Streptococcus</i> in 3.
	With Friedländer's bacillus in 2.
With <i>Streptococcus pyogenes</i>	9 times, alone in 5.
	With <i>Staphylococcus</i> in 3.
	With Friedländer's bacillus in 1.
With Friedländer's bacillus	7 times, alone in 4.

The two most important and best known microbes of pneumonia are those of Friedländer and Fränkel, called respectively the *Bacillus* and *Diplococcus pneumoniae*, and of these a short account may be given.

Bacillus pneumoniae (Friedländer's bacillus).—This was originally described by Friedländer¹¹ as a coccus owing to the rarity of the bacillus form in the lung. These microbes were found in the hepatised parts, and more in the red than in the gray; frequently in the lymphatics, and in

¹ *Arch. f. exp. Path.*, 1877, vol. iv.

² *D. Arch. f. klin. Med.*, 1881.

³ *Virch. Arch.*, 1882.

⁴ *Verh. d. Ver. f. inn. Med.*, 1885.

⁵ *Wien. med. Jahrb.*, 1886.

⁶ Supplement to 18th Ann. Rep. of Loc. Gov. Bd., 1888-9, p. 323.

⁷ Cf. for literature of subject, Mendelssohn, *Zeitsch. f. klin. Med.*, 1884. Charcot, *Médecine*, 1893. Sternberg, *Lancet*, 1889, i. 370. Cornil and Babes, *Les Bactéries*. Ziegler, *Pathol. Anat.*, 1890.

⁸ *Comptes Rendus hebdom. de la Soc. de Biol.*, 1887.

⁹ *Ann. de l'Institut. Pasteur*, ii. 8, 1888.

¹⁰ Netter, Charcot, *Médecine*, 1898. Cf. also Kreibich, quoted *B. M. J.*, epitome, Sept. 12, 1896.

¹¹ *Virch. Arch.*, 1882. *Fortschr. d. Med.*, 1883, i. 715; 1884, ii. 333.

one case they were so numerous in the capillary blood-vessels as to produce a varicose condition. They were also found¹ in the pleuritic exudation as well as in the pericardial and other inflammatory complications of pneumonia. They occur singly, in pairs, chains or colonies, and are surrounded by a capsule which forms a common sheath round the groups. The capsule is a hyaline mucin-like substance, soluble in alkalis and insoluble in acetic acid. When treated by Gram's method the bacilli do not retain the stain, in this respect differing from the diplococcus, which does. They grow readily on nutrient gelatine, at ordinary temperatures, and form a glistening porcelain-white button-shaped culture, but in this condition they do not develop capsules. Needle cultivations produce a "nail-culture," which, however, is not characteristic of this bacillus only. They may also be grown upon potatoe. Rabbits are refractory to inoculation. Mice develop a disseminated pneumonia; they die in eighteen to thirty hours, and in the exudations as well as in the blood the capsulated bacillus is found.

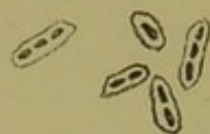


Fig. 58.

Bacillus pneumoniae (+500)
(Friedländer).

Diplococcus pneumoniae (Fränkel's diplococcus).—This was discovered by Fränkel² in 1884, and fully described by him and Weichselbaum.³ It is an oval- or lancet-shaped micrococcus, sometimes naked, but at other times surrounded by a gelatinous capsule. It usually occurs in pairs, but may be in chains or colonies, or even singly. It is found not only in the consolidation and in the sputum, but also in the various inflammatory lesions complicating pneumonia, e.g., in pleurisy, pericarditis, peritonitis, meningitis, otitis, also in endocarditis and nephritis, as well as in inflamed tissues in the throat, neck, and mediastinum, and even in the conjunctiva and in the nose. It has also been found in the splenic tissue and in the blood. It is very common in healthy saliva, and is identical with the organism described by Pasteur, and named by Sternberg *Micrococcus lanceolatus Pasteuri*. It is probably the same as that previously described by Talamon, but by him confounded with Friedländer's organism.

The diplococcus stains readily with Gram's method. It is difficult of cultivation, and will not grow at all on potatoe nor on gelatine at the ordinary temperature. On agar-agar and blood serum it grows, but not freely, at a temperature of 22 C. (71.6° F.); best, however, at the body temperature, and forms on the surface tender, transparent, glistening dewdrop colonies resembling the moisture deposited on a cover glass. The presence of the capsule seems to depend in some measure upon the mode of growth—thus on solidified serum or in broth there is a distinct capsule, but on nutritive gelatine there is none.

Inoculation succeeds in rabbits, guinea-pigs, and mice, the organisms multiplying freely in the blood and serous cavities. Pneumonic consolidation is sometimes produced with hæmorrhagic effusion into the pleura. Rabbits are extremely susceptible to this organism, and die in from thirty-six to forty-eight hours with the symptoms of septicæmia.

The microbes lose their virulence very rapidly if exposed to the air, or if cultivated on milk, or on agar-agar and blood serum, at a temperature of 42 C. (108° F.) for one or two days; in fact, in artificial cultures of all kinds.

Similar diplococci, except that they are of larger size, have been found in the pleuropneumonia of horses, a disease which is eminently contagious (Mendelssohn).⁴

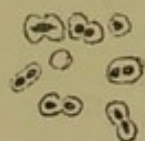


Fig. 59.

Diplococcus pneumoniae (Fränkel)
(+500).

Epidemic Pneumonia.—The occurrence of pneumonia in widespread epidemics or in local outbreaks due to apparent infection assumes fresh importance now that the bacterial origin of pneumonia is established.

Most of our information⁵ on this subject dates from the last twelve or fifteen years, and consists chiefly of accounts of local outbreaks of the disease which have been called epidemics. Of epidemics of pneumonia in the sense in which that term is applied to such diseases as small-pox or scarlet fever we have no recent instance. The descriptions of the old epidemics of pneumonic fever are too vague to be of much value nowadays, and in all probability they were really epidemics of some other disease, such as influenza or typhoid fever, in which respiratory complications were unduly frequent. Thus in 1847 pneumonia was

¹ Cf. Ziegler, *l.c.*

² *l.c.*, and *Zeitsch. f. klin. Med.*, x., Hft. 5 and 6, 1886.

³ *l.c.*, and *Wien. med. Blt.*, 1887, Nos. 10-14.

⁴ Mendelssohn, *Zeitsch. f. klin. Med.*, vii. 178.

⁵ The most important references on this part of the subject are the following:—Mendelssohn, *Zeitsch. f. kl. Med.*, 1884. Sturges and Coupland, *Pneumonia. Coll. Inv. Rep.* Netter, *Arch. gén. de Méd.*, May 1888. Flüggé, *Micro-organismen*.

remarkably prevalent in Europe and America, so that out of nineteen places in Europe which had a continuous mortality record for twenty-one years, the pneumonia mortality reached its maximum in twelve, being very high in the rest, but not reaching its actual maximum until the following year; but this was the very time that influenza prevailed, being indeed the last great epidemic of that disease until that of 1890 and 1891, when the same remarkable prevalence of, and mortality from, pneumonia was observed. In the same way the occurrence of influenza deprives of much of its value the epidemic of pneumonia in Iceland recorded by Hjaltelin,¹ and frequently quoted by writers. Other so-called epidemics of pneumonia have also been associated with the prevalence of typhoid fever, and I do not know of any widespread outbreak which can be regarded as one of pneumonia simply.

What are usually spoken of as epidemics of pneumonia are almost entirely local outbreaks of limited extent, restricted to a district, a town, a village, a group of houses, a street, or a large building. In all cases alike the mere occurrence of many cases in the same locality is no evidence by itself that the affection is transmitted directly from the sick to the healthy, but may, with at least equal probability, be explained as the result of some common cause. Many of these outbreaks can, without doubt, be traced to sanitary defects. Indeed, so close is the connection with insanitary conditions, that the name pythogenic pneumonia has been used by authors to denote what other writers would call epidemic, though of course the former term would apply equally well to those outbreaks affecting a single building, house, or room. In such limited outbreaks it is often easy to trace conclusively their dependence upon defective sanitary conditions.

For example, Dr Gooch,² of Eton, records an instance in which twenty persons were taken ill in one house in the course of three weeks—the butler and two boys with pneumonia, and the rest with sore throats or diarrhoea. The cause was ultimately found in faulty trapping of the butler's sink. This record also illustrates one of the great difficulties in the question of direct contagion, for besides the inmates, one boy was attacked who constantly visited the house, and whose attack might have been quoted as an instance of direct contagion had the defect in the drainage not been discovered.

Similarly in the case of a boys' school at East Sheen,³ the exciting cause was the opening of a ventilator to the main sewer near the house. Here three boys and two servants were taken ill with pneumonia within a day or two of the opening of the ventilator, and one of the servants died. Previous to the opening of the ventilator the house had been healthy, and no further illness occurred when it had been closed. A still more limited outbreak is recorded by Mendelssohn,⁴ in which a coachman, his wife, and three children all got pneumonia one after the other, on removing to a cottage which had been left by the former occupants in a filthy state.

Outbreaks in large buildings have also been in many cases traced to similar causes.

This was the case with the outbreak in the Moringer Reformatory, which Kuhn⁵ referred to overcrowding, and that in the prison at Kentucky, when 14 per cent. of the inmates were attacked in the space of four months, and which Rodman traced to gross sanitary defects.

House Pneumonia.—In this place reference may be made to cases in which pneumonia has appeared to stick to particular houses, and to recur constantly in the same house at irregular intervals.

Thus in one house⁶ during the space of fifteen years pneumonia occurred no less than 31 times, only three years escaping entirely. In another house⁷ in the same period it occurred 16 times, and in another⁸ 8 times in the course of 8½ years.

¹ *Coll. Inv. Rep.*

⁴ *Zeitsch. f. klin. Med.*, 1884.

⁶ Schroeder, *Zu Statistik der Cr. Pneum.*, Kiel, 1882.

⁷ Jürgensen, *Cr. Pneum.*, 1883.

² *Ibid.*

⁵ *Berl. klin. Woch.*, 1888, No. 17.

⁸ *Ibid.*

³ Botan, *D. med. W.*, 1887.

The difficulties are, of course, much greater in tracing to the proper cause the outbreaks in large districts, such as streets, villages, or towns, but even here the possibility of sanitary defects being the real cause cannot be dismissed without due consideration.

Banti records an outbreak in Florence which he regarded as pythogenic, and which closely resembled typhoid fever, but in the fatal cases no typhoid lesion was found.

Still in this connection it must be remembered that it *may* be true, as Gerhardt has asserted, that typhoid fever may attack the lungs alone while the intestines escape, though for myself I must say that I have not met with any case which supports such a view.

So far, then, there is little evidence forthcoming that pneumonia is transmitted by direct contagion. Many of the authors, indeed, who describe these epidemics expressly state their opinion that the cases were not in their experience infectious.

Direct infection.—The evidence in favour of direct infection is very scanty.

Thus Netter,¹ a thoroughgoing contagionist, can only find recorded 30 cases which he regards as conclusive. Out of the 1065 cases upon which the report of the Collective Investigation Committee is based, only 20 support the theory of contagion, and some of the cases quoted as proof of contagion admit of other interpretations, many of them being instances of house-pneumonia limited to a house or room. In Patchet's² series all the inmates of a small house—viz., four brothers and one sister—were swept off by pneumonia within three weeks. In Daly's³ series four children, the mother and grandmother, followed one another in rapid succession with pneumonia, and the two latter died. In Finlayson's⁴ case five out of thirteen inmates of the same house were attacked. Similar cases are recorded by Slade-King,⁵ Muller,⁶ Oliver,⁷ and others. Besides these, there are the still more striking cases in which pneumonia attacked a healthy person after sleeping in the same bed with a patient suffering from it. Instances of this are recorded by Proby,⁸ Kuhn⁹ and others.

Still, however striking such cases may be, none of them are really conclusive, for the influence of the house or room cannot be eliminated.

A few instances, however, remain which point strongly in the direction of the possibility of contagion, for in them pneumonia has been apparently given either by a convalescent from pneumonia or by a third person to one who had not otherwise been in contact with the disease.

Dr. Wynter Blyth¹⁰ records a case in which a farmer with pneumonia was nursed by his niece, who acquired the disease and went home, where soon after her husband was attacked with it. In a second instance a farmer with pneumonia was nursed by his servant, and died, after which the servant also developed pneumonia and went home, where, within a few days, her married sister also fell a victim to it.

Butry's¹¹ case is of the same kind. A man contracted pneumonia and was sent home to his family. They lived in an isolated mill, and shortly following the arrival of the patient the mother, father, and one of the nephews residing in the house were also attacked. Schrötter's¹² case is a striking one of the same kind. A man had been ill for seven weeks with rheumatic fever, and had never left the room, when his father was brought into it suffering with pneumonia and put into the son's bed. The son slept with him during his illness, and eight days later was attacked with pneumonia also. In the outbreak already referred to in the Moringen Reformatory, Kuhn¹³ records that several cases arose in the families of attendants who lived out of the buildings and had not themselves had pneumonia, so that, unless the pneumonia was due to some general cause affecting the district, it must have been conveyed by the attendants indirectly by their clothes or persons. Flindt¹⁴ also records a case in which the

¹ *Arch. gén. de Méd.*, May 1888.

² *Lancet*, 1881, ii. 824.

³ *Practitioner*, 1884, p. 305.

⁴ *Lancet*, 1895, ii. 760.

⁵ *Berl. kl. Woch.*, 1888, 667.

⁶ *Arch. klin. Med.*, xxix., 1881.

⁷ *Loc. cit.*

⁸ *Lancet*, 1882, i. 305.

⁹ *Coll. Inv. Rep.*

¹⁰ *Arch. f. klin. Med.*, xxi. 127.

¹¹ *Lyon Méd.*, 1889, i. 191.

¹² *Lancet*, 1875, Sept. 18.

¹³ *Schmidt's Jahrb.*, 1862.

¹⁴ *Virch. Jahrb.*, 1884, ii. 170.

bedclothes which had been used to cover the corpse of a man, dead from pneumonia, was, a little while later, used without washing to cover the bed of a child, and this child was a few days later attacked by pneumonia. In another case the source of infection was supposed to be a chair which had been brought home by a workman to his house to mend out of the room of a patient who had died of pneumonia.

Netter¹ and Mendelssohn² record cases also in which patients in the ward have apparently acquired the disease from neighbouring pneumonia patients.

In Talamon's³ cases, the mother died on the ninth day of pneumonia. The husband and son went out in cold weather to register the death, and were both seized with a rigor on their return and developed pneumonia, the husband dying in a few days. The son had a very severe double pneumonia, but ultimately recovered.

In Mosler's⁴ case the father died on the fifth day, Jan. 22nd; the wife was attacked on the day of the father's death and died five days later. The son, aged 30, who visited his parents but did not reside in the house, was attacked the day before the mother's death and died after twelve days' illness. The daughter who stayed in the house for four days, from the death of the father to that of the mother, developed pneumonia three days after leaving, but recovered.

This case is interesting, as the bacteriological examination showed the case to be one of septic infection.

Lastly, cases are recorded in which the infection is supposed to have been conveyed in utero, the mother suffering from pneumonia at the time the child was born.⁵

In one case the infant was ill at birth and died shortly after, when the apex of one lung was found to be consolidated and to contain pneumococci.

In another case the child lived four days, and *post-mortem* was found to have pneumonia and pleurisy, suppurative pericarditis and meningitis, the pneumococcus being present in all the organs.

In Levy's⁶ case the mother died one day after parturition, the infant three days later with lobar and lobular pneumonia. Pneumococci were found in the pleura, lung, and blood.

Pneumococci have also been found in the milk of nursing women with pneumonia, but usually the secretion of milk is arrested.

As concerning all these cases it must be said that they are extremely rare, even if they are all accepted without criticism, as conclusive.

Against them must be set the common experience of all observers, that pneumonia, as it is ordinarily met with, is, at any rate in this country, not infectious, *i.e.*, it is not, under ordinary circumstances, transmitted from the sick to the healthy.

It may be that these instances of epidemic or infective pneumonia are due to some other infection than the pneumococcus, but so far this has not been conclusively proved by bacteriological investigation. Indeed, in some of the so-called epidemics the pneumococcus has been the prevailing germ. If this be so we have two alternative theories; either that the pneumococcus may under certain, so far unknown, conditions become specially virulent and infective, or that the prevalence of some other epidemic affection, like influenza, renders the body specially susceptible to the attack of the pneumococcus. This latter seems to be the most probable explanation. If so, and remembering that the pneumococcus appears to be almost constantly present in the saliva of healthy persons in a non-virulent form, it may be that cleanliness and a careful mouth-toilette may do much to diminish the prevalence of pneumonia as a complication of those diseases, *e.g.*, influenza and measles, of which it is so common a sequela.

Longstaff's⁷ investigations into the mortality statistics confirm the conclusion to which clinical evidence leads, for they show that the mortality-curves of pneumonia differ so markedly from those of typhoid fever as to afford no support to the view that they are diseases of a similar

¹ *Loc. cit.*

³ *Méd. Mod.*, Mar. 20, 1895.

⁵ Thorner, Thesis, München, 1884. Netter, *Soc. de Biol.*, 1889. Cf. Charcot, *Méd.*, *l.c.*

⁶ Levy, *Arch. f. exper. Path.*, 1889.

² *Loc. cit.*

⁴ *Lancet*, June 25, 1890.

⁷ *Studies in Statistics*, p. 382.

character and spread in a similar way, and although the statistics do not actually exclude the possibility of a communicable pneumonia, having no connection with cold, they show that such a theory could account for but a small proportion of the deaths attributable to pneumonia.

Incubation Period.—If pneumonia be at the most only in rare instances an infectious disease, the period of incubation becomes of slight importance. The period as usually given is short, viz., two or three days, but the variability of the supposed incubation is one of the difficulties which the contagionists have to meet, for it is often necessary to assume much longer periods, *e.g.*, as many weeks or even months, in order to bring all cases into conformity with the theory.

Immunity.—One attack of pneumonia by no means confers immunity, *i.e.*, protects the patient against another; on the contrary it seems greatly to increase the liability, so that about 30 per cent. of those who have had pneumonia once have it again at some time or another. This is the average of a number of statistics given by Jürgensen.¹

In the Collective Investigation Reports, in 10 per cent. of the cases a history of a previous attack was obtained.

Netter² holds that the liability even goes on increasing, for while 26·8 per cent. of those who have had it once have it again, 35 per cent. of those who have had it twice have it more than twice.

Riesell's³ results are as follow:—Out of 102 cases in which pneumonia had recurred 31 had it three times, 10 four times, 3 five times, 1 seven times, and 1 eight times.

Repeated attacks are not at any rate uncommon. For pneumonia to recur 3, 4, and 5 times is by no means rare. I know an instance in which it recurred 6 times, the last being fatal, and Leyden records a case fatal in the 7th attack. Instances of an 8th, 9th, 10th, 11th, and 16th attack are recorded by Grisolle, Keiler, Chomel, Traube, and Andral respectively,⁴ and even a 28th attack is recorded by Rush and a 30th by Moellmann.

This being so, the chances of discovering any means of preventive inoculation against pneumonia are small at present; but the other line of investigation which has been so successfully followed in the case of diphtheria is more promising.

Various animals have been rendered immune to pneumococcic infection, *e.g.*, rabbits, cows, asses, and horses, and the blood-serum obtained from such immune animals has been employed as an antitoxin injection for pneumonia in man.

The antitoxin treatment, first initiated by Klemperer,⁵ has been actively pursued by many investigators. The fluids employed have been (1) the expressed juice of pneumonic tissues, (2) the filtered and sterilized products of pneumococcus cultivations, and recently the blood-serum of immunised animals, *e.g.*, the cow, ass, and rabbit. Recently blood serum from persons convalescent from pneumonia has also been employed.⁶

The reports of different investigators vary greatly, but they all seem to agree in the opinion that the injections at any rate do no harm; at the same time they have not been followed by any striking success, so that for the present the serum treatment of pneumonia has not justified the promise which it first held out.

MORBID ANATOMY.—Whatever difference of opinion there may be as to the bacteriology of pneumonia, there is none as to the anatomical changes produced by it in the lung.

¹ Ziemssen, *loc. cit.*

³ *Viertel. Jahrb. gerichtl. Med.*, 1889, N.F., i. 145.

⁵ Mosny, *Arch. de Méd. Expér.*, 1893, No. 2, p. 260.

⁶ Eyre and Washbourne, *Lancet*, April 8, 1899.

² *Loc. cit.*

⁴ *Cf.* Ziemssen.

Pneumonia consists of a fibrinous exudation into the alveoli, which expels the air and completely fills the vesicles, so as to convert the parts of the lung affected into a solid airless mass, which sinks in water and presents on section a uniform granular surface. As in other inflamed tissues, the first change which the pneumonic lung undergoes shows itself in the circulation, the vessels becoming greatly distended with blood, and accordingly this is spoken of as the stage of *hyperæmia* or *engorgement*. The hyperæmia is rapidly followed by exudation, by which the parts affected are rendered solid and look on section not unlike a piece of liver, a resemblance which suggested the name *hepatisation*, by which this stage is usually described. The hepatised portions differ in colour according to the amount of blood they contain, and to the changes which the exudation has undergone, and are described as *red* or *gray* accordingly.

The three stages, then, which are usually described are (1) the stage of engorgement, (2) that of red and (3) that of gray hepatisation. No sharp line can of course be drawn between them, for they pass gradually one into the other, and are not infrequently all present at the same time, the parts affected first being in the gray stage, those affected later in the red, while the margins are still in the stage of engorgement, so that by sections taken from different parts of the same lung the whole evolution of the disease may be easily studied.

1. *The stage of engorgement.*—In this stage the parts are bright red, or if dark at first, rapidly become bright on exposure to the air. The tissues are gorged with blood, and there is excess of secretion in the small air-tubes. The vessels which run in the alveolar walls and round the infundibula are distended and project into the lumen. Some of them may even rupture and extravasations of blood be found in the alveoli.

2. *The stage of red hepatisation.*—In this stage the vessels are still distended, but copious exudation has taken place from them and filled the vesicles. This

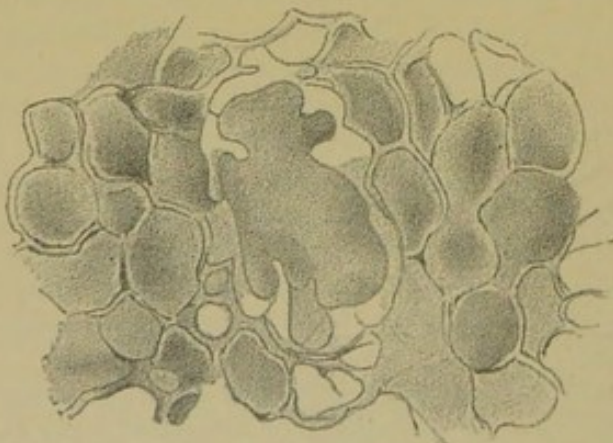


Fig. 60.

Section of lung in acute pneumonia. In the centre is a cast of a small bronchus and its alveoli (from Cornil and Ranvier).

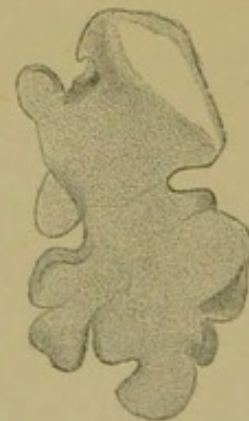


Fig. 61.

Cast of a small bronchus and its terminal alveoli (+40) (from Cornil and Ranvier).

exudation is composed of white and red blood cells derived from the blood, and a few large epithelial cells derived from the walls of the alveoli, the whole felted together with threads of fibrin to form solid casts of the vesicles and infundibula. These casts can always be obtained easily from the surface of a section by scraping, and are frequently found in the sputum. The proportion of red to

white cells as well as the amount of fibrin differs much in different cases and in different parts, even in adjacent alveoli. It was this resemblance in structure to that of a croupous membrane that suggested the name of croupous pneumonia.

3. *The stage of gray hepatisation.*—In this stage the exudation consists almost entirely of white cells, but the alveoli contain an increased number of epithelial cells, which are most abundant between the exudation and the walls of the vesicles, showing that to the acute inflammation there has succeeded more or less of a catarrhal process by which the loosening of the casts and their softening and removal are facilitated. The red blood cells and the fibrin have disintegrated and almost entirely disappeared. The cells, both white and epithelial, are all in a condition of granular and fatty degeneration, and it is to the colour thus produced, associated with the disappearance of the vascular congestion, that the gray or yellow tint is due. The consolidated parts, whether in the stage of red or gray hepatisation, are distended to their maximum, and the tension is indicated upon the outside of the lung by the protrusion of those parts corresponding with the intercostal spaces, so that deep depressions or furrows are left corresponding with the ribs, and the same evidence of tension is given on section by the retraction of the tissues, and the protrusion of the exudation, thus giving the cut surface its granular appearance.

When the hepatisation is near the surface, the pleura over it, as well as the opposed portion of the costal pleura, is in a condition of acute inflammation. It has lost its polish, is roughened and covered with flaky lymph, and if not separated by effusion, the opposed surfaces here and also between the lobes have become adherent.

The inflammation, as a rule, spreads from the affected vesicles some little distance along the bronchi, and usually takes the form of a more or less severe catarrh, which, if widespread, *i.e.*, not confined to the immediate neighbourhood of the pneumonia, may constitute a very serious complication of the case. In some cases a fibrinous

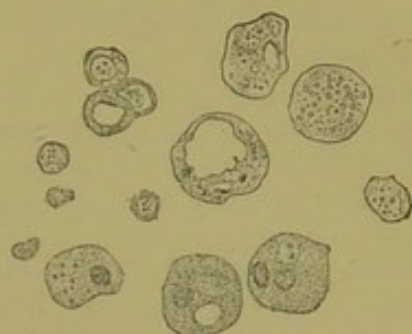


Fig. 62.

Epithelial and white blood cells, obtained by scraping the surface of a pneumonic patch in various stages of fatty and granular disintegration.

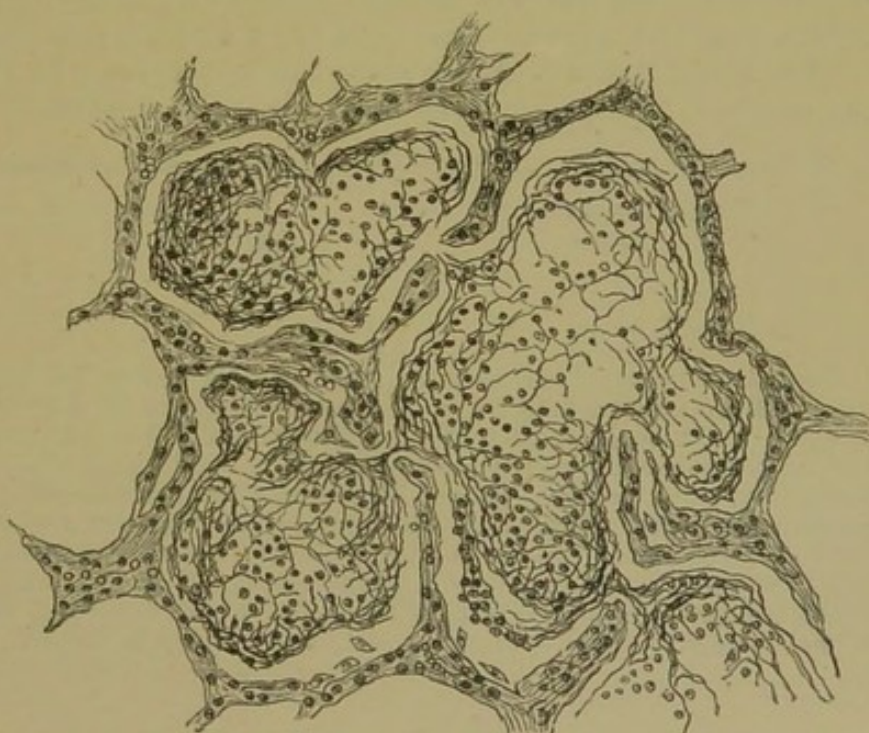


Fig. 63.

Acute pneumonia, to show fibrin network.

exudation similar to that in the alveoli takes place into the tubes, so that dendritic casts are formed, and may be expectorated, as in plastic bronchitis or in croup.

The question has been discussed whether the exudation comes from the bronchial or the pulmonary vessels. It has been assumed that the bronchial arteries, being the nutrient vessels of the lungs, are chiefly concerned in the inflammatory process. Pneumonia has, it is true, been met with where the pulmonary artery has been plugged, but it is most likely that both sets of vessels take their part in the process.

The parenchyma, or actual tissue of the lung itself, is, except for the transitory congestion, little if at all affected. In some long-standing cases a slight degree of small-celled infiltration is found in the lymphatic and perivascular tissue, but if it be more than a slight amount it is a question whether we have not to deal either with changes antecedent to the pneumonia, or with another form of affection different from, and perhaps not even related to, pneumonia (*cf.* Indurative Pneumonia).

The circulation, through both lymphatics and blood-vessels, remains free throughout, and to this is probably due the rapid absorption and removal of the exudation after the inflammation is past.

The most striking peculiarity of pneumonia, as the name lobar implies, is the rapidity with which large portions of the lung become involved, so that in the course of a few hours a complete lobe, the whole of one lung, or even a considerable part of both may become consolidated. At the same time a patient may die of pneumonia, and yet the area of consolidation be not larger than a small orange. The weight of the exudation may thus vary from an ounce or two to some pounds. Whatever its size, the part affected presents the same appearances. It is fleshy, airless, firm, but tearing easily, and has a uniform granular surface on section.

The consolidated lungs are always much increased in weight. They are often two or three, and may be even four or five, times as heavy as normal.

The average weights of the healthy lungs in adults are, according to Reid—

For the Male.				For the Female.			
24 ozs.	.	.	right	.	.	17 ozs.	.
21 „	.	.	left	.	.	15 „	.
Total, 45 ozs.				32 ozs.			

In the 100 cases taken from the *post-mortem* record of St. Bartholomew's Hospital, the weight of the affected lung lay in 22 instances between 40 and 50 ounces; in 6, between 50 and 60; in 2, between 60 and 70; and in 6, between 70 and 80; while the 3 highest were 92, 94, and 102 ounces respectively. The increase in weight is not, however, confined to the pneumonic lung alone, but the opposite lung, even when not also pneumonic, is often much above the normal weight. This is no doubt due to the collateral congestion, *i.e.*, to the increased amount of blood present in it. A good idea of the actual diminution of the respiratory capacity may be obtained by taking the sum of the weights of the two lungs together; and it is by no means rare for the combined weights to amount to two or even to three times the normal, the normal average being for the male 45, and for the female 32 ounces.

Weights of the two lungs together, and of each separately where one lung only was pneumonic.

Right, . . .	102	94	92	83	81	77	76	76	70	70	67	64
Left, . . .	18	36	27	28	25	...	28	...	28	20	24	28
Both, . . .	120	130	119	111	106	...	104	...	98	90	91	92

The heaviest weights are all found, as would be anticipated, where the right lung is the seat of the consolidation.

The figures where both lungs are affected are interesting, for though the weights of each single lung are much smaller, the aggregate of the two is still very considerable.

Weights where both lungs are affected.						
Right, . . .	49	53	52	48	45	
Left, . . .	48	40	36	37	27	
Both, . . .	97	93	88	85	72	

It is very remarkable how large a portion of the lungs may become involved before death occurs. In one of the cases cited the whole of the right lung was solid except the anterior fringe of the upper lobe, as well as the lower two-thirds of the left, the weights being 53 and 40 ounces respectively.

The parts of the lung attacked.—The right lung is more frequently attacked than the left, and the base than the apex. An analysis of 750 cases in St. Bartholomew's Hospital shows that the right lung was the seat of the lesion in 48 per cent. and the left in 44 per cent., while both lungs were attacked in 8 per cent.

With these Pye-Smith's figures closely agree. Right lung, 47·4 per cent.; left lung, 39·9; both lungs, 12·7.

Jürgensen's¹ statistics give a somewhat higher percentage for the right lung, as do also those of Moellmann.²

Right.	Left.	Both Lungs.
53·1	36·5	10·4 (Jürgensen.)
53·7	38·23	8·0 (Biach.)
54	37	9 (Moellmann.)

Biach's detailed figures are as follows:—

	Right, 53·7.	Left, 38·23.	Both, 8·0.
Upper lobe, . . .	12·15	6·96	1·09
Middle, . . .	1·77
Lower, . . .	22·14	22·73	3·34
Upper and middle, . . .	2·65	...	3·64
Lower and middle, . . .	5·64	...	
Whole lung, . . .	9·33	8·54	...

The base of the lung is attacked in from 75 to 80 per cent., and the apex in about 18 per cent. My own statistics give 75 per cent. for one base, 16·5 per cent. for one apex.

Base pneumonia does not exhibit much preference for either side, for it is but a little more frequent on the right than on the left. Apex pneumonia, on the other hand, occurs nearly twice as often on the right side as on the left, as the following figures show:—

	Right.	Left.	Right.	Left.	
Own { Apex, . . .	11·5	5·0	13·8	4·6	Pye-Smith's.
{ Base, . . .	36·0	39·0	32·2	34·8	

If both lungs are affected it is usually both bases, and only rarely some other combination of parts, the most frequent being the association of the right apex with the left base, and the rarest that of both apices.

¹ *Loc. cit.*

² *Berl. kl. Woch.*, 1889, Nos. 16, 18, 20. Clifford Allbutt, *Med.*, v. 132.

Thus out of the 8 per cent. in which both lungs were affected, both bases were involved in 6.2 per cent., and both apices in only 0.1 per cent. The right apex and left base were associated in 1.5 per cent., and the left apex and right base in 0.2 per cent.

In children, although, as stated, croupous pneumonia is much rarer than in adults, it much more frequently attacks the apex; and that the more so the younger the child is, as is shown by Schroeder's statistics:—

Age.	Apex Pneumonia.	Base Pneumonia.
0-5 years,	44 per cent.	56 per cent.
5-10 „	38 „	62 „
10-15 „	37 „	63 „
Adults,	32 „	68 „

The parts of the lungs which are not consolidated present the signs of more or less congestion, emphysema, and bronchial catarrh.

They contain an unusual amount of blood, and section is followed by the abundant escape of frothy blood-stained fluid. The bronchi contain a good deal of secretion. This is often mucopurulent, as in ordinary bronchitis, but at other times it is serous in character, and may be so abundant as to constitute actual œdema of the lung and cause it to pit on pressure. This condition of œdema is most marked in the lowest parts—that is, at the bases behind, where it is called “hypostatic congestion.” When one base is the seat of pneumonia the other may, in this way, be rendered absolutely airless. This hypostatic congestion may be the explanation of the fact that when pneumonia spreads to the opposite lung it is the base that is usually attacked.

The *bronchial glands* are swollen, red, and congested, *i.e.*, are in a state of secondary inflammation, but this rarely, if ever, reaches the extent of suppuration, and, when the pneumonia subsides, quickly disappears.

The *heart* presents no characteristic changes peculiar to pneumonia, for pericarditis and endocarditis are to be regarded as complications, and will be referred to later; but the cavities are often greatly dilated, especially those of the right side, and are filled with *ante-mortem* clot. These clots are often bulky, firm, white, and clearly formed during life. They often occupy both auricle and ventricle, and even extend some distance into the pulmonary artery. They are not infrequently the cause of death, either by the obstruction they cause to the circulation or by a portion becoming detached and being carried into the pulmonary artery.

The *spleen* is sometimes enlarged, but by no means invariably. In the series of cases referred to, it was not rarely of normal size and weight, and sometimes even smaller than usual.

Thus in 10 cases only was it described as markedly enlarged, in two of them reaching the weight of 15 to 21 ounces. On the other hand, it was remarkably small in two instances, and weighed only 2½ ounces.

Importance has been attached to enlargement of the spleen as evidence of the infective nature of the disease, but if so, the enlargement should be more frequent than it is, and it must not be forgotten that there are other causes present which are sufficient to produce it, notably the venous congestion due to the obstructed circulation through the lungs.

In the same way the small hæmorrhagic points, which are not infrequently found upon the pleura and pericardium, beneath the peritoneum, and in the mucous membrane of the stomach, and which are so often pointed to as evidence of infection, may be nothing more than the results of the mechanical obstruction to the circulation.

With these exceptions other lesions when present are to be regarded as accidental complications rather than as essential parts of the disease.

When the active inflammation ends, the consolidation is rapidly removed. The cells of the exudation undergo fatty and granular degeneration, the consolidation liquefies owing to the changes in the cells, assisted by the catarrhal exudation from the walls of the vesicles and bronchi, and the softened products are then removed by absorption or expectoration. In this process, or *resolution*, as it is called, absorption plays a predominant part, owing, doubtless, to the fact that the circulation through the blood-vessels and lymphatics continues free and unimpaired throughout the disease.

In the course of time, perhaps of only a few days, resolution is complete, and the lungs are restored to their original state. In the case of the pleura, however, resolution is often incomplete, and more or less extensive adhesions remain over the seat of the pneumonia.

COMPLICATIONS.—Of the results of pneumonia by far the commonest are, on the one hand, resolution with recovery, more or less complete, and, on the other, death from exhaustion or suffocation.

When the case is fatal the lung may be found, of course, in any stage of inflammation. Most frequently, however, it is in that of gray hepatisation, but it is not rare to find it in a state of purulent or puriform infiltration. In the puriform state the lung is simply in the condition of gray hepatisation, undergoing softening and liquefaction as described, but in the purulent state a condition akin to actual suppuration exists. The lung is then dull gray, soft, semi-fluid, and very brittle, as if beginning to break down. It is believed by many that this condition never occurs except in fatal cases, and that it is not the result of the pneumonia but of a separate infection with suppurative organisms.

In the 100 fatal cases referred to the condition is specified in 64, and out of these it was that of gray hepatisation in 33, or about one half; of red hepatisation in 17, or about a quarter; of partly red and gray in 4; and of purulent infiltration in 10. In 2 of the last 10 the tissue had almost broken down into suppuration, and in 2 others the colour was greenish gray, and looked as if it were gangrenous, though the characteristic odour was absent.

The other pathological conditions to which it is usually stated pneumonia may lead are *abscess*, *gangrene*, and *interstitial induration*. They are all, however, at the most but rare sequelæ.

Abscess after pneumonia is extremely rare.

Of 1165 cases reported in the medical statistics of St. Bartholomew's Hospital, abscess occurred in 2 only, a boy of 11 and a man of 30.

In my own series of 100 fatal cases abscess was found in 1 only, and its walls were thick and fibrous, so that it could not have owed its origin to the recent pneumonia.

Sello¹ in 750 cases found abscess in 11, *i.e.*, 1·5 per cent. Of these 7 recovered with evacuation of the pus by expectoration, or by operation; and 4 died.

Even when abscesses are found they may be due to other causes, *e.g.*, pyæmic infarcts or septic and other foreign substances in the air-tubes, round which pneumonia has developed, so that the mere co-existence of abscess and pneumonia proves nothing as to the causal relation between them. In those rare cases in which abscess really follows pneumonia it is most probably the result of a special septic infection akin to that which causes general suppuration or gangrene. Some abscesses certainly originate in local gangrene, for the slough may be found *post-mortem*, or be expectorated during life.

¹ *Zeitsch. f. klin. Med.*, xxxvi.

Abscess does not, however, lead necessarily to a fatal result, so that the frequency, or rather rarity, of abscess after pneumonia must not be determined from the experience of the dead-house alone.

Gangrene is as rare as abscess, perhaps even rarer. It may be diffuse or localised, and in the latter case, if not fatal, may subsequently present itself as an abscess. The gangrenous parts are crowded with septic organisms, with which also the vessels are plugged, and it may be to this obstruction of the circulation that the gangrene is in great part due. Gangrene is most likely to occur when the sources of putrefactive infection are already present in the lung, and thus it may follow upon pneumonia in patients who have already bronchiectatic or other cavities with putrid contents.

The rarity of gangrene is shown by statistics, for in the Collective Investigation Reports only 2 instances are recorded out of 1065 cases, and therewith the St. Bartholomew's statistics almost exactly agree, for they show only 2 cases out of 1165. The one, a youth of 21, died, the other recovered. Coupland, out of 39 cases of gangrene met with in the course of ten years at the Middlesex Hospital, found only 14 associated with pneumonia, and in only 8 of these was the gangrene consequent on the pneumonia.

In my series of 100 fatal cases only 2 were in a condition approaching gangrene, but in neither of these was the characteristic odour present.

Sello's¹ statistics give 3 cases in 750, *i.e.*, 0.4 per cent.

Induration—Fibrosis—Interstitial Pneumonia.—When induration is spoken of as following pneumonia, what is meant is not the local fibrosis which spreads inwards along the trabeculae of the lung from a chronic thickened pleura (*pleurogenic induration*), nor those patches of induration in the substance of the lung which may have many origins, but a more or less diffuse widespread induration involving those parts of the lung which have been previously the seat of pneumonia.

What has been described in such cases is as follows:—In the early stage, the affected parts remain solid, airless, and granular on section; the interstitial tissue of the lung is found infiltrated with small cells in active proliferation; the alveolar contents become gradually organised and converted into fibrous tissue by the passage into them of blood-vessels from the walls, so that in the end the alveolar contents are continuous with the interstitial induration, until by the contraction of the newly-formed connective tissue the alveoli are in great part obliterated, and a solid mass of fibrous tissue is produced. It is, however, an open question whether this condition is not an affection, *sui generis*, independent of and not in any way necessarily related to acute pneumonia.

Theoretically it must be admitted on the analogy of other organs that acute pneumonia, if its resolution be long delayed, may end in interstitial induration, and this no doubt happens in certain cases. The question is whether this event be a common or likely occurrence. Common it certainly is not, and many of the most protracted cases of pneumonia end at last in complete resolution. Even where induration is found in the lungs of a patient who has previously had pneumonia, it does not of course follow that it has been the result of the pneumonia. The difficulty is similar to that which besets the question of the relation between acute nephritis and granular kidney. It is not improbable that the same conclusion is true in both cases alike, *viz.*, that while interstitial change may sometimes follow the acute inflammation, instances of it are rare, and that most cases of interstitial induration are to be referred to other causes. The probabilities are, at any rate, in favour of this view, but the subject will be more fully discussed under the head of Chronic Interstitial Pneumonia.

CLINICAL COURSE, PHYSICAL SIGNS AND SYMPTOMS.—Pneumonia is such a characteristic disease that no better description can be given than the account of a typical case.

¹ *Loc. cit.*

A youth, 18 years of age, of fairly good health, was suddenly attacked in the later part of one afternoon with a violent shivering fit, which lasted nearly half an hour, and was almost immediately followed by a sharp stitch in the left side. He vomited several times, felt extremely ill, had a racking frontal headache and a constant sense of nausea, and as soon as possible was put to bed. The pain in the side continued and became more severe, so that it caused great distress in breathing, while at the same time a short, dry, frequent cough came on, which, on account of the pain it gave, the patient did his best to control, but without success. That evening he became very feverish, and could not sleep because of the pain and cough. During the following day he remained in much the same state, except that he grew gradually worse. He lost all appetite and suffered much from thirst; the stomach was so irritable that he vomited almost everything he took, and at the same time diarrhoea set in, which lasted for the next thirty-six hours. It was on the third day of illness that he came under observation.

3rd day.—The patient was found to be a fairly developed but somewhat weakly-looking lad. He was lying on his back in bed with an expression of pain upon his face, breathing rapidly and with evident distress, for the respirations were short, shallow, and jerky, while the alæ nasi dilated widely with each inspiration. The cheeks were red and flushed, the complexion somewhat dusky,

and the lips dry; the tongue was coated with a thick fur, while the tip and centre were dry, brown and cracking. The skin of the body felt pungently hot and dry, and the temperature proved to be 104° . The pulse was 120 and of slightly increased tension; the respirations 42, short, shallow, frequent, and interrupted by cough. The patient could speak only in short, jerky sentences, for he did not dare to breathe freely. He made complaint of pain in his side, of cough, frontal headache, and thirst. The cough was frequent, short, hacking, restrained, and without expectoration. The urine was scanty, 1030, acid, and deposited on cooling a copious sediment of urates, but was free from albumen. The ratio of the pulse to the respiration was diminished $\frac{120}{42}$, so as to be less than 3 to 1. Physical

examination revealed nothing abnormal except in the chest. Here the lower part of the left side appeared somewhat distended, and hardly moved at all on respiration. The percussion in the lower part of the front below the nipple, and in the upper part of the axilla, was hyper-resonant (skodaic resonance). At the base behind it was dull up to the angle of the scapula, and over this area the vocal vibrations were increased and bronchophony and bronchial breathing audible. Just above the dull area fine crepitation was heard, which was increased by coughing, and in the axilla pleuritic friction was found.

Into the upper part of the left lung the air entered fairly, though not freely, owing to the restricted movements.

Over the whole of the right lung the respiration was somewhat loud and exaggerated, being clearly complementary or puerile.

The patient was much distressed by any movement, and was not in a condition to bear prolonged examination.

The diagnosis was clear. The patient evidently had acute pneumonia of the lower part of the left lung.

That night the patient slept very badly, was actively delirious, and very restless.

4th day.—The condition next day was somewhat worse in all respects, and the temperature had still further risen, remaining for some hours over 105° . The cough was more troublesome

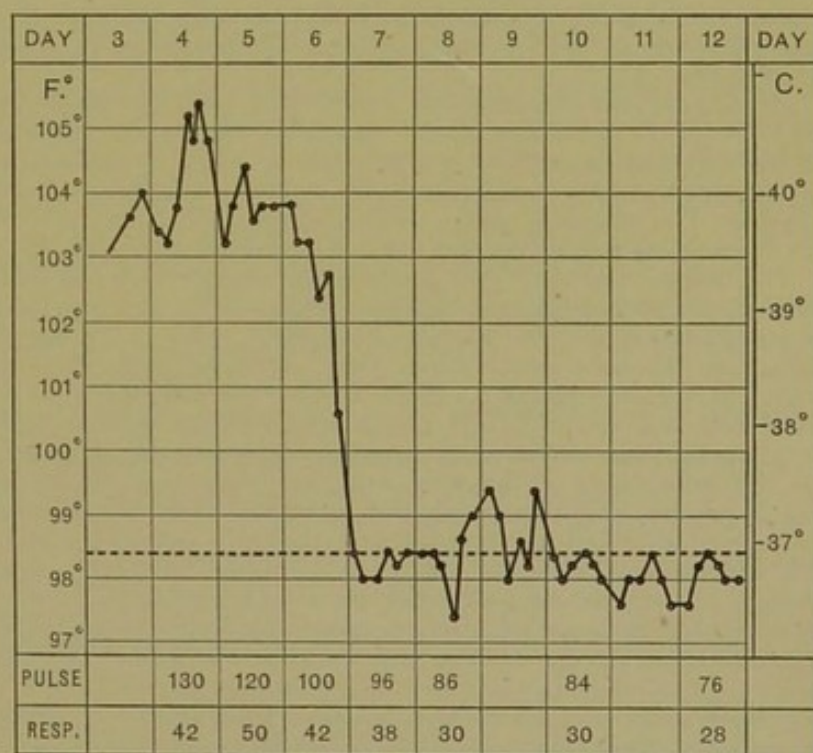


Fig. 64.

and the difficulty on breathing considerable, though, owing to the greater delirium, the patient was less conscious of his state and therefore less distressed. The pulse remained the same, but the respirations had risen to 50, so that the pulse respiration ratio was still further reduced $\frac{120}{50}$, i.e., about $2\frac{1}{2}$ only to 1.

The succeeding night was spent, like the former, in restless delirium, but the next morning (5th day) the temperature had fallen nearly 2 degrees, and with this was associated an improvement in the general condition and a decrease in the delirium. Physical examination showed that the dulness had extended upwards to the middle of the scapula and forwards in the axilla, so that the whole lower lobe was clearly involved. Over the whole dull area bronchophony and bronchial breathing were heard, except at the lower part behind, where both breath- and voice-sounds were inaudible. The temperature ranged between 103° and 104° , the pulse was 120, and the respirations 50. The general condition had somewhat improved, the cough was looser and associated to-day for the first time with the characteristic expectoration, the sputum being scanty, viscid, and rusty.

The improvement continued throughout the day, and the night, like the day, was better than the last, so that the patient was less delirious and obtained more sleep.

6th day.—The morning found the patient still better, and refreshed by his better night. The delirium was less, the cough and expectoration about the same, T. 103.3 ; P. 100, and R. 44, the most important tangible facts being the drop of 20 in the pulse rate and of 6 in the respiration rate. This morning a patch of herpes made its appearance upon the upper lip on the left side.

At 6 p.m. the temperature, which had been gradually falling through the day from 103.3° to 102.6° , suddenly dropped, and before midnight reached 98° . The crisis was accompanied by profuse sweating and by a further drop in both pulse and respiration rate. Great general improvement at once took place, the delirium disappeared, and the patient lost almost all distress. The urine was scanty, concentrated, and deposited a very copious sediment of urates, but was now, as it had been throughout, free from albumen.

The night was spent in quiet sleep, undisturbed by delirium, and the next (7th day) morning the patient looked a different being. The face was pale, but the flush and dusiness were gone, the expression of pain and anxiety replaced by one of quiet weariness. The cough continued, but was almost without pain, and the expectoration was much reduced, though still rusty. No change could be made out in the physical signs, but the general improvement continued, and the pulse and respirations fell still further to 86 and 30 respectively.

The subsequent course of the case was one of uninterrupted convalescence. The temperature rose on the evening of the 8th, as it also did on that of the 9th day to 99.6° , but after this became permanently subnormal, and continued so for the next few days. The cough ceased to give any trouble, and the expectoration, though it became less and less, continued rusty until the 10th day, when it ceased entirely.

Rapid improvement took place in the physical signs, and by the 14th day the bronchial breathing had disappeared and vesicular breathing was beginning to be audible. On the 17th day the patient was up, looking pale but otherwise well. Ten days later he was sent into the country convalescent in all respects, and without physical signs, except that the breathing over the lowest part of the left base behind was sometimes accompanied with a little wheezing. On his return from the country he looked, and was said to be by his friends, in better health than he had ever been in his life before.

This instance illustrates, as well as any single case is likely to do, the essential features of the disease. It is characterised by the sudden onset of acute fever indicated by the usual *general signs*, viz., shivering, frontal headache, sickness, rapid pulse and breathing, rise of temperature, concentrated urine, and gastrointestinal disturbance.

The *localising symptoms* indicating the seat of the lesion all point to the chest, viz., the unusually rapid respiration and dyspnoea, the altered pulse-respiration ratio, the pain in the side and cough. These symptoms are confirmed by the *physical signs*, which prove the existence of consolidation of the lung. The diagnosis is thus evident, viz., extensive acute inflammatory consolidation of the lung—in other words, acute lobar pneumonia. The disease which sets in so acutely reaches its acme in the course of some hours, and after remaining at its height for a few days longer, terminates, as abruptly as it begins, in about a week. While the general signs of fever subside quickly, the physical signs continue, and though they too change rapidly, they take some days to disappear completely. Often, within a fortnight from the time the temperature fell, except for the

weakness left behind by the fever, the patient is well; a few days' change in the country will complete the convalescence, so that by the end of six weeks or so the health will be perfectly re-established, and no trace of the mischief be left behind in the lungs.

Characteristic as pneumonia so frequently is, still a case as typical as that just given is by no means common. Analysis of the separate symptoms and physical signs will make evident how large a scope for variation there is in any individual case.

ANALYSIS OF SIGNS AND SYMPTOMS.

A. THE GENERAL SIGNS.

The onset of the disease is, as a rule, very sudden, and not only the day, but often the hour, can be fixed. *Premonitory signs* are usually entirely absent, or, if present, are of a very slight and indefinite character, so that the disease appears to strike the patient down in the midst of health. Undue stress is often laid upon premonitory symptoms, as though they were a necessary part of the argument in favour of the germ theory, but their presence or absence does not really affect the question.

The onset is usually marked by the ordinary general symptoms, viz., *shivering, frontal headache, vomiting, and fever*, and is attended with so great prostration and *feeling of illness* that the patient is driven to take to bed at once. Where pneumonia occurs as a complication of bronchitis, or in the course of some other fever, such as typhoid or scarlet fever, the onset is often not abrupt but gradual, and the same holds good in aged or cachectic persons, in whom, even with extensive pneumonia, the fever may be but moderate throughout.

There is, however, a remarkable group of cases, in which the constitutional symptoms are so slight that the patient is able to remain up and about, and may even continue at work, and that when the consolidation in the lungs is by no means small.

It is in these cases that grave symptoms may set in abruptly, and the patient die suddenly. Every year a certain number of cases of this kind are brought in dead into the hospitals, having dropped in the street or at their work. Of this the following is a good instance.

A man of middle age was seen to fall in the street, and was brought into the hospital dead. The *post-mortem* revealed acute pneumonia of the right upper lobe of considerable extent, in the stage of gray hepatisation, the right lung weighing 48½ ounces and the left only 16 ounces. Except that there was slight atheroma in the arteries and a little interstitial nephritis, there was no other gross lesion. The heart muscle was healthy, but all its cavities were filled with firm dark clot, which extended into the pulmonary vessels and aorta. Possibly this clotting may have been the actual cause of death.

Shivering is rarely absent as an initial symptom in some degree, and in most cases takes the form of a very severe **rigor**, which may last twenty minutes to half an hour. There are few diseases in which the initial rigor is so severe or prolonged. Usually the rigor, if it has been severe, is single, and is not repeated during the attack. It may, however, now and then happen that there are two or three well-marked rigors, but under any circumstances they occur during the first few hours only. The place of the rigor is sometimes taken by slight shiverings. If so, they may be repeated and last throughout the first or even the second day.

Sir Andrew Clark records¹ a case in which there were thirteen well-marked rigors in twenty days, but the long duration of the attack renders the case peculiar.

¹ *Med. T. and G.*, 1884, ii. 841.

A well-marked rigor or rigors occur in from 60 to 80 per cent. of all cases.

In my own series of 100 cases the rigor was absent in 21, and in 4 of these the pneumonia occurred in the course of some other fever or acute disease. In 25 instances the rigor was noted as being specially severe, and in 2 others the rigor recurred during the first few hours. In one instance the patient had four very severe rigors within twelve hours, and died ultimately on the 10th day, of pericarditis. Of the remainder, one patient had slight shivering attacks, which were repeated at short intervals, and lasted till the end of the second day.

The most frequent time for pneumonia to commence, as indicated by the rigor, is during the afternoon or early evening, and the rarest the middle part of the day or night.

In young children the place of the rigor is often taken, as in other fevers, by a convulsive fit. When the fits recur and the child remains unconscious, the difficulties of diagnosis between pneumonia and meningitis may be by no means slight.

Temperature.—With the commencement of the attack the temperature begins at once to rise, and rises rapidly, so that within a few hours it reaches 103° or 104° ; it remains at or about this level with but slight remissions for some days, and then falls almost as suddenly as it rose, the affection ending in most cases with a well-marked crisis. The typical chart may be divided into three parts—(1) the rise, (2) the continued fever, (3) the fall, and each of these parts shows variations and peculiarities in special cases.

The *rise* is, as already stated, very rapid; that is to say, within twelve hours, in most cases, the temperature will reach almost its maximum, and it is by no means rare for the temperature to rise to 104° or even 105° within three or four hours, so that if the rigor occurs, as it often does, in the late afternoon, the fever may be well marked by the evening. Still more rapid rises are recorded, as, for example, a case of Quinquard's quoted by Jürgensen, in which the temperature rose in the fifteen minutes following the rigor from normal to 103° (39.3° C.), and at the end of the hour reached 105.8° (41° C.).

Similar rapid rises are, of course, met with after severe rigors in other affections, notably in pyæmia, ulcerative endocarditis, and such-like cases. The most remarkable instance in my own experience occurred after the passage of a small catheter for stricture: a rigor followed almost immediately, and lasted for twenty minutes, when the temperature was taken and found to have risen from normal to 108.6° .

The rise may, however, be much more gradual, and this is especially the case in aged and cachectic persons, and where pneumonia occurs in the course of bronchitis or some specific fever.

The continued fever.—The temperature having risen to a certain height, remains very much at the same level for several days. Daily oscillations are present, but slight; they average about one degree, the evening temperature being the higher, but, as in other fevers, this may be reversed. The daily oscillations may be more considerable, and even so extreme as to deserve the name, which has been applied to them, of *remittent*, or *hectic*. Such extreme irregularities, like other eccentricities in the disease, are always uncomfortable signs, but still not of necessarily bad prognosis, for they may end, like simple cases, with a crisis and recovery.

Wunderlich, in his typical temperature chart of pneumonia, lays stress on a drop which he states occurs on the fifth day, but this drop is not constant, and has not the importance he attached to it.

Sometimes at about the time the crisis is expected a drop of two or more degrees may occur, as if the crisis were commencing (*pseudocrisis*; *precritical drop*), but the temperature may then cease to fall and remain at the lower level,

or even rise again above the previous level during the next twenty-four to thirty-six hours, after which the crisis may develop in the ordinary way. In both cases these are accidental variations only, and not in any way characteristic.

It is true that the height of the continued fever gives a rough measure of the severity of the case, other things being equal, but too much stress must not be laid upon it, for the temperature may be high without other anxious signs in cases which do perfectly well, and not high in otherwise grave cases which end in death. A temperature of 106° or more becomes of itself a grave symptom, but such an amount of fever is exceptional. It may occur on the second or third day, and if reduced by appropriate measures may not affect the favourable course of the disease. More often

the unusual temperatures occur late in the case, about the time when the crisis might be expected, and usher in death.

Wilson Fox's table shows that fever above 106° occurred only in 4.6 per cent. of all cases, and that in 84 per cent. the temperatures ranged between 103° and 106° , being in 40 per cent. between 104° and 105° .

On the other hand, the temperature may be quite low, and never rise above, for example, 101° .

Cases are also recorded on good authority in which the temperature has never been raised at all (*Apyretic Pneumonia*).

Thus Koranyi¹ records a case of a chlorotic girl who had an ordinary pneumonia, lasting for nine days, but without any rise of temperature throughout, and Walshe² also refers to this fact. Many of the earlier cases recorded were observed before the clinical thermometer was used systematically. The temperature in the axilla has been sometimes found higher on the affected side.

The Fall.—The fall in temperature is abrupt, the fever ending in most cases by *crisis*. The crisis consists, however, of much more than the mere fall of temperature, for besides this and the drop in the pulse and respiration-rate which go with it, there is the most marked change in the general condition, so that the patient seems to have passed almost miraculously from a state of the utmost distress and danger to one of comparative comfort and safety. Accompanying the crisis there often occur what have been described as *critical discharges*, of

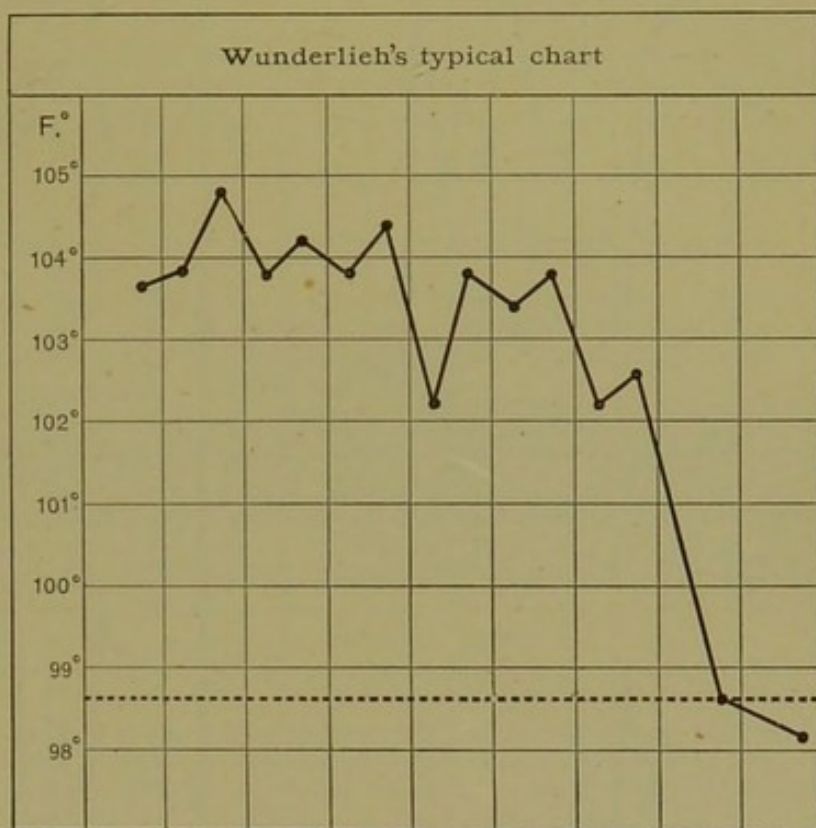


Fig. 65.

¹ *Real. Encycl.*, vii. 372.

² *Gerhardt's Handb.*, iii. 643.

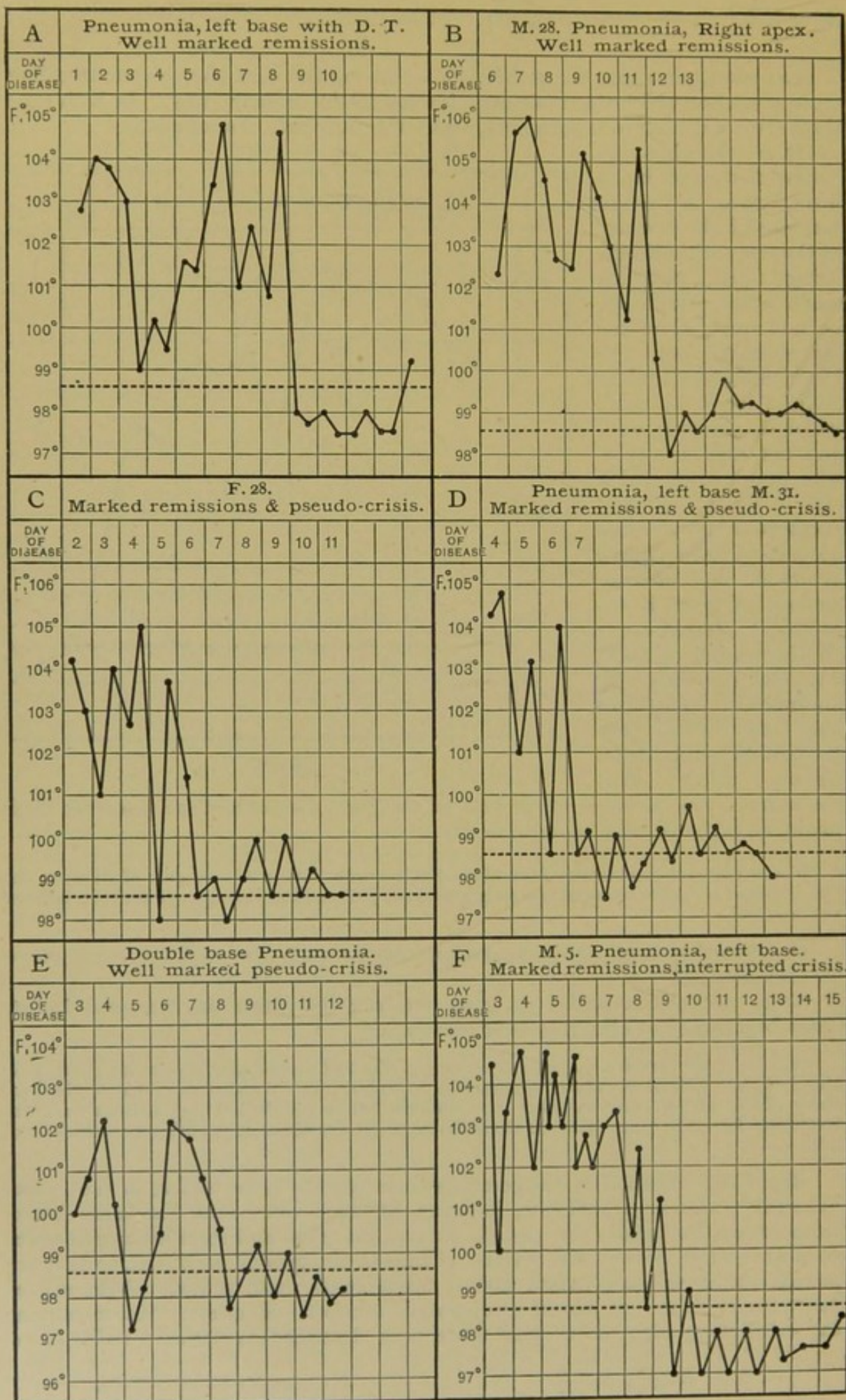


Fig. 66.

of some hours that the normal is reached. The length of the crisis varies a good deal in different cases, but the average is stated to be about sixteen hours. The distinction between sudden fall or crisis, and gradual fall or lysis, is purely artificial. All cases in which the temperature reaches the normal within twenty-four hours would be certainly said to end by crisis, while those in which the fall is spread over more than forty-eight hours are commonly said to terminate by lysis. The line between the two has been artificially drawn at thirty-six hours, and in this sense about two out of every three cases which recover end by crisis, but the estimates given vary from 60 to 80 per cent.

The statistics of Croce dealing with 461 cases give 62 per cent. as ending by crisis and 38 per cent. by lysis.

Of my own series of cases which terminated by crisis, in 22 per cent. the crisis was complete in twelve hours or less, in 45 per cent. in more than twelve and less than twenty-four, while in the remainder (33 per cent.) it was spread over from twenty-four to thirty-six hours. In one of my cases the temperature fell from 104° to normal in eight hours. Other cases are recorded in which the crisis has been complete in six or even four hours.

The following case is, I believe, unique, for in it the temperature fell from 103° to 96° in the space of one hour. I had been for some time interested in the fall of temperature during the crisis of pneumonia, and had given instructions that the temperature should in some of my cases be taken every hour, and it is to this that the observation is due. The patient was a lad of 9 years of age, who had been in good health, except that he had once or twice in his life had a fit. On Dec. 13th, having been previously as well as usual, he was suddenly seized with a fit, and became very ill. Remaining unconscious and growing feverish, he was brought to the hospital, and was admitted. He was found with a temperature of 103° , pulse 110, and respiration 52, and proved to have left apex pneumonia. During the next twenty-four hours he had two more fits and remained deeply unconscious and delirious. For the next two days the patient continued in much the same state of delirium and fever, and the signs at the apex

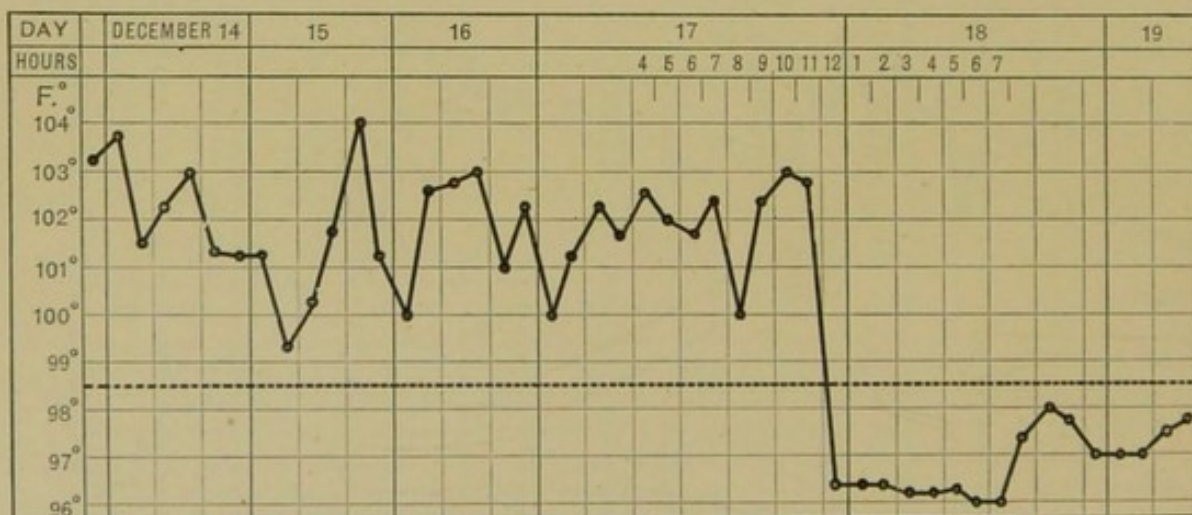


Fig. 69.
One hour crisis.

developed further. On the 17th—i.e., the fifth day of the disease—I gave orders that the temperature should be taken every hour. At eleven o'clock that evening the temperature was 103° , and at twelve o'clock 96.3° , having thus fallen nearly seven degrees within the hour. The nurse stated that at eleven o'clock the patient woke up suddenly in what she thought to be a fright, and seemed to be extremely ill. At one o'clock, when the house physician saw him, and confirmed the temperature taken by the nurse, the lad was sweating profusely, was less delirious, and seemed to be much better. The next day he was convalescent. Bronchial breathing continued until the 24th, after which the physical signs rapidly cleared up and he was soon quite well. If the pulse and respiration-rate be followed, it will be seen that the crisis was preceded, as it so often is, by a drop in the pulse-rate, accompanied in this case by a drop in the respiration-rate also—16th: Pulse 120, respiration 60.—17th (morning): Pulse 96, respiration 56; evening, pulse 80, respiration 36.—18th: Pulse 68, respiration 32.—28th: Pulse 80, respiration 24. On leaving the hospital the pulse was 70 and the respiration 20.

As a rule the drop is continuous and uninterrupted, that is to say, the fall once begun continues until the normal is reached. At times, however, it is interrupted, a fall of one or two degrees taking place and the temperature then either continuing at this lower level or rising again to the original level, or it may be higher, during the next twenty-four or thirty-six hours, after which the crisis develops in the usual way. This is spoken of as an interrupted crisis, or the disturbance of temperature is described as the *perturbatio-critica* or *pre-critica*.

The crisis, as stated, usually marks the commencement of convalescence, but this is not always so. It is always attended with some prostration; generally this is so slight as to attract no notice, being merged in the general improvement, but in bad cases the prostration may be so extreme as to amount to collapse, and this collapse may be fatal. It is as though the patient died of the shock of the crisis. Or, again, the critical fall of temperature may occur, but the pulse and respirations continue at their previous rate and the general improvement be absent. In such cases a fatal result may be anticipated.

In fatal cases, then, the temperature may either continue high and unchanged to the end; or the usual crisis may occur either immediately before death or even one or two days before; or, again, the temperature may rise rapidly, even, it may be, to 107° or higher. More commonly no crisis occurs, but the temperature falls gradually, the prostration increases, and the general condition becomes worse, until asthenia ends in death.

Critical days.—That pneumonia tends to terminate on the odd days, was taught by Trousseau, although the theory dates from Hippocrates. Statistics do not confirm this opinion.

The combined results of Jürgensen, Quincke and Fischer give a percentage of 51.7 for the odd days against 48.3 for the even, but all statistics alike show that the 7th is the day of preference for the crisis; after that comes the 5th and then the 6th, and on these three days together the crisis occurs in about 56 per cent.

Diagram constructed from the figures given by Sturges, collected by him from various sources. The black areas show the percentage number of cases which have their crisis on any given day, the actual percentage figures being written at the side. The upper limit of the dark area shows the total percentage of cases on any given day which on or before that day will have experienced their crisis. Thus in 73 per cent. the crisis has occurred before the end of the 7th day, and in 92 per cent. before the end of the 9th day.

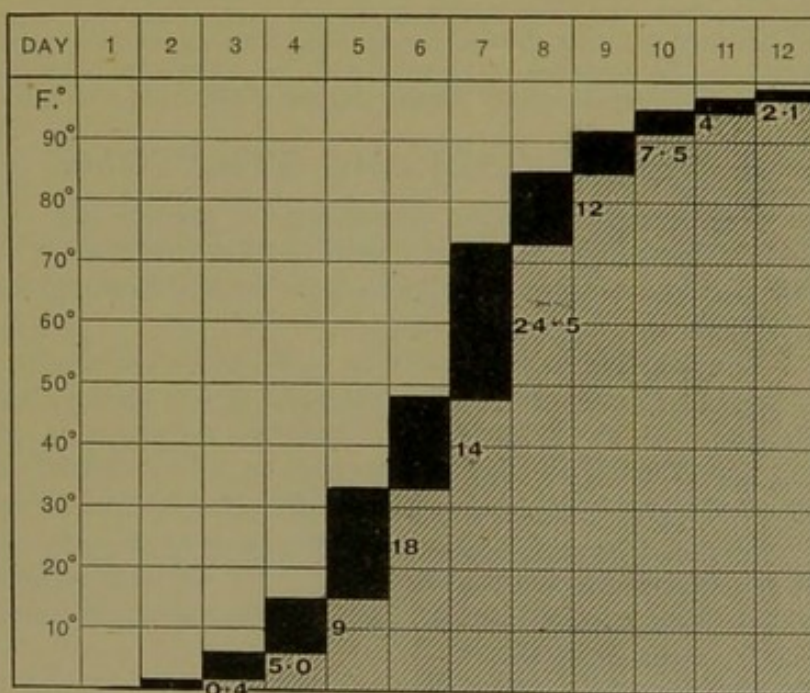


Fig. 70.

Pneumonia ends by *lysis* in about one-third of the cases which recover, *i.e.*, the temperature takes more than thirty-six hours to reach the normal. As a rule, lysis

is complete within forty-eight hours, but it may extend over three or even four days. If the temperature continue raised for longer than this, it is very probable that there is something beyond pneumonia to account for it, perhaps empyema.

The critical fall is followed, as in other fevers, by the ordinary reaction, and the temperature remains below normal for some days, or it may be some weeks. Much less importance is often attached to this period of depression than it deserves, for during this time the general functions and strength are, like the temperature, below normal, and convalescence cannot be said to be really established until the temperature has risen to, and is maintained at, the normal level.

There are several facts in the life-history of the pneumococcus which are of great interest in relation to the crisis of pneumonia. The crisis appears to correspond with the rapid attenuation of the pneumococcus and its death, for in cultivation the pneumococcus becomes inactive in the course of six days.¹

Its development is checked by high temperatures: thus in a gelatine culture the pneumococcus can not resist a temperature of 109° F. more than six hours, and a temperature of 105° for more than twelve hours; while a broth culture loses its virulence after twenty-four hours at 106°, and after five days at 105°.

Rabbits can resist pneumococcus infection as long as they are kept at high temperature, but succumb when the temperature falls.

Where the fever is unusually persistent it may be due to a creeping inflammation, which, as it resolves in one part of the lung, develops afresh in another. This is not uncommon in the pneumonia which follows influenza. In most instances it is due to some complication, of which the commonest is empyema. But there remains a small group of cases in which, without any cause, apparent at the time or afterwards, the temperature continues raised for some time.

For instance, in a child of 6 years of age, who passed through a severe attack of double pneumonia, the temperature continued hectic for six weeks, for which the most careful examination could find no explanation. In the end the child made a complete recovery.

B. THE LOCALISING SYMPTOMS.

Respiration.—The breathing of pneumonia is peculiar, being frequent, shallow, panting, and interrupted by the catch in the side and by the short cough. For this reason also the speech is jerky and broken, the words coming out in twos or threes and in short sentences.

The shortness of breath is not only evident to others but is felt by the patient, and is in the early stages, after the stitch in the side, the chief source of distress. In the later stages, when the dyspnoea is really greater, the patient is less conscious of it, and, where there is active delirium, it is remarkable how much effort the patients are capable of in spite of the dyspnoea; thus a patient may be so restless and struggle so violently as to require one or two nurses constantly at the bedside to keep him in bed, just as with delirium tremens. Towards the end in fatal cases Cheyne-Stokes breathing sometimes occurs: it is not common, but is of fatal omen.

The respirations number, on the average, in the adult about 40. A higher rate than this is in itself a cause of anxiety, for though some cases with a respiration rate of 50 or even 60 may recover, most of them are fatal. In children a rapid respiration rate has not the same importance, for in them it may reach 60, 70, or even 80 without exciting on that account any serious apprehension.

On the other hand the respiration may be slow throughout, perhaps hardly above the normal rate of health. This is rare and remarkable. A slow respira-

¹ Netter, *l.c.*

tion rate is only of grave significance when it develops towards the end and with signs of increasing dyspnoea. It is then an evidence of carbonic acid poisoning, and like Cheyne-Stokes breathing, is a warning of impending death.

The respirations are increased out of proportion to the pulse, so that the pulse-respiration ratio becomes of diagnostic importance. The ratio in health is about 4.5 to 1, and though in any fever the respiration and pulse are both accelerated, the same ratio is approximately preserved. In pneumonia, however, the ratio may be reduced to 3 or 2 to 1, or sometimes even below 2 to 1. It is rare for such a pulse-respiration ratio to be met with in any other disease than pneumonia, and when it is associated with high fever the presumption in favour of pneumonia is very strong.

In hysterical asthma the respirations may number as many as the pulse or even more, but there is then no fever. I have seen a case of acute rheumatic pericarditis with effusion associated with right pleurisy in which the ratio was 2 to 1. It may also be borne in mind that any pain in breathing, whether it be due to pleurisy or to some cause external to the pleura, will increase the rate of breathing and alter the pulse-respiration ratio, and the same is true in acute peritonitis where the movements of the diaphragm are checked.

Pulse-Respiration Ratios.¹

1. In adults during the fever :

$\frac{120}{40}$	$\frac{120}{42}$	$\frac{120}{44}$	$\frac{120}{52}$	$\frac{120}{50}$	$\frac{120}{64}$	$\frac{112}{36}$	$\frac{112}{60}$	$\frac{136}{40}$	$\frac{138}{60}$
------------------	------------------	------------------	------------------	------------------	------------------	------------------	------------------	------------------	------------------

In a case of bronchitis before the pneumonia, $\frac{P}{R} = \frac{88}{20}$;

with the pneumonia, $\frac{P}{R} = \frac{140}{40} \quad \frac{108}{44} \quad \frac{120}{40}$.

2. Cases in which the pulse was unusually slow :

$\frac{90}{54}$	$\frac{88 \text{ to } 104}{38}$	$\frac{100}{40}$	$\frac{108}{48}$
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3. Cases in which both pulse and respiration were unusually slow and that with extensive pneumonia : $\frac{56}{25} \quad \frac{52}{20} \quad \frac{50}{18}$.

4. Cases in which the respirations alone are slow are very rare.

In a girl of 11 with apex pneumonia and a temperature of 104° $\frac{P}{R} = \frac{140-156}{30}$.

5. Cases in which both pulse and respiration were unusually rapid ;

this being exceptional in adults : $\frac{160}{52} \quad \frac{140-160}{50} \quad \frac{160}{62} \quad \frac{160}{80}$;

but common in children : $\frac{132}{76} \quad \frac{160}{56} \quad \frac{180}{72}$.

In all the above cases recovery took place.

The following cases were fatal, and show the gradual acceleration in both respiration and pulse as death approaches.

$$(1) \quad \frac{116}{48} \quad \frac{120}{52} \quad \frac{132}{60} \text{ D.}$$

$$(2) \quad \frac{120}{40} \quad \frac{136}{48} \quad \frac{176}{60} \text{ D.}$$

$$(3) \quad \frac{112}{36} \quad \frac{120}{50} \quad \frac{130}{72} \quad \frac{134}{74} \quad \frac{136}{70} \text{ D.}$$

In a case of malignant disease of the œsophagus the usual $\frac{P}{R}$ was $\frac{38-48}{18}$;

when pneumonia developed it became $\frac{88}{44} \quad \frac{86}{42}$ D.

¹ West, *St. Barth. Hosp. Rep.*

The most marked instances of perverted ratio are met with in young children, but yet not so often as might be anticipated, for the pulse is so much accelerated in fever in children, and especially when the fever is associated with chest symptoms, that the ratio still remains fairly high; for example, even where the respirations reach 70 the pulse may run up to 160 or 180, giving a ratio still of $2\frac{1}{2}$ to 1.

Pulse-Respiration Ratio during Fever and Convalescence.

	Fever.	About Crisis.	Convalescence.	Remarks.
1	$\frac{130}{42}$	$\frac{96}{48}$	$\frac{72}{30} \quad \frac{68}{28} \quad \frac{68}{24}$	
2	$\frac{120}{44}$	$\frac{80}{44}$	$\frac{72}{50} \left\{ \begin{array}{l} 2 \text{ days} \\ \text{after} \end{array} \right. \frac{56}{26} \left\{ \begin{array}{l} 5 \text{ days} \\ \text{after} \end{array} \right.$	
3	$\frac{132}{36} \quad \frac{144}{52}$		$\frac{80}{44} \quad \frac{60}{36}$	
4	$\frac{144-160}{60} \quad \frac{130}{64}$	$\frac{100-112}{50-36}$	$\frac{80}{36}$	
5	$\frac{120}{54}$	$\frac{96}{40}$	$\frac{60}{36} \quad \frac{80}{28}$	
6	$\frac{132}{50} \quad \frac{144}{64}$		$\frac{68}{32} \quad \frac{66}{36} \quad \frac{92}{28}$	
7	$\frac{120}{48}$		$\frac{84}{34} \quad \frac{64}{24}$ for 3 weeks.	This low pulse was rapid for 3 weeks, $\frac{P}{R} = 2\frac{1}{2}$ to 1.
8	$\frac{120}{40}$		$\frac{80}{28} \quad \frac{72}{24}$	
9	$\frac{124}{38}$		$\frac{88-80}{44} \quad \frac{78}{26}$	
10	Two attacks of pneumonia $\left\{ \begin{array}{l} 1 \quad \frac{136}{52} \quad \frac{120}{50} \\ 2 \quad \frac{110}{52} \quad \frac{124}{62} \end{array} \right.$		$\left. \begin{array}{l} \frac{88}{40} \\ \frac{80}{36} \quad \frac{64}{24} \quad \frac{68}{25} \quad \frac{72}{24} \end{array} \right\}$	Both cases illustrate the gradual rise towards the normal in the $\frac{P}{R}$ as convalescence progresses.
11	$\left\{ \begin{array}{l} 1 \quad \frac{120}{52} \\ 2 \quad \frac{9116}{50} \end{array} \right.$		$\left. \begin{array}{l} \frac{84}{30} \\ \frac{78}{30} \quad \frac{78}{24} \quad \frac{80-96}{20-24} \end{array} \right\}$	
12	$\frac{126}{24} \quad \frac{108}{32} \quad \frac{96}{40}$		$\frac{64}{24}$	In this case the diagnosis of typhoid fever was first thought of on account of the slowness of the respiration, but $\frac{P}{R}$ gradually fell and the case ultimately proved to be nothing but pneumonia.
13	$\frac{108}{36} \quad \frac{104}{56}$		$\frac{70}{40} \quad \frac{52}{32} - \frac{68 \quad 72 \quad 82 \quad 88}{24}$	The gradual rise during convalescence in the $\frac{P}{R}$ is shown.
14	$\frac{120}{42}$	$\frac{48-58}{30}$	$\frac{64}{24}$	An extraordinary drop in the pulse is shown during crisis.

The pulse-respiration ratio may be of diagnostic value both in the adult and in the child.

The altered pulse-respiration ratio continues even for some time during convalescence, for though with the cessation of fever the pulse and respiration both fall, still the pulse falls more rapidly than the respiration, and at the crisis the drop in the pulse often precedes by some hours that in respiration.

Pain is a prominent symptom and rarely absent. It is sharp, stabbing and distressing, like the stitch of pleurisy, to which it is in most cases due. It is usually one of the earliest symptoms, following sharply upon the heels of the rigor, and when the rigor is absent it may be actually the first symptom of which the patient makes complaint.

According to Grisolle, it is present within the first twenty-four hours in 96 per cent. of the cases, and in 84 per cent. of those within the first twelve hours.

It may, however, occur late in the attack, or even be absent altogether; this generally occurs when the pneumonia is deep-seated, and does not reach the surface until late or not at all. It is, as a rule, most acute at first, and tends, as the affection lasts, to become less severe.

It is usually felt over the seat of the pneumonia, but over an area by no means co-extensive with the inflammation of the lung or pleura. With base-pneumonia it is referred as a rule to the lower part of the mid-axilla, but it may be reflected to other parts, *e.g.*, to the clavicle, shoulder, or even, it is said, to the opposite side; in children it is often referred to the epigastrium and the upper part of the abdomen, completely below the costal arch. With apex-pneumonia it is less severe and more frequently absent, probably because the movements of the upper part of the chest are so much less free and so much more easily inhibited. The peculiar seat of the pain seems to me to be best explained by the theory that the irritation of the trunk of the intercostal nerves within the chest is transferred to their peripheral distribution, for example, to the lateral branches in the axilla with base-pneumonia. This must at any rate be the explanation of the case, in which, besides the localised stitch, there is cutaneous hyperæsthesia over the whole affected side.

The pain greatly increases the distress and, if associated with much cough or hiccough, may become of real gravity from the suffering and increased dyspnoea it causes, and from the broken rest to which it leads.

The patients strive to relieve the pain by breathing short and shallow, by restraining cough, speaking little, and avoiding all movement. They lie in that position which is found most comfortable, as in pleurisy, but what that may be in any given case it is hard to foretell. Some patients find relief from pressure, and they will lie upon the affected side; in others, pressure aggravates the pain, and these will lie upon the sound side. But, as a rule, pneumonia patients lie neither on one side nor the other, but upon the back, with the head a little raised. Orthopnoea is rare and only occurs when there is some complication.

Cough, like pain, is a very early symptom. It is short, hacking, painful and restrained. It stands in no definite relation to the amount of expectoration, being often severe where there is little expectoration and slight when there is much. In the aged, weakly, or delirious, it is generally feeble and ineffectual, and it may be absent. Severe, and especially paroxysmal, coughing is rare, and only met with when the large air-tubes or the larynx are involved, but it becomes a grave complication on account of the distress and disturbance it causes. When provoked by swallowing, as it sometimes is, it may further aggravate the case by increasing the repugnance to food. On the whole, cough in the first few days does little good and may do harm, but

in the later days, and when resolution is progressing, it is of unmixed advantage by assisting expectoration; still it is not an essential symptom, for well-marked cases may run their course and rapidly recover in which cough has been entirely absent throughout, or so slight as to be almost overlooked. The worst indication given by the cough occurs when, with increasing signs of secretion in the tubes, the cough, which has been present and effective in bringing up secretion, becomes ineffectual or ceases altogether, and that with the signs of increasing obstruction to the tubes. Such a change is of the gravest significance, and indicates great danger, and probably death.

Hiccough is an unmixed evil, but fortunately of rare occurrence. It has the same clinical significance as in peritonitis and other abdominal affections. It is, I suppose, due to irritation of the diaphragm or phrenic nerves, but the exact causation is not clear, for it is absent more often than present even with extensive diaphragmatic inflammation, whether on the pleural or peritoneal surface. When present it is of grave omen.

The Expectoration is scanty and viscid, sticking to the side of the vessel into which it is expectorated, full of minute air bubbles and of a rusty colour. Such sputum is said to be pathognomonic, and is practically so; still something very like it is met with occasionally in the course of acute tuberculosis of the lung, and in some cases of passive congestion. It consists of the contents of the alveoli and infundibula, mixed with mucus from the bronchial tubes. The cells are for the most part red and white blood cells mixed with a few swollen epithelial cells derived from the alveoli, together with columnar and round catarrhal cells from the bronchial tubes, all in a condition of more or less granular disintegration, and many containing fat drops.

The contents of the alveoli and infundibula may be expectorated as such, and be recognised with the microscope in the sputum by their form. Small membranous or croupous casts of the smaller bronchi are also not uncommon, but the larger dendritic casts, such as are met with in plastic bronchitis, are really rare, though every museum contains specimens of them. To detect them the sputum must be received into water, and then the pellets which they form when expectorated uncoil themselves and are easily recognised. The sputum of course contains many microbes, and among them those that have been described as the pathogenic organisms of pneumonia (*cf.* Morbid Anatomy).

Chemical analysis shows the sputum to consist of mucin with a little albumen. A small amount of iron exists in the ash, the fixed salts, especially the chlorides, are in great excess, and the alkaline phosphates are absent.

Bamberger¹ gives the following comparative analysis of the sputum of pneumonia and of bronchitis:—

Pneumonia.	Bronchitis.
No alkaline phosphates.	10-14% of alkaline phosphates.
Na : K :: 15 : 41.	Na : K :: 35 : 20.
Sulphuric acid = 8 per cent.	Sulphuric acid = 3 per cent.

During resolution the sputum changes its character and becomes catarrhal in type.

It may be noted that sugar was found present by Beale and Walshe,² and tyrosin by Griesinger; the latter is probably a decomposition change.

Colour.—The typical sputum is, as stated, rusty, and this tint is due to the presence of red blood cells and of blood-colouring matter. The blood exuded undergoes the usual colour changes, and thus the sputum in the later stages may be brown, yellowish or green. A yellow or green tint may suggest the

¹ *Reynold's Syst.*, iii. 628.

² *Med. Chir. Tr.*, vol. xxxv.

presence of bile, especially if the patient be jaundiced. Bile-colouring matter may be demonstrated in some cases by the ordinary reactions, but in most these reactions fail and the colour is found to be due to altered blood only.

If abscess or gangrene occur, the sputum will become characteristic of these affections.

The amount of blood is rarely more than enough to give the sputum the characteristic rusty tint, but, if larger in quantity, it may make it of a dark red or purple colour. This is what is described as *plum* or *prune juice* expectoration.

Bright-coloured blood, as in ordinary hæmoptysis, is rare in pneumonia, and when it occurs is, with few exceptions, in small amount. In these few cases it may be in sufficient quantity to be fairly described as profuse hæmoptysis. This is met with, according to Walshe, only in connection with tubercle, and it has been suggested that in such cases the bleeding takes place not from the pneumonic but from the tubercular parts of the lung.

Huss has observed it with morbus cordis, and it has been attributed, but apparently without much reason, to atheroma of the pulmonary artery.

In the following cases the lungs were free from tubercle, and the bleeding was clearly only an extreme exaggeration of the ordinary hæmorrhage of pneumonia.

In the first, that of a young man 19 years of age, the attack began according to the patient's own account with the spitting of 5 or 6 ounces of bright blood, and the hæmoptysis continued till admission on the 2nd day; about 6 ounces were brought up on the 2nd day, the same quantity on the 3rd, and 3 ounces on the 4th. The sputum then continued rusty till the 7th day, when it ceased altogether, and the patient died on the 9th day. At the autopsy the whole right lung was solid and weighed 54 ounces. No trace of tubercle could be found in any part of either lung.

In the second case, also that of a young man, the hæmorrhage was still more profuse, amounting to about half a pint daily. This quantity was brought up for four or five days until the patient died. The hæmorrhage was so considerable as to make it probable that it came from an old cavity in the lung, though no physical signs except those of pneumonia could be found. As in the previous case, the autopsy revealed no lesion except acute pneumonia of the whole of one lung. Besides these two cases, which were rendered complete by the autopsy, I have notes of a few others in which similar hæmoptysis occurred, though to a much smaller extent. These patients recovered completely and gave no signs of tubercular mischief either at the time or subsequently.

Occasionally, as in the first case, the attack is stated by the patient to have commenced with the spitting of bright blood, which did not recur, the sputum remaining only rusty afterwards. From what has been said, there is no reason to dismiss these statements as mere exaggerations. Other things being equal, the amount and kind of hæmorrhage may be taken as a measure of the intensity of the inflammation, and thus becomes an important factor in prognosis.

Where pneumonia occurs in a patient with morbus cordis, and especially with mitral stenosis, it might be thought that copious hæmoptysis would be not uncommon, but this does not appear to be especially the case. Yet one or two remarkable cases of the kind have come under my observation, and will be referred to later.

Not infrequently the characteristic expectoration is mixed with a considerable amount of catarrhal secretion. This is not infrequent in aged, cachectic and feeble persons, as also in drinkers, and is due to the presence of bronchitis or cedema of the lung as a complication.

Amount.—The sputum is scanty, unless complications are present; probably not amounting to more than one or two ounces in the twenty-four hours. It may even be absent throughout. This occurs most frequently in children, for children under five years do not expectorate, but swallow what they cough up, and so, too, do many adults. Still, in the adult there may be no sputum, just as there may be no cough; and, indeed, the two not uncommonly go together. The

absence of expectoration is said to be more often seen with apex- than with base-pneumonia, but I do not think this is correct. If the sputum be large in amount and catarrhal, the prognosis is unfavourable; and still more so if, having once been copious, it become scanty or cease altogether, without any improvement in the condition of the chest.

The characteristic rusty sputum makes its appearance, in about 50 per cent. of all cases, within the first two days, but it may be seen quite early on the first day. On the other hand, it may not appear until the 4th or 5th day, or, as in a case of Wilson Fox's, not until the 10th day, and sometimes even not until the fever is over and convalescence is begun.

In the pneumonia which has occurred during the epidemics of influenza since 1890, in not a few cases the sputum has become rusty only during convalescence.

Once rusty, the sputum may continue rusty for many days, and last so even into convalescence.

I have notes of cases in which the sputum continued rusty after the temperature had fallen to normal, in one instance for two days, in another for three days, in two others for several days, the exact number not being specified, while in the two longest cases, the sputum did not cease to be rusty till the 18th and 23rd days respectively.

Sturges suggests that in such cases the rusty sputum comes from the spreading edges; though this may be true where the fever continues high and the pneumonia is extending, it is difficult to accept this explanation where the fever is gone and the pneumonia resolving.

C. THE PHYSICAL SIGNS.—In discussing the physical signs, we have to consider first, those yielded by the pneumonic parts, and secondly, those present in the other parts of the lungs. In the pneumonic parts again, the physical signs will vary according to the stage of the inflammation, being at first those of congestion or localised bronchitis, and later, when the pneumonia is extensive and fully developed, those of consolidation, viz., dulness to percussion, increase of vocal vibrations, bronchophony, and bronchial breathing. In the neighbouring parts, the physical signs are those due to the complementary congestion and its results.

Simple and true as such a general statement is, still there are many points of minor importance and certain exceptions to these general rules, so that it will be convenient to discuss the physical signs in detail systematically under the usual headings of inspection, palpation, percussion, and auscultation.

The Physical Signs in the Pneumonic Parts of the Lungs.—

Inspection.—The face has the flush of fever. It has been stated that this flush is most marked upon the side corresponding with the pneumonia, but it is quite as often on the opposite side, and, generally, equal on both. The eyes are bright and glistening, the pupils often dilated and the conjunctivæ somewhat suffused. The expression is anxious and indicative of pain.

The breathing is rapid, often panting, obviously difficult and painful, and the *alæ nasi* dilate with each inspiration.

If there be much dyspnoea, there is some degree of cyanosis, most evident upon the lips, tip of the nose and ears, and, as in other cases, beneath the finger nails; the veins of the neck are full, and pulsation is visible in them, as also in the epigastrium.

The patient lies as a rule upon the back with the head slightly raised. Sometimes the body is turned a little over upon the affected side, in order, no doubt, to give the rest of the lung freer play, but orthopnoea is unusual and indicates some serious complication.

The movements of the affected side are much impaired, and may be abolished over the seat of inflammation. In the early stage the side may be somewhat

retracted, but, in the later, it is usually expanded even to the maximum inspiratory distension, and this is the more striking from the absence of movements in it.

Palpation.—The deficiency in movement is often better felt than seen. The vocal vibrations are markedly increased, unless the tubes are plugged, and then they are absent. It is also not rare to feel pleuritic friction and rhonchus if they are well marked; and if the consolidation be extensive and superficial, the sense of resistance is greatly increased.

Thoracic pulsation over the region of consolidation has been described by Stokes, Graves, and others. It is attributed by them to the transmission by the solid lung of the impulse of the heart. Others have suggested that it is due to the pulsation of the arteries of the solid lung itself (Grisolle). Whatever the explanation, the condition is very rare.

Percussion.—The percussion-note is greatly impaired, or dull, but, even when absolutely dull, has not that stony character which is so common with large pleuritic effusions. The explanation usually given of this difference is that in pneumonia the bronchial tubes are patent and contain air, while in pleuritic effusion they are compressed and contain none, but if so, it is difficult to understand why, in cases of pneumonia, where the tubes are plugged with secretion or casts, the percussion does not also become stony.

If the pneumonia be deep-seated or of small superficial extent, dulness may be absent. The smallest superficial area of consolidation which will yield a dull percussion note is stated by Wintrich to measure not less than 2 inches (5 cm.) in extent and 0·8 inches (2 cm.) in thickness. With base-pneumonia the part to become impaired in resonance first is usually below the angle of the scapula in the posterior axillary line. The percussion note is usually not much altered until twenty-four hours after the rigor, but by that time it may be dull, and the dulness may then rapidly extend in the course of the next day to a large part of the side. Occasionally above the solid and dull parts the note has a *boxy character*, i.e., a flat semi-tympanitic resonance as if an air-containing cavity with thick walls were being percussed: this, which is described as the *tracheal tone*, is more frequently met with over the upper parts of the lung than the lower; in front, rather than behind; and on the left rather than on the right side. Over the parts adjacent to the consolidation, the percussion note is often tympanitic. When the consolidation is behind, this is often so marked in front that the seat of the affection may be diagnosed correctly in cases in which, owing to the condition of the patient, the back of the chest cannot safely be examined.

This tympanitic percussion note is often referred to collateral emphysema, but a similar note is obtained from lung floating upon a pleuritic effusion, and in that case it is the result of the relaxed condition of the partly collapsed lung. It is not improbable that the same explanation holds in the case of pneumonia, except that the loss of elasticity is then due to impairment of the nutrition of the lung in the neighbourhood of the inflammation. This tympanitic condition is as a rule only met with in the early stages of the pneumonia, and tends to disappear as the disease lasts, but it may continue for several days, or even until the pneumonia resolves. Over the tympanitic area sometimes the breath sounds have a somewhat amphoric character, so as to suggest the possibility of a cavity, which, however, the course of the case disproves.

Even a well-marked *Bruit de pot fêlé* may be obtained. This is but another phenomenon due to relaxed lung over a solid mass below.

The best *Bruit de pot fêlé* I ever met with occurred in a patient with heart disease, and was obtained from healthy lung lying over a greatly enlarged right ventricle.

Auscultation.—The auscultation signs vary with the stage of the disease. Thus, at first, immediately after the rigor nothing definite is audible. The breathing is unduly accelerated and is somewhat noisy, but the air enters freely

everywhere. If the stitch occur early, the movements are impaired in the corresponding part, and the air entry is in consequence defective there. After an hour or two fine-hair crepitation may be heard, but the chest is rarely examined early enough for this, and the affection has generally advanced to the next stage before the patient is seen. In this stage the respirations begin to lose their vesicular character, and the expiration becomes prolonged, at first in a very limited part of the lung, but soon over a rapidly extending area. As the consolidation increases, the expiration grows more and more distinct, until it becomes as loud and as long as inspiration, and both inspiration and expiration acquire that harsh grating character peculiar to *bronchial breathing*. So rapid is the consolidation that bronchial breathing may be fully developed within twelve hours from the rigor. When once established it usually persists until resolution commences, and then the respiratory sounds change back in reverse order to the normal.

The voice sounds, whether vocal or whispered, show a similar gradual increase in intensity as consolidation advances up to well-marked *bronchophony*, and in children the cry undergoes similar changes to the voice.

Pectoriloquy, though common, is not a constant sign. It is no doubt a peculiar form of bronchophony, and is often more striking with whispered sounds than with the ordinary voice. It is usually limited in extent, bronchophony being heard over a wide area, and pectoriloquy only here and there. When once present in any part it usually persists until resolution is established.

Crepitation in all its forms may occur in the course of the attack, but after the initial fine-hair crepitation has disappeared it is not unfrequently absent until resolution is established. The ordinary crepitation, when present, is due to the ordinary cause, viz., the presence of bubbles in the air-tubes, and when it is heard over the region of consolidation it has a sharp, clear, ringing quality which is very characteristic.

In a case in which nearly the whole right lung was consolidated, the crepitation could be heard clearly at the distance of 6 inches from the chest wall, and bronchial breathing at about half that distance.

Fine-hair crepitation is often spoken of as pathognomonic of pneumonia, probably because the conditions which the theory of its production requires can only, it is assumed, occur in the early stage of inflammation. However this may be, crepitation which cannot be distinguished from it by the ear is met with in some forms of dry pleurisy, capillary bronchitis, and early phthisis, in œdema of the lung, and in the partially collapsed lung over pleuritic effusion, while it may be heard even in pneumonia quite late in the affection at the edges of the consolidation. Lastly, it may be heard in perfectly healthy persons after they have been lying in bed and have not fully expanded their lungs for some time; then the first few inspirations are often attended with crepitation of the kind under consideration.

The physical signs vary much, according as the tubes are pervious to air or not. When the air-tubes are completely occluded, as they often are by secretion or in rarer cases by bronchial casts, both the breath and the voice sounds will disappear, and the diagnosis from pleuritic effusion by means of the physical signs alone will be very difficult, until, by the removal of the obstruction, the tubes are cleared, and the characteristic physical signs reappear.

It sometimes happens that though the onset of the illness be abrupt and characteristic, the physical signs are those of acute congestion of the lung; sometimes of both lungs, but more frequently of one only, and that this general congestion passes off as the disease localises itself in one or other lobe. This condition in the lungs may be compared with what is commonly observed in a furuncular inflammation of the skin, where, though the boil itself may not prove ultimately bigger than a marble, the early induration involves an area of several inches, and the zone of acute congestion is much greater still.

A few remarkable cases of this kind have come under my observation. The most striking have occurred in association with mitral stenosis.

A lady of 35, who had mitral stenosis of many years' duration, was attacked one evening with high fever and great dyspnoea. Her dyspnoea was so great that her life seemed in much danger. The only physical signs were those of acute congestion of the whole right lung, viz., rhonchus, sibilus, and abundant fine crepitation. She immediately began to expectorate pure blood, bright and frothy, and brought up several ounces in three or four hours with considerable relief. Gradually the signs of general congestion diminished, and became limited to the top of the upper lobe, where the ordinary signs of pneumonia subsequently developed. In spite of the acute and alarming onset, the pneumonia ran a short course, terminating on the fifth day, and the patient made a rapid recovery. A similar case is recorded by Kahane.¹

Where both lungs are involved in this general congestion, the cases closely resemble those of acute suffocative catarrh in children. An excellent instance of the kind is described in the chapter dealing with that subject (*q.v.*).

The Physical Signs in the Non-Pneumonic Parts of the Lungs.—

These are the result, on the one hand, of the complementary breathing in those parts, and on the other of the congestion which is the result of the extra work. The movements on the sound side are exaggerated, and that in proportion to the defect of movement on the affected side. The increased entry of air makes the respirations noisy, and of the kind described as puerile or complementary. The congestion may yield no physical signs, but if it be considerable it will give those of bronchitis, viz., rhonchus, sibilus, and crepitation, with frothy sputum. Localised bronchitis on the affected side in the parts adjacent to the consolidation is very common and of no special importance, but when the bronchitis is general and affects the opposite lung as well, it becomes important in respect of prognosis, although its gravity even then depends upon whether it preceded or followed the pneumonia. In the former case, where pneumonia has arisen in the course of bronchitis, the bronchitis forms an awkward but not necessarily a serious complication, while in the latter case, where bronchitis has developed in the course of pneumonia, the bronchitis is of the gravest import and the case generally ends fatally.

The tympanitic percussion met with above the seat of consolidation has been already referred to, but the opposite lung also occasionally becomes tympanitic; this may depend upon the bronchitis and be of no special significance, but it may occur without relation to bronchitis and be due to complete loss of elastic tone in the lung tissue. It has been called paralytic, though not paralytic in the sense of being due to loss of muscular power. This condition may be fairly compared in regard of prognosis and causation with tympanites of the bowel, and, like it, indicates grave neuro-muscular prostration, and is almost invariably of fatal omen.

In bad cases, where the patients lie for long upon the back without shifting, the blood gravitates to the lowest parts, and the base behind passes into the condition of hypostatic congestion. This may be sufficient to give a dull note on percussion and cause some exaggeration of the breath sounds, thus leading to the diagnosis of pneumonia there. If pneumonia does invade the opposite lung, it is usually the base which is attacked, and no doubt the hypostatic congestion is the predisposing cause, but the physical signs alone are not always sufficient to determine the diagnosis. Pneumonia at the opposite base is by no means rare, but it is often found when its existence was not expected, and, on the other hand, nothing but hypostatic congestion may be found when the physical signs seemed to point to pneumonia.

¹ *Ctbl. f. klin. Med.*, 1891, No. 10.

The physical signs, striking as they are, are really of less essential importance in diagnosis than the general clinical signs of the disease; for they are not present at first, and may not appear for some days, or even not at all, if the pneumonia remain deep-seated. I have once or twice seen them develop after the crisis had taken place. They may even be misleading, as where the tubes are plugged and the absence of voice- and breath- sounds points to pleurisy rather than pneumonia, or where there is so much bronchitis as to mask the more characteristic physical signs. So, again, where after some days' illness an effusion, and perhaps a purulent one, is found, the question may arise as to whether it had not been a case of pleuritic effusion *ab initio*, but the acute onset of high fever with the acceleration of the respiration and pulse will in most cases indicate the correct diagnosis.

The circulation.—The *pulse-rate* is accelerated, but not, as a rule, out of proportion to the fever; thus, with a temperature of 103° it is usually about 110 or 120. In *children* it is often much more rapid, running up in otherwise ordinary cases to 140 or 180, and in infants even to 200, and that without being of any very serious import. In the *adult*, however, any departure from the rule is of significance. Thus a pulse rate above 130 indicates danger, and the greater danger the more rapid it is, especially if at the same time it become weak, ill-sustained and irregular, for it is an evidence of cardiac failure. A rapid pulse is stated by Wilson Fox to be very common, if not constant, in drinkers. On the other hand, even with high fever the pulse rate may not be accelerated at all, but remain at 70 or 80, or, again, it may fall much below the normal; for instance, I have seen the pulse at 30 with a temperature of 103°, and this patient recovered. Still, as a rule a very slow pulse is, like a very rapid pulse, of bad prognosis. In discussing the pulse-respiration ratio reference was made to the *fall* in the pulse which occurs at and after the crisis, and it was shown that, though both pulse and respiration fall at the crisis, the pulse falls the most and continues to be slow for some time during convalescence. This is one of the signs of reaction, and until the pulse and respiration have both returned to the normal, convalescence cannot be regarded as established.

The arterial *tension* varies, but is, as in most sthenic fevers, somewhat raised. In grave, and especially asthenic, cases the tension falls below normal; if at the same time the heart be feeble, the pulse-beats will be irregular in force and frequency, indistinct from one another, and the pulse acquire a running character and be difficult to count; if, however, the heart be acting with moderate power, the pulse-wave is short and jerky, falling rapidly, while the up-stroke is fairly high, but not sustained. Under these conditions diastolic murmurs are often met with, but its importance will vary according to the circumstance under which it occurs; for instance, if it occur during the acute fever, it indicates loss of tone in the vessels, and is therefore a bad sign; if it occur when the fever is subsiding or has gone, it may be a good sign, as indicating that the high tension of fever is passing away and the heart recovering its power.

An intermittent pulse during the fever is of bad augury, but is of little importance after the crisis. It is common in children, after pneumonia as after other acute illnesses, and also in the aged, but it quickly passes off as convalescence is established.

The pulse, therefore, gives most important indications in pneumonia, and ought in every case to be carefully studied in respect of number of beats, character of wave, and tension of vessel.

One of the gravest dangers of pneumonia is the liability to *cardiac failure*. This is indicated by the usual symptoms; the action becomes feeble, the sounds

weak, the beats rapid, not infrequently irregular in force as well as frequency, and intermittent. Actual *dilatation* can be often demonstrated by the increase in the cardiac dulness. The dilatation may involve the whole heart or be limited to the right side. In the latter case the cause is to be found in the mechanical obstruction to the circulation through the lungs. In many cases the increase of the dulness to the right can be easily made out, but in others it is masked by the tympanitic percussion or the emphysematous condition of the lung on the right side, but in all cases alike pulsation will be marked in the epigastrium and in the vessels of the neck, and there will be considerable cyanosis. Many robust, full-blooded patients are in imminent danger from this cause from the commencement, and may die from over-distension and paralysis of the heart, unless saved by a timely cupping or venesection. In other instances the right-sided dilatation develops more slowly, but it is even more fatal, for it will not then yield so readily to treatment. When the whole heart is dilated, the cause lies in some condition affecting the whole organ; for example, it may be the result of the high temperature, or of some cardiac poison generated by the fever, or of some grave cardiac complication, as, for instance, acute pericarditis.

Cardiac failure is, of course, most likely to occur where the heart is already diseased. Thus it is very common and very grave in old people, whose hearts are so often more or less fatty; in those who are the subjects of valvular disease, both mitral and aortic; and in those in whom the muscle is unsound as the result of pericardial adhesions, of syphilitic or other myocardial disease, or of granular kidney. Among the autopsies of pneumonia it is remarkable how often the heart shows evidence of past disease; in other words, how fatal pneumonia is in those whose hearts are not healthy. It is probable that to this cardiac debility is to be referred the great mortality of pneumonia in those who are either actually suffering from, or convalescent from, other acute diseases.

Extensive general dilatation of the heart may, however, exist, as shown by the physical signs, without any symptoms of cardiac failure. This is especially the case with children, and in them it is often discovered by accident. It usually persists during the fever and vanishes rapidly when that is past. During the convalescence from pneumonia, as from other acute fevers, dilatation may be produced by exertion before the patient is strong enough for it, as, for instance, by permitting them to get up too soon.

The dilatation is not infrequently accompanied with murmurs, which, as usual, rapidly disappear with convalescence. These murmurs, as in other cases of dilatation, may be heard over the whole præcordium or at the apex, but most commonly at the left base. The pulmonary systolic murmur has been referred by some authors to the pressure upon the pulmonary artery of acutely enlarged glands,¹ of a dilated auricle, or of the consolidated lung itself. Without denying the possibility of these causes, they can be but rarely demonstrated, and there seems no adequate reason to look for other than the ordinary explanation.

The cardiac weakness suggests one or two rules of practice; viz., never during the acute stage to sit patients up to examine the back, but to roll them gently over from side to side, for the mere effort of assuming the erect position has not rarely led to fatal syncope; and not to permit a patient who has had a severe attack to get up too soon.

Changes in the Blood.—The amount of fibrin is greatly increased (from 4 to 10 parts in the 1000).

¹ Tordieu, *Journ. de Méd.*, 1890, No. 41.

There is only one other change of importance, and that is, the *leucocytosis*. This is well marked in most cases, beginning early, continuing throughout the

attack, and gradually passing away with defervescence. The leucocytes may increase up to 50,000 or 60,000 or more, and appear to be at their maximum shortly before the crisis.

The accompanying diagram, quoted from Billings by Osler, shows the relation between the fever and the leucocytosis.

Even the mildest cases of pneumonia show this leucocytosis to some degree. In malignant cases it may be very slight, or even absent. Conversely, if it be slight or absent in otherwise severe cases, the prognosis is thereby rendered more grave. Indeed, such cases rarely, if ever, recover. As no similar leucocytosis occurs in typhoid fever, malaria, or influenza, it possesses a diagnostic value in relation to these affections.

The pneumococcus is but rarely found in the blood.

Pneumonia never, I believe, occurs in the hæmorrhagic form, at least I know of no such case recorded. Hæmorrhage, except from the lungs, is rare in pneumonia. Epistaxis may occur, but not to any great amount, and the cata-

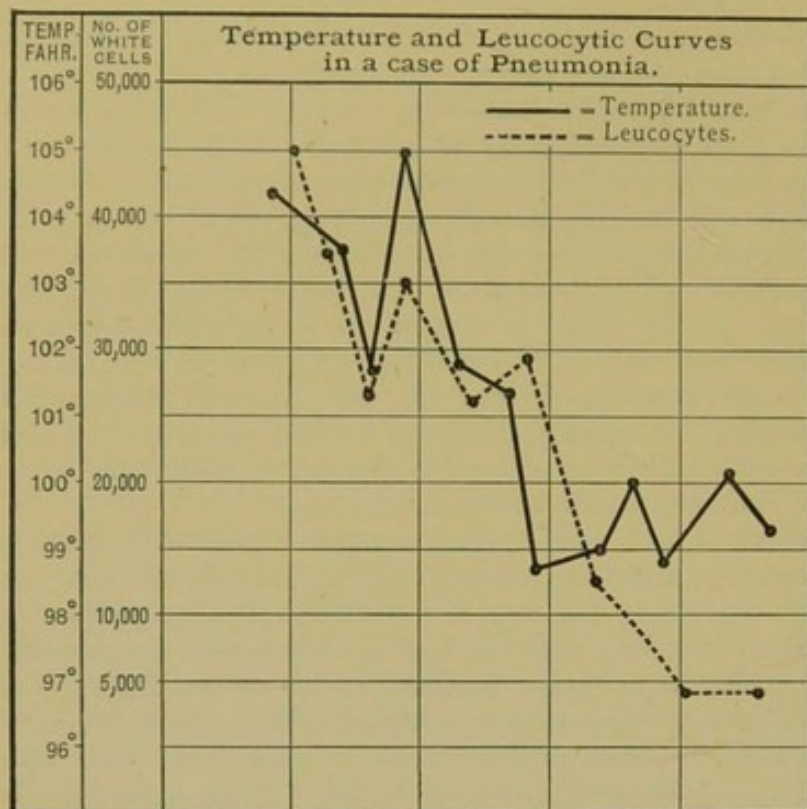


Fig. 71. — From Osler's *Med.*, p. 121.

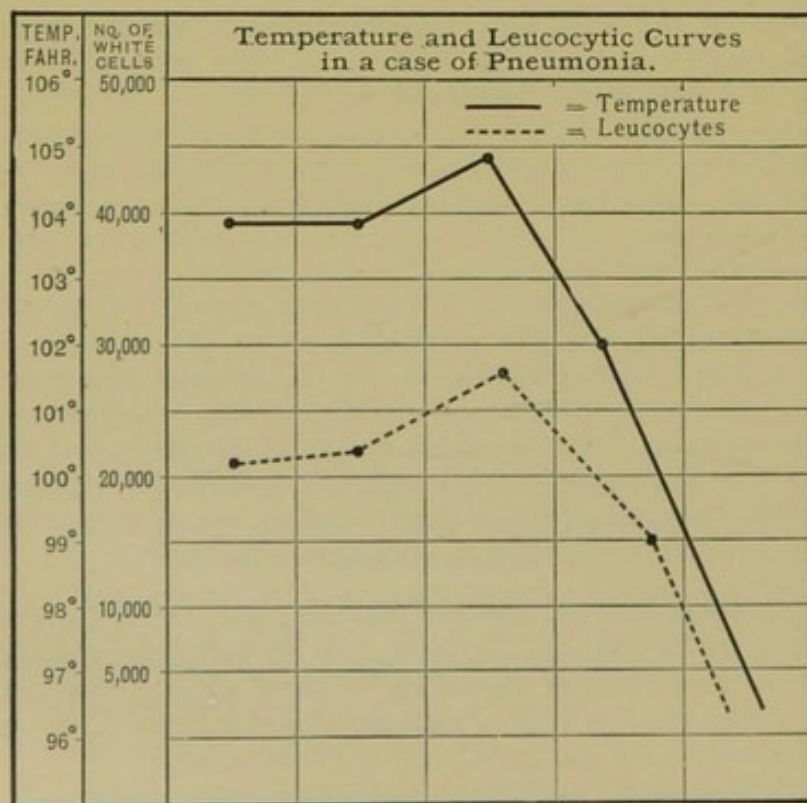


Fig. 72. — From a case of my own. M., aged 22.

menia, if they supervene during the attack, may be profuse, but they are more often scanty or absent.

Digestive System.—The general nutrition is but little affected, for the disease, though very acute, is short, and what is lost in flesh and strength during the few days of fever is quickly regained when the fever is over.

The *tongue* within the course of a few hours becomes thickly coated, and will probably by the second day have a dry central streak. It may sometimes remain moist, and in rare cases even be clean throughout. In bad cases the whole tongue may become dry, brown, and leathery, or even be black, cracked, and bleeding. The lips are dry and cracked, and in severe cases sordes may cover the whole mouth, gums, and teeth. In other cases, where the mucous membranes are not so dry, thrush may develop or aphthous ulceration. Thrush in adults is a bad sign and indicates greatly lowered vitality.

The *appetite* is greatly impaired or lost, and there may be so great repugnance to food that everything is refused except water. As a rule, however, the thirst is so great that liquids are grateful, and milk, especially if iced and diluted with effervescing water, is taken freely. Little children often refuse food entirely, and may have to be fed forcibly. Swallowing sometimes causes cough or increases the dyspnoea, and may thus increase the difficulty of getting food to be taken.

Vomiting is a very common early symptom, as it is in other fevers; it rarely lasts beyond the first few hours, and then is not severe. Sometimes it may continue for several days; this is more common in children, and, when accompanied with delirium and unconsciousness, may suggest the diagnosis of meningitis. It is rare in aged persons, but may in them be a very grave complication and even the cause of death. There is no reason to suppose that the vomiting in pneumonia is in any way different from that which occurs in other acute fevers, and it seems hardly necessary to refer it, as has been done (Huss), to pneumogastric irritation by the affection of the lung. In some cases the vomiting is due to the collection of mucus in the pharynx or larynx and the coughing which it excites; in others to the overloading of the stomach with milk, which, being undigested, ferments and causes much flatulent distension and sometimes aggravates the dyspnoea. Great relief may be given under these circumstances by removing the fluid and gas with a tube and washing the stomach out.

It must not be forgotten that the sickness may also be excited or kept up by some drug given too often or too largely. This is especially the case with digitalis, carbonate of ammonia, and ipecacuanha when it has been administered for some time or at short intervals.

The *bowels* are, as a rule, confined, but diarrhoea is not altogether rare, and may be very copious, so as even to raise the question of the presence of typhoid fever.

In certain rare cases the motions have a bright grass-green colour. Though met with only in severe cases, this is not necessarily of bad prognosis.

Diarrhoea occurred in my series of 100 cases in 6, and of these 3 died. I have only seen three or four cases of grass-green motions, but I do not think more than one died. This colour is due, no doubt, to altered bile, but it stands in no relation whatever to jaundice.

Osler, among his 105 fatal cases, found 5 of croupous colitis, but with no ulceration, and 1 of croupous gastritis. These are unique observations. Bristowe also records a case of dysenteric ulceration (*Path. Soc. Tr.*, viii. 61).

The *liver* is frequently somewhat enlarged, but the enlargement is associated with no other morbid change, except a granular condition of the cells, of the same kind as that met with in other cases of fever.

Jaundice in any great degree is rare. A little sallowness during the fever is common enough, but the motions contain bile. In this form it appears to be of no prognostic importance. Deep jaundice is, in all probability, of a catarrhal nature and runs its ordinary course, the colour persisting for some time after

the fever is past. Among the 100 cases, I have notes of well-marked jaundice in two only, and both of these recovered. In children jaundice is even still rarer than in adults.

When pneumonia is associated with pyæmia, or other septic disease, jaundice is likely to be present, as in other similar cases not complicated with pneumonia. It is then an evidence of general infection, but does not modify the prognosis.

Jaundice may, of course, occur in the course of any serious case, but it is not of itself of grave significance unless it be associated with some actual hepatic disease, as, for example, with cirrhosis. The patient will then in all probability die, but not because of the jaundice so much as of the cirrhosis.

Urinary System.—The urine is like that met with in other fevers. It is reduced in quantity, so as to be not more than a third of the normal amount, *e.g.*, 15 or 20 ounces. Its colour is high; the specific gravity is raised and ranges between 1020 to 1030, or may reach even 1035; and its reaction is strongly acid. It deposits on cooling a copious sediment of amorphous urates, more or less deeply coloured, with which a few crystals of uric acid, and occasionally of oxalate of lime, may be mixed. Epithelial cells from the urinary passages and leucocytes are also not uncommon in small numbers, but casts are very rare and do not occur unless there be nephritis.

The *urea* is stated by most writers to be very greatly increased even to the amount of 86 grammes (1330 grains) in the twenty-four hours. It is true that a few cases¹ are also recorded in which the amount of urea was below normal, but these are regarded as quite unusual and exceptional. Among the cases, however, which I have myself examined, I have not found any increase, but, on the contrary, a very remarkable diminution, in some instances even much below the amount which should have been excreted as calculated from the nitrogen contained in the food alone.

The results of my own observations were as follows:—During the fever the percentage of urea was constantly raised to between 3 and 4, or even in some rare cases higher. This percentage² in any given case remained fairly constant until the crisis, and then fell gradually to below the normal, rising slowly again to the normal as convalescence was completed.

The total urea was obtained, of course, by multiplying the percentage into the amount divided by 100. The volume of urine passed was greatly reduced, and, almost without exception, the total urea proved to be far below the average of health. The volume of urine, moreover, fluctuated greatly from day to day independently of any recognisable cause, and produced, of course, similar irregular fluctuations in the total amount of urea.

The total amount of urea often fell even short of the amount which should have been excreted as calculated from the food; for instance, in two cases taking the same amount of food the amount of nitrogen contained in it was, at a low estimate, 280 grains, which was equivalent to 600 grains of urea, but in the urine in the one case only a daily average of 212 grains was found, and in the other of 345 grains.

In these cases it seems that we have three alternative explanations—(1) that the food was not absorbed, or that the urea formed was eliminated by the bowels, but there was no diarrhoea, vomiting, or other symptom to support this view; (2) that the nitrogen was discharged in the urine but in some other form than urea, which escaped detection by the methods employed. Other investigation³ showed that this was not the case, but that practically the urea might be taken as the measure of the total nitrogen in the urine. (3) There seems, then, to remain only one other alternative, *viz.*, that the surplus nitrogen was stored up in the body possibly in the consolidation. If this were so we might expect to find a critical discharge at the end of fever. This, however, does not occur. I have only met with two instances in which at the crisis, or immediately after it, the percentage of urea rose. In both of these it reached 5 for one day, and in one of these only was the total amount of urea large, *viz.*, 900 grains, having been during the fever much reduced.

¹ Parkes on Urine, p. 271.

² "Elimination of Urea in certain diseases," *Med. Chir. Trans.*, vol. lviii.

³ Russell and West, *Proc. Roy. Soc.*, 1880.

It seems fair to conclude, therefore, that the statement usually made that the total urea is almost invariably increased in pneumonia requires considerable modification, and I believe that the same may be said of other fevers; in other words, that fever is not associated constantly with excessive elimination of urea.

The uric acid may be slightly increased, but this is not constant.

As regards *the phosphates and sulphates* the statements vary, but there is nothing constant.

The chlorides are generally greatly diminished or even absent. This reduction is met with also in other fevers, though in none is it so constant or marked as in pneumonia. It must be borne in mind that some of this reduction may be due to the diminution in the amount of chlorides in the food.

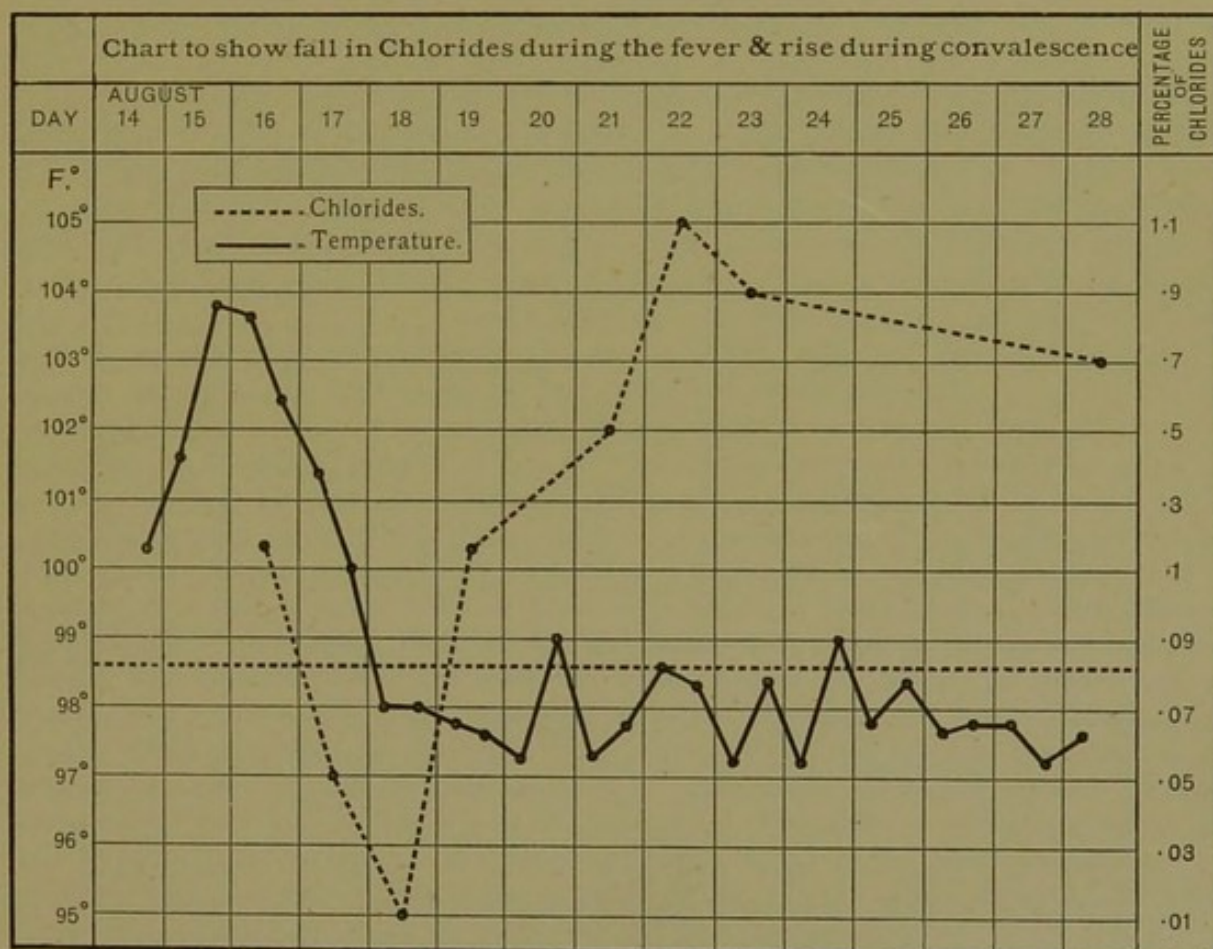


Fig. 73.

Albuminuria.—There is no fever except typhus and diphtheria which is so frequently associated with albuminuria as pneumonia. The amount of albumen is, as a rule, small, rarely exceeding one-twelfth, but I have seen it amount to more than one-third. It belongs to the category of febrile albuminuria, for it is present only during the fever, and disappears as soon as the temperature falls. It is but very rarely associated with blood or casts, and if so, depends upon nephritis.

Renal casts are stated by Jürgensen to be very common, and, indeed, rarely absent, but this is denied by others, and is certainly not in accord with my own experience, but I am afraid casts are not usually looked for carefully.

In the adult it is important to bear in mind the frequency with which *granular kidney* is found in fatal cases of pneumonia. It is difficult to say

whether pneumonia is more than ordinarily frequent in the course of granular kidney, but it is certainly more than usually fatal.

Acute nephritis is rare. Ziegler and Nauwerk found it in 13 out of 550 cases, or in 2·3 per cent., but among my own series of 100 cases there is not a single instance of it.

Nervous System. — *Headache.* — Severe frontal headache is one of the most constant early symptoms, as in other fevers; as the disease develops it usually becomes less severe, or, at any rate, is less complained of. It may, however, be a distressing symptom throughout, especially where in other respects the patient is not very ill.

Insomnia. — The sleep is always more or less disturbed and broken. The patients usually doze or sleep in short snatches as the pain, cough, or dyspnoea permits them. Now and then it happens that, without obvious cause, the patients cannot close their eyes, and lie wide awake hour after hour, weary and longing for sleep, but unable to get it. This is a condition of gravity and indicates a state of great nervous excitement, which will be followed by great depression, and not improbably end in death. Undesirable as it is to employ opiates or hypnotics in pneumonia, still sleep must be obtained in some way, and it will be wise in such a case to choose the least of two evils, viz., to run what risk there may be with narcotics, and procure sleep.

Delirium is very common; few cases of any severity run their course to the end without it. It varies much in degree and in kind, and seems to be nearly twice as frequent in apex- as in base-pneumonia. It may be due to the fever or to some poisonous substance generated by it, to hyperpyrexia, to acute complications of which pericarditis is the most important, to serious changes in the lung, *e.g.*, gangrene or suppuration, to debility, and, lastly, but very commonly, to previous alcoholism. It is usually a prominent symptom in the young and in the aged. In mild cases it may not amount to more than a little wandering or light-headedness at night; in other cases it persists throughout the day and is aggravated at night, as is especially the case in the aged and the feeble. In asthenic cases the delirium is of a low wandering character, with weak hallucinations, the patients lying only half asleep with the eyes half closed, picking constantly at the bedclothes or at something in the air. In very severe cases the patients may, from the beginning, be in a state of more or less stupor and be almost comatose, or they may pass into this condition from any of the other states of delirium. In another group of cases again the delirium may be of a much more active character, and be like that of delirium tremens. This is rare except in drinkers. In them delirium tremens develops with all its active and lively hallucinations, and cannot be distinguished from the ordinary *delirium e potu* except by the fever. Such cases are almost invariably fatal, but the delirium stands in no sort of relation to the extent of the consolidation, being sometimes extreme and the case ending fatally where the consolidation is too small to yield physical signs. The delirium is sometimes extremely violent, and it has not a few times been diagnosed and treated as acute mania.

The delirium may change its character, and thus the most active forms pass, as the case grows worse, into the low, wandering, asthenic forms, and the patient die at last almost comatose.

Wandering at night may sometimes develop for the first time, or, having disappeared, return again during convalescence. This is generally due to debility, and is cured at once by stimulation.

Children, old persons, and drinkers are usually delirious from the beginning. In all cases alike, gradually and steadily increasing delirium is of bad prognosis.

Subsultus tendinum is only met with in severe asthenic cases, among whom also *muscular tremors* are common. These tremors affect any part of the body which is put in motion, *e.g.*, the hands and arms, the lips or tongue. They are absent during sleep and when the parts are at rest, and are not necessarily associated with delirium. Similar tremors occur in the alcoholic cases, but in them depend upon the drink.

Tympanites, or extreme flatulent distension of the abdomen, deserves mention here, for it is neuro-muscular in origin and indicates the greatest nervous prostration. It is really a paralytic phenomenon, the muscular tone of the intestines being entirely lost. The distension which is the result leads to great pain from the stretching of the abdominal muscles, and to great aggravation of the dyspnoea from the pushing upwards of the diaphragm into the thorax. It is a very serious symptom, extremely difficult to relieve, and almost invariably fatal.

Cheyne-Stokes breathing is but rarely met with in pneumonia. It probably depends more upon the condition of the nervous system or of the heart than of the lungs, and is, I believe, also invariably fatal.

General cutaneous hyperæsthesia sometimes develops, but is of no clinical importance.

The predominance of nervous symptoms is especially marked in children. Thus convulsions often take the place of a rigor, and delirium sets in from the first. The child may lie almost unconscious night and day, recognising no one, rolling the head from side to side or putting the hand to it as if in pain, crying loudly at night and grinding the teeth. All these symptoms lead, naturally, to the thought of meningitis, and to make the difficulty even greater, strabismus has been recorded as sometimes present; the pupils, however, are equal and dilated. The diagnosis is usually determined easily in the absence of definite physical signs by the characteristic pulse-respiration ratio.

Disturbances of vision, photophobia, and occasionally amblyopia have been described, but are very rare and probably accidental.¹

Transient aphasia is stated to occur, lasting a few hours, but occasionally four or five days, but ending almost invariably in complete recovery. This is a rare complication in this country; most of the cases are recorded in French literature.

The Skin.—The skin is hot and dry, and gives a pungent burning sensation to the hand which is very characteristic. Now and then the skin is moist throughout, and then the temperature may be very much higher than it appears to be to the hand.

Sweating during the fever is very rare and of bad omen. In fatal cases, as death approaches, the body becomes bathed in a cold, clammy sweat; this is an evidence of far advanced carbonic acid poisoning, and is met with when the cyanosis stage has passed into that of pallor.

Sweating is the rule at the crisis, and is the commonest of the so-called critical discharges. It is most profuse when the crisis is abrupt, and is then often associated with considerable prostration and faintness. It commences on the forehead, side of the nose, and palms of the hands, and rapidly becomes general. It lasts usually from three to four hours, but when the crisis is prolonged, or the case ends by lysis, the skin becomes gradually moist and profuse sweating is absent. When profuse, it often leads to a copious development of sudamina, especially upon the front of the chest and abdomen.

¹ Chantemesse, *Bull. et Mém. de la Soc. Méd.*, Dec. 1893.

Herpes.—No other fever is so frequently associated with herpes as pneumonia, but even in pneumonia it is by no means constant. The actual percentage is differently given by different authors, and varies from 13 to 43. In my own 100 cases it occurred only in 12. It most commonly develops round the mouth, and usually, it has been said, upon the same side as the consolidation in the lungs, but this is not the case, for it is quite as frequently on the opposite side and often in the middle line. Besides this, it is often found in other parts of the face, upon the chin, side of the nose, and the cheeks. It has been described also upon the arm, the back, round the anus,¹ upon the cornea, and I have seen it upon the sternum. It may be also widespread,² and sometimes assume the form of herpes zoster. Wilson Fox observed a similar rash upon the tongue, soft palate and tonsils.

It usually appears from the second to the fifth day. It takes twenty-four to thirty-six hours to develop, and the traces of it may remain for a week or ten days after the fever is past. It may even develop after the temperature has become normal.

In one of my own cases it appeared round the mouth on the second day after the crisis, and a second patch developed on the manubrium sterni two days later still. Wagner records the case of a man who had three attacks, the first, on the third day, on the lips; the second, on the sixth day, along the left lower jaw; and the third, on the tenth day, on the left side of the neck.

Besides herpes, other rashes have been recorded, chiefly, as would be expected, of an erythematous type.

I have seen four separate attacks of well-marked but transient erythema develop in a child during pneumonia with fever of eight days' duration.

Acute pemphigus and localised gangrene of the skin have been described, but if not actually pyæmic in nature, they are associated with the septic form of pneumonia and are probably mere accidents.

The same applies also to purpura, which is even rarer still, but, as already stated,³ no case of what might be strictly called the hæmorrhagic form of pneumonia is known.

Eruptions which have been present previously may, as in other fevers, temporarily disappear during pneumonia and return again when the pneumonia is past. This is especially the case with psoriasis, pemphigus, and eczema, and it has been also observed⁴ with early syphilitic eruptions.

A little child of 5½ had been under my care for two years with pemphigus, and though sometimes better and sometimes worse, had never been really free from blebs. She was attacked with pneumonia of the left apex with acute symptoms; the blebs which previously existed rapidly vanished, and no new ones formed until eight days after the crisis. One bleb appeared in the dorsum of each foot; the next day blebs appeared on the labia and face, and subsequently on other parts of the body.

COMPLICATIONS.—Under this head are ranged those affections which are liable to arise as the consequences, more or less direct, of pneumonia. Many of them are of course pulmonary, *e.g.*, pleurisy, bronchitis, gangrene and abscess; others are possibly due to the direct spreading of inflammation, as pericarditis; others, again, are of a more general character, such as acute affections of the heart, kidneys, or meninges.

Pleurisy is almost part of the disease, for it is never absent unless the pneumonia be deep-seated, so that it does not reach the pleural surface. In most

¹ *Arch. d. Heilk.*, viii. 748.

⁴ *Prog. Med.*, 1885, Nos. 36 and 38.

² "Purpura with recovery," *Virch. Jahrb.*, 1887, ii. 349.

³ Grisolle, p. 465.

cases the pleurisy is dry, and disappears rapidly as the pneumonia resolves, leaving, at the most, nothing but a few adhesions behind. When an effusion develops it does not become evident, as a rule, until the fourth or fifth day. When the tubes are plugged the diagnosis is difficult, and can often only be settled by puncture. Pneumonia may leave effusion behind, and it is not rare for patients to come under observation with effusion, in which, from the history of acute onset, the affection probably commenced with pneumonia. Empyema may, it is true, set in with very acute symptoms, but, as a rule, these cases do not present much difficulty in diagnosis.

Dulness after pneumonia does not necessarily mean fluid or even thickened pleura, for it may be due to imperfectly resolved lung.

The general clinical condition may assist the diagnosis. If the temperature continue raised after the pneumonia is past, pleurisy should be suspected; but on the other hand effusion may develop after pneumonia and the temperature remain normal, or the temperature may continue raised without the presence of effusion in cases of imperfect resolution of the lung. The most conclusive cases are those in which after an apex-pneumonia the base becomes dull, the fluid, derived no doubt from the upper part of the pleura, collecting in the lowest parts.

The nature of the fluid can only be suspected with greater or less probability, and can only be determined with certainty by puncture.

Some authors state that empyema is most frequent, but my own experience leads me to adopt the general opinion that the effusion is usually serous.

Many of the serous effusions are small, and most of them, whether small or large, disappear spontaneously without operation. On the other hand, most of the empyemata are often operated on, so that it is, no doubt, true that most of the cases which require operation are empyemata. It is probably this which explains the conflict of statistics. There are no general statistics which conclusively settle this point, nor if any general statement could be made would it help much in any given case.

Out of the 100 fatal cases previously referred to, serous effusion was found in 3, sero-purulent fluid in 5, and purulent in 1. In 3 cases the effusion was associated with recent pericarditis, and in 1 of these the pericardium contained 15 ounces of dirty turbid fluid, and recent vegetations were present on the mitral and bicuspid valves.

Double pleurisy is not altogether rare, even with one-sided pneumonia.

In a case not included in this series, following right base pneumonia, both pleural cavities and the pericardium contained several ounces of pus.

Serous effusions, if small, are of no importance, and rapidly disappear; if larger, most of them also disappear spontaneously, but require longer time; if they demand paracentesis, they too recover as a rule in the usual way, leaving more or less of adhesion behind. In some instances the fluid develops very insidiously, its presence being only betrayed by the gradually increasing shortness of breath.

Empyema is, I think, not very common after pneumonia.

Thus in the St. Bartholomew's statistics it occurred in only 15 cases out of 1165, or in 1·3 per cent., and an analysis of 700 cases at St. Thomas' gives a percentage just under 1. Sello's (*l.c.*) percentage is much higher, 4·5.

The absolute frequency cannot be determined except by systematic puncture, especially if, as there is good reason to believe, small localised empyemata not unfrequently spontaneously disappear. Empyema after pneumonia is commonly localised, and is more frequent in children than in adults. It is to these two facts that the relatively favourable prognosis may to a great extent be attributed. Even empyema after pneumonia may develop insidiously.

A man of 20 was attacked with what appeared to be acute pneumonia of the right base. He developed herpes on the second day, and the temperature fell to normal by lysis on the ninth day of illness. He appeared to be convalescent, and there was nothing in his condition to draw attention to him, when, on the sixth day after the fever had left him, he died quite suddenly. At the *post-mortem* the base of the right lung was found in the condition it might have been at such a time after acute pneumonia, but there were several ounces of pus in the right pleura, a smaller quantity in the left, and a considerable quantity in the pericardium.

These effusions developed in great part, if not entirely, after the temperature had fallen. The case illustrates the fact, now well known, that it is possible to have active suppuration in the pleura without fever.

A good deal has been written in recent years upon the empyema which follows pneumonia under the name *meta-pneumonic empyema*, and it deserves the importance which has been given to it on account of its relatively favourable progress.

Thus Netter¹ states that out of 43 cases treated in the usual way following pneumonia, only 1 death occurred, giving a mortality of 2·3 per cent.; while of 117 cases treated in a similar way, due to other causes, 29 died, giving a mortality of 25 per cent., and although these figures perhaps somewhat magnify the difference which exists, they represent what is a fact, viz., that empyema after pneumonia has a much more favourable prognosis than empyema resulting from other causes.

Bacteriological investigation of empyema has shown that a very large number of these cases are associated with the presence of pneumococci, and that even when there has been no definite history of pneumonia. These facts may explain, to some extent, the difference which exists between the results of empyema in the adult and in the child; for it is found that two-thirds of all cases of empyema in children are associated with the pneumococcus, but only one-third in the adult.

The relative benignity of pneumococcus-empyema has been connected with the short vitality of the pneumococcus, which loses its virulence in a few days in the lung (eight or nine days), though it retains it a little longer in the pleura. Similar differences have been observed in artificial cultivation, the pneumococcus retaining its activity longer in bouillon than when grown on solid media.

Whatever the explanation, the fact remains that the prognosis of empyema after pneumonia is relatively favourable.

The results of operation are eminently successful, and even in cases where no operation is performed recovery frequently takes place; in some, after perforation of the lung and expectoration of the pus; in others, a single paracentesis or a second may suffice to cure, while in a few instances the pus is spontaneously absorbed, and that apparently completely.

For more detailed information on these points reference must be made to the article upon Pleuritic Effusion.

Bronchitis.—Bronchitis is always present to some extent in the consolidated parts and in their neighbourhood, but it does not assume the importance of a complication until it is general, *i.e.*, until it affects both sides. The collateral congestion of the rest of the lungs, which is the result of pneumonia in any part, produces no physical signs so long as the rest of the lungs is equal to the extra work thrown upon it, but so soon as it fails, the clinical signs of congestion, *i.e.*, of bronchitis, appear. Bronchitis thus becomes an evidence of functional incompetence, and though the breakdown may be as much cardiac as pulmonary, its meaning is the same, for such cases end almost without exception fatally. The secretion is abundant, and greatly increases the amount as well as alters the character of the expectoration. The tubes become more and more choked, and the patient dies of gradually increasing suffocation.

¹ *Bull. d. l. Soc. Méd. d. Hop.*, 1880, Jan. 11.

The tympanitic percussion met with over the *non-pneumonic* lung may be due to the transient emphysema caused by the bronchitis, but not always, for it may occur when no bronchitis is present, and it is then the consequence of loss of pulmonary tone or tension, and is, as stated, a very ominous sign.

Gangrene, abscess, and interstitial pneumonia as rare complications, or results of pneumonia, have been already referred to when speaking of the morbid anatomy.

Pneumothorax, as the direct result of pneumonia, is perhaps the rarest of all complications. It can only occur when the tissue has suppurated, or where abscess or gangrene has developed, but in the case of abscess the pleura has usually been sealed by previous adhesions, and in the cases of gangrene death takes place before the pleura has had time to give way. Very few cases are recorded, but two have been recently published by Talma,¹ and in one of them recovery took place.

The Heart.—Cardiac failure, though really the most important complication of all, is so common a result of pneumonia that it is often described rather as a part of the disease. It is recognised in the usual way, and as it rarely leads to any lesion of the muscular substance, there is nothing to be seen after death except dilatation. The commonly described cardiac complications are three—pericarditis, acute endocarditis, and thrombosis.

Pericarditis is not rare, but its frequency during life must not be reckoned from the results of *post-mortem* observation.

In my own 100 fatal cases, acute pericarditis was found in 8. Leudet gives 7·2 per cent.; Osler, 5 per cent.; Austin Flint, 6 per cent.; Banti, 5·4 per cent.; Netter, 8 per cent. The series from St. Thomas' Hospital yields 11 per cent.; Ormerod's, 12 per cent.; but the highest percentage of all is given by Sturges, viz., 16·6.

Roughly speaking, we may say that pericarditis is found *post-mortem* in 1 case out of 10.

The percentage during life is much smaller.

In my other series of 100 consecutive cases it were diagnosed in 3; the St. Thomas' series yields 2·7 per cent., with which Behier's figure, 2·5, almost exactly agrees.

The slighter degrees of pericarditis may, of course, be easily missed during life, but making all allowance for clinical oversight, it is obvious that pericarditis greatly increases the mortality.

The pericarditis is in most cases dry, *i.e.*, with hardly any effusion. When effusion occurs it is not rarely purulent.

In one of my cases 15 ounces of pus were found *post-mortem*, and in another, not included in that series, about 50 ounces were found, and in neither case was it suspected during life.

The pericarditis is often latent, *i.e.*, produces no symptoms, but it may be associated with delirium, as in rheumatic fever, so that the sudden occurrence of delirium or its sudden exacerbation should always suggest the possibility of pericarditis. It is a grave complication, and in many cases leads to a fatal result.

Acute endocarditis.—Fresh interest has been excited by this complication since the description of ulcerative endocarditis in association with pneumonia, and the discovery, in the vegetations, of the pneumococcus.

Netter found pneumococcus-endocarditis twice in 26 fatal cases of pneumonia, and Banti twice in 37 fatal cases. Osler records acute endocarditis as present in no less than 16 out of 105 fatal cases, and in 11 of these it was ulcerative. This must be a very unusual experience.

¹ *Virch. Arch. f. klin. Med.*, x. 306.

Instances of old morbus cordis in which vegetations are present must be excluded from this category, and this being done, Sturges and Coupland found only 3·5 per cent. of the fatal cases in which recent vegetations could in any way be connected with the pneumonia.

In my own series it occurred once only, the vegetations being found on the mitral, and also on the tricuspid valves, and was associated with purulent pericarditis.

In most cases it appears to give rise to no special symptoms, but in the ulcerative form the usual symptoms would arise, viz., a pyæmia-like state, with embolisms in various organs.

I do not know of any evidence to show that endocarditis after pneumonia ever leads to chronic valvular disease as the rheumatic form does, probably because these cases are almost invariably fatal.

Cardiac Thrombosis.—The clot which is found in these cases is not *post-mortem*, but clearly formed during life, for it is decolourised and tough. It is often almost white and felted in between and behind the columnæ corneæ. It is flat and thin, its shape being that of the cavity at the end of systole. It often extends through the auriculo-ventricular orifice into the auricle, where it expands again, and may be similarly attached to the walls. Its commonest seat is in the right side. If it extends slowly into the pulmonary artery it will cause gradual obstruction of either the main trunk or of one of the branches; in one of my cases, the pneumonia being at the apex, the artery going to the lower lobe was completely plugged.

The whole clot may even be detached *en masse*, and the case end suddenly, as with embolism of the pulmonary artery.

Such clots as these are usually found in patients who have been slowly dying for some days, but they may develop even during what seems to be normal convalescence, and they may then lead to sudden and quite unexpected death, either from failure of the heart or from embolism. These clots are at any rate not rare, and they were present in no less than ten out of my hundred fatal cases.

The following complications are all of them of great rarity.

Acute Nephritis is so rare that it would be of little practical interest were it not for the discovery, in some of the cases, of the pneumococcus in the inflamed tissues. Instances of this are described by Netter, and are used by him to support the view that pneumonia is a general infective disease.

Its occurrence during life is recognised by the usual signs, viz., albumen, blood, and casts in the urine, but general œdema seems not infrequently absent. Sturges describes two cases of this kind, one of which recovered and the other died. In one case of mine nephritis was discovered *post-mortem*, but the signs during life had been indefinite. Other cases are recorded by Wilberg.¹ Nauwerk states that the inflammation first attacks the glomeruli as in scarlatinal nephritis, and then the tissues round the veins and capillaries of the cortex.

Milder forms of *blood poisoning* are seen in the swelling of the joints occasionally met with, and perhaps also in tonsillitis, the latter being most frequent in those cases which arise from drain poisoning.

Parotid abscess, in the very rare instances in which it occurs, is probably also pyæmic.²

Wilson Fox met with it once only, in a girl, and she recovered. Sturges describes another case which also recovered. Behier³ records three cases, but they were all fatal; Aldrick one, in a man of 43, who recovered.

Otitis media sometimes develops, but it is one of the rarest complications. It is always suppurative, and as it usually goes with other septic symptoms, it is present as a rule only in fatal cases. The pneumococcus has been found in the pus here also (Gamaleia, Weichselbaum).⁴

Although it is true that the pneumococcus has been found in all these cases, still, considering how rare all these complications are and how frequently they are associated with endocarditis or disintegrating clots, it seems highly probable that they are all alike due to secondary infection from these sources, and not improbably often by other organisms than the pneumococcus.

¹ *Virch. Jahrb.*, 1885, ii. 155.

² *Med. News*, 5th Nov. 1898.

³ *Cf.* Coupland and Sturges, p. 112.

⁴ *Monatschr. f. Ohrenheilk.*, 1888, p. 149.

Meningitis is extremely rare. It did not occur once in either of my two series of 100 cases. Osler, however, met with it in 8 out of his 105 fatal cases. This is as unusual as is his experience of acute endocarditis, and as 5 of the 8 cases were associated with that affection, it is probable that these, at any rate, were embolic.

The inflammation appears to be always purulent, and in the pus the pneumococcus has been found, but meningitis associated with the pneumococcus has been found without pneumonia.

In many of the cases meningitis has been found on *post-mortem* examination without having been suspected during life, but when symptoms are produced they are of the ordinary kind.

Epidemic cerebro-spinal meningitis is frequently associated with pneumonia (9 times in 30 cases, Immermann), and in a considerable number of cases, in which there has been no pneumonia, the pneumococcus has been found in the pus; I think it would be safer to say an organism like the pneumococcus, and difficult to distinguish from it, rather than to assume that it is the pneumococcus itself; for it is difficult to accept the view that the same organism can be the cause of two diseases so different in themselves and so rarely associated except when cerebro-spinal meningitis is epidemic.

Pneumonia, like other severe illnesses, is sometimes followed by grave mental symptoms, *e.g.*, mania, melancholia or imbecility. As a rule, the mental functions are in the end restored, though convalescence may require months of care and watching.

Pyæmia with multiple abscesses has been recorded. Jaccoud¹ has published two such cases, but in these the germ found was not the pneumococcus, but the streptococcus and the staphylococcus pyogenes.

In connection with this subject an interesting paper by Foulerton, on the general pathology of pneumococcus infection, may be referred to.²

Relation of Pneumonia with other Diseases.—In this, two questions are involved, first as to the effect produced by any given disease upon the pneumonia, and secondly, as to the effect produced by the pneumonia upon it.

This much can at any rate be said at once, that the association of pneumonia with any pre-existing disease greatly increases its gravity, even, according to Huss, to the extent of making it four times as fatal as in uncomplicated cases.

In my 100 fatal cases 23 showed some chronic disease of the lungs or pleura, 8 some chronic affection of the heart, and 10 of the kidney.

Bronchitis.—Pneumonia is frequently preceded by bronchitis, but hardly so often as stated by Grisolle, *viz.*, 25 per cent. The pneumonia which follows bronchitis, as a rule, neither begins nor ends as abruptly as usual; the temperature shows greater remissions, and is of a more hectic type and falls gradually. In these respects, as well as in the liability to relapse, it shows more resemblance to the broncho-pneumonia of children. The difference in prognosis according as the pneumonia precedes or follows the bronchitis has been already spoken of.

Chronic emphysema is frequently found in fatal cases. It greatly increases the gravity owing to the increased danger of cardiac failure, but it does not otherwise modify the course.

The same may be said of *chronic pleurisy*.

In 8 out of the 100 fatal cases, both pleural cavities were obliterated by old adhesions, and in 8 others emphysema was present.

Phthisis.—Phthisis certainly does not increase the liability to pneumonia, and but few cases of active phthisis die of it. The acute febrile attacks which are frequent in the course of phthisis are of tubercular nature and belong to the category of catarrhal rather than of croupous pneumonia. Yet in fatal cases of pneumonia old tubercular lesions are very often found. This means, however, no more than that patients with lungs damaged from any cause—chronic pleurisy, emphysema, or tubercle—are bad subjects for pneumonia. When pneumonia does occur in phthisis, it often runs its ordinary course and may resolve completely. Not rarely, however, it seems to start the tubercular process into fresh

¹ *Gaz. d. Hop.*, Paris, 1886, No. 6^e.

² *British Med. Jour.*, 21st Sept. 1901.

activity, so that the disease makes rapid progress. From all that we now know it seems clear that pneumonia cannot caseate, and that caseation, when it occurs, is due to infection with the tubercle organism.

In the 100 fatal cases there was only 1 with active phthisis, but 5 with old tubercular lesions, and in the other series 1 case only had chronic cavities at both apices and recent tubercles in other parts of the lung, the patient dying with double base-pneumonia.

Morbus Cordis does not specially predispose to pneumonia, but it greatly increases the risk. Among my fatal cases, 8 had chronic heart disease, and 10 others chronic pericarditis, making 18 in all.

Sturges' figures yield the percentage of 11. He draws attention also to the frequency with which the pneumonia is double. Thus half (53 per cent.) of the cases of double pneumonia were associated with chronic morbus cordis. The pneumonia was right-sided in 16 per cent. and left-sided in 10·7 per cent.

Not only does pneumonia increase the risk at the time, but there is danger that the cardiac compensation will be found incomplete when the attack is past, for not a few cases of heart disease date the final cardiac failure from an acute illness.

Among the forms of morbus cordis, adherent pericardium seems to be the most serious, and, of the valvular affections, mitral disease.

Out of the 100 fatal cases the mitral was the valve affected in 5; three times alone, once with aortic disease, and once with aortic disease and pericarditis. In two others the aortic valves alone were involved.

In any given case the prognosis will depend upon the extent to which compensation is complete; those in which it is defective generally die when attacked by pneumonia, but in others, in which it is adequate, the pneumonia runs its ordinary course and often ends in recovery. The existence of old lesions does not seem to increase the chances of recent vegetations being formed.

In 21 cases of valvular disease, Sturges found recent vegetations without old heart mischief in 6, and with old heart mischief in 4. In my own cases 1 only had recent vegetations and 7 chronic lesions. In the 100 fatal cases 25 in all had some affection of the heart, old or recent.

Kidney Disease.—Pneumonia is very rare in the course of acute nephritis. On the other hand chronic lesions of the kidney are very frequently found in fatal cases, and although there is no conclusive evidence to show that chronic kidney diseases increase the liability to pneumonia, there can be no doubt that they greatly increase the mortality.

Thus a man of 39 had double pneumonia, first at the left base and then at the right apex. He was at first very sleepless and then very delirious. He developed pericarditis on the 10th day and died. *Post-mortem*, besides the lesions of pneumonia and granular kidney, there were recent pericarditis and left pleurisy; the heart weighed 18 and the liver 84 ounces. The urine contained no albumen at all until the 4th day, and after that only a trace, and the presence of granular kidney was not suspected during life.

Sturges, out of 94 cases examined *post-mortem*, found the kidneys granular in 7 and large and white in 7 more, together giving a percentage of 15. In my 100 cases the kidneys were granular in 8 and large and white in 2, giving a percentage of 10.

The signs of granular kidney can be very easily overlooked in life, and the affection may be more frequent than is commonly supposed, but in patients of middle life it is a disease that should always be thought of, as it so seriously affects the prognosis.

Rheumatic Fever.—There is a strange difference of statement in books as to the frequency of pneumonia in the course of rheumatic fever. I should certainly agree with Grisolle, who says that it is one of the rarest of phenomena, and

this also appears to be Sturges' opinion. If it be rare for pneumonia to occur in the course of rheumatic fever, it is still rarer for rheumatic fever to develop in the course of pneumonia.

Some cases bearing on this question are recorded by Sturges, but the two affections are so common that it is easy to believe that they might occasionally be associated without any relation of cause and effect existing between them.

Specific Fevers.—Pneumonia is rare in any of the specific fevers.

Where the temperature is already high from the fever, it is difficult to diagnose pneumonia from acute congestion and hypostatic congestion of the lungs, both of which are probably to a great extent the mechanical result of cardiac failure.

Inflammatory affections of the lung are not uncommon in all the specific fevers, but they are nearly always lobular rather than lobar. This distinction is not sufficiently made in most of the statistics.

When lobar pneumonia does occur it is usually fatal. According to Murchison, though rare in typhoid it is still rarer in relapsing fever, and rarest of all in typhus.

In *Scarlet Fever, Measles, and Diphtheria* the inflammation is almost invariably of the lobular form, and true pneumonia is very rare. Sturges quotes, with reference to diphtheria, some statistics from the Children's Hospital which show that lobar consolidation was found in 14 out of 100 cases which died without tracheotomy, and in 13 out of 60 which died after tracheotomy, and in some of the latter the complication did not arise until two or even three weeks after the operation. In all these cases there is the ever-present difficulty of deciding whether the consolidation was not a rather extensive form of the lobular inflammation due to direct infection from the wound.

In *Typhoid Fever* pneumonia has always excited interest on account of the theory that the typhoid germ can localise itself in the lung and there excite lobar pneumonia. Whether this be so or not in typhoid as in other fevers future bacteriological investigations only can determine.

So far they seem to show that the infection is usually heterologous, *i.e.*, not due to the typhoid bacillus, but to the pneumococcus or some other germ.

Certain it is that in some cases of typhoid it is by no means easy clinically to diagnose pneumonia, for the bronchitis which is so frequent an accompaniment of typhoid may set in very early and produce well-marked lung symptoms, so that when associated with the high temperature of the fever, the diagnosis of pneumonia is very likely to be made; but the correct diagnosis is usually soon determined by the course of the case. Undoubted cases of true pneumonia occur in typhoid, but I believe with Murchison that they are rare.

In *influenza* pneumonia assumes greater importance, for though not a frequent complication as reckoned by percentage, it becomes unusually prevalent owing to the wide diffusion of the epidemic.

The organisms found in influenza pneumonia differ according to the period at which it occurs. When a sequel of influenza, the pneumonia is of pneumococcal origin, but when it occurs during the attack of influenza the organism found is the influenza bacillus (Klein).

Its frequency varies greatly in different epidemics, and perhaps also its mortality, for Peacock, in his account of the epidemic of 1848, states that pneumonia was very common but less than ordinarily fatal. Certain it is that during the recent epidemic pneumonia was responsible for a very considerable number of the deaths.

The dominant feature of the pneumonia has been its asthenic type, and the rapidity with which it has often proved fatal. The mortality has been much

above the ordinary average for pneumonia, and where recovery has taken place convalescence has been tardy.

As a complication it has developed as a rule about a week or ten days after the original attack of influenza, and usually as the result of exposure to cold or chill; but besides this there has been an undue general prevalence of pneumonia, as though the existence of the epidemic predisposed to pneumonia, even in persons who did not actually acquire influenza, or perhaps had it in so slight a form that it was not recognised.

One other marked peculiarity must be mentioned, viz., the extraordinary want of relation between the clinical symptoms and the amount of the lesion in the lungs. It has not been uncommon with the most typical clinical symptoms of pneumonia for the physical signs to be very difficult to find, or absent altogether, and on the *post-mortem*, a small patch of consolidation to be found not larger than a Tangerine orange; in other cases the lungs have been greatly congested, almost œdematus, and, in part, especially at the base, collapsed, but without, in any part, the characteristic consolidation of pneumonia.

In some instances the pneumonia has been of the wandering type, creeping about from part to part, and thus prolonging over three weeks or more the duration of fever.

I saw a case in which the attack commenced in the right base and gradually spread up to the apex, by which time the base had begun to clear up. Then the opposite base was attacked, and by the time this was solid, the left base was clear—but the patient's strength did not hold out any longer; she died, however, of asthenia and not of suffocation.

Some of the cases have exhibited an unusual amount of hæmorrhage, and where the pneumonia has crept about and been associated with recurrent hæmoptysis, the suspicion of phthisis has naturally been excited.

A previously healthy young man of 30 years of age, after an ordinary attack of influenza, did not regain health, but lost flesh and strength, spat a small amount of blood on two or three occasions, and had a temperature constantly above normal, reaching in the evening 102° or 103° . This had continued for three weeks when I saw him. The physical signs were indefinite. I could not satisfy myself that he was phthisical, and sent him away for two months, at the end of which time he returned perfectly well, having gained flesh rapidly, and being on his return much above his normal weight. He has continued in perfect health and in active work, and has developed no further signs or symptoms.

This class of case was not uncommon in past epidemics, but has been singularly rare in the present. I have only seen one other instance of it, and that was not quite so clear, as it occurred in a young man who was at the time suffering from active syphilis, and it was possible that the affection of the lung was syphilitic in nature. He also came to me with the diagnosis of acute phthisis, but he completely and rapidly recovered, and has continued perfectly well.

Malaria.—The terms "intermittent" and "remittent" as applied to pneumonia refer usually to the character of the fever, and indicate that the inflammation recurred after an interval of complete or partial defervescence, and thus an intermitting or remitting temperature becomes the characteristic of all the pneumonias of the wandering type. But the terms have also been applied to cases which are supposed to stand in a closer relation to malaria, and it would be less confusing, if for such cases the terms "malarial" or "paludal" were employed.

Pneumonia may of course develop in the course of an ague attack, just as it may in the course of any other fever. Again, in persons who have had ague previously or are in an ague country, an attack of pneumonia, as of any other fever, may determine an attack of ague.

Besides these cases, in which the pneumonia is presumably of the ordinary type, and merely accidentally associated with ague, there remains a group in which the pneumonia is supposed to stand in a more intimate relation to ague, and to be actually due to the malarial poison. Of these cases there are two

classes: *in the one*, it so happens that in districts where ague is rife at certain times of the year, the annual outbreak may fail and its place be taken by a similar outbreak of pneumonia; an instance of this is quoted by Hadji-Costa¹ in Thessaly: *in the second*, each ague fit is associated with pulmonary symptoms and physical signs. In both instances it is stated that the pneumonia is of short duration and not grave, that the consolidation appears at once, and that in the intervals, when the temperature falls considerably if not quite to the normal, the consolidation resolves somewhat but extends again with the recurrence of fever. These attacks tend, it is said, to subside of themselves after three or four recurrences, but yield readily if treated with full doses of quinine.

Four cases of pneumonia of this kind are recorded by Koranyi,² and the two charts which he gives there speak for themselves.³

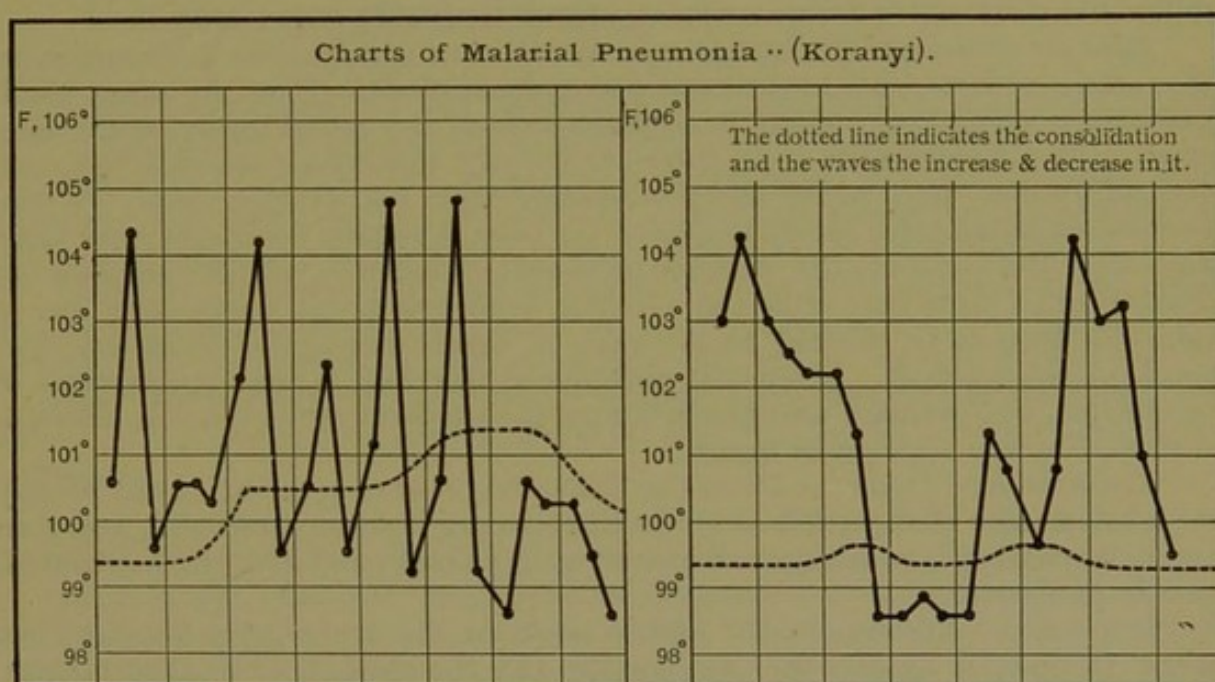


Fig. 74.

Alcoholism does not predispose to pneumonia, but more than doubles its mortality. Huss stated that over 50 per cent. of alcoholics attacked with pneumonia died, and I think this is not above the mark, but it is, of course, impossible to state this numerically on account of the difficulty in agreeing upon what exactly constitutes alcoholism in a pathological sense.

Doubleday, *New York Medical Record*, 1885, March 28, gives the following figures:—

	Cured.	Died.	Per cent. of Fatal Cases.
No excess,	21	7	=25
Slight,	32	16	=33
Marked,	12	27	} =68.4
Very great,	12	25	

The Collective Investigation statistics show the same facts, although the general percentages are lower throughout.

Total abstainers,	11.2
Temperate,	18.4
Intemperate,	40.5

¹ *Rev. de Méd.*, Nov. 10, 1891.

² *Real. Encycl.*, viii. 384.

³ Cf. also Tham, *Virch. Jahrb.*, 1886, ii. 120.

Pneumonia tends in drinkers to assume the nervous asthenic form, delirium sets in early, and the patient passes rapidly into a typhoid state, and often dies after a very short illness of only three or four days' duration.

The delirium of pneumonia is of two types; the one, sthenic, active, busy, with wild hallucinations as in ordinary delirium tremens; the other, asthenic, low and muttering as in the asthenic forms of other fevers.

In the pneumonia of alcoholics the delirium is at first of the sthenic kind, and it is remarkable how great exertion such patients are for a time capable of in spite of the dyspnoea they suffer from. Soon the want of sleep and constant restlessness produce extreme exhaustion, and if the active delirium last more than a day or two, it passes into the second or asthenic forms and quickly ends in death.

The prognosis of the first form is doubtful; but the second form, whether it follow the more active delirium or not, almost invariably ends fatally.

Injury.—Considerable damage may be done to the lung, as in contusion or laceration, without any inflammatory symptoms resulting; on the other hand, pneumonia may follow a shake or accident without any actual injury to the lung at all. In all cases alike it is, however, ordinary pneumonia which occurs, and the injury has been rather the predisposing than the exciting cause. Where pneumonia occurs after actual lesions of the lung, it usually develops two or three days after the accident, but whether preceded by lesion or not, it often does not develop until some time after the accident, even five or six weeks later, and is probably then to be referred to the want of vigour due to the confinement and shock of the accident. Traumatic pneumonia, therefore, is not a special form of pneumonia, but ordinary pneumonia occurring in a person whose health has been shattered by the accident or injury.

Embolism and Infarct.—These conditions may lead to pneumonia, but usually no inflammatory changes follow unless there be septic infection as well, and then the inflammation is of a septic character, as in pyæmia, and may end in suppuration or gangrene.

Pregnancy.—Pneumonia but rarely arises in the course of pregnancy, but when it does, it usually causes abortion and the child is lost. The mother, however, frequently escapes.

After parturition inflammation of the lung is usually septic, and not of the nature of true pneumonia.

Nervous Diseases.—Acute and often destructive inflammations of the lung are not uncommon causes of death in nerve diseases, especially in those which are attended with loss of consciousness or with bulbar symptoms. There is no doubt that in the majority of these cases the inflammation is due to the passage into the lung of foreign substances, chiefly particles of food, as the result of the loss of sensation, or of muscular power, in the parts around the glottis.

Schiff's¹ observations that inflammation of the lungs follows section of the trunk of the vagi are often quoted in support of the view that such inflammations may be produced by trophic influences. These observations have been called in question, and it has been shown that such lesions are the result of the passage of foreign substances into the lung owing to the paralysis of the glottis-closers by the division of the vagi.

FORMS OF PNEUMONIA.—What are often called forms of pneumonia do not deserve to be raised to the dignity of special varieties of the disease, for in many instances nothing more is meant than that certain symptoms predominate or that certain opinions are held as to the cause, etc.

¹ Gärtner, *Allg. Wien Med. Ztg.*, 1885, Nos. 4 and 5.

The following forms are often spoken of:—

Primary Idiopathic, i.e., pneumonia which develops of itself without recognisable cause; and *Secondary* or *Consecutive*, i.e., pneumonia which is the apparent result of some other cause. Many of the secondary forms are not really pneumonia at all, but cases of inflammation of a different kind excited, e.g., by septic embolism, foreign bodies, or such-like causes.

Sthenic and *Asthenic*.—This, as in other cases, is a bad classification, for *sthenic* refers to the fever which is strong, and *asthenic* to the patient who is weak. So that *asthenic* pneumonia means only that the pneumonia is attended with prostration.

Cerebral or *Gastric* indicate the predominance of such symptoms in the case, and *bilious* points to the association of jaundice.

Typhoid pneumonia is a term of doubtful meaning, sometimes implying that the patient passed into the typhoid state, at others that it occurred in connection with typhoid fever, or was due to the same poison.

In other cases the supposed cause is taken as the basis of the classification, e.g., *rheumatic* and *gouty*, *pythogenic*, *puerperal*, *embolic*, *metastatic*, etc.

There is no evidence that *rheumatic* or *gouty* pneumonia has anything to do with gout or rheumatism. It is simply pneumonia occurring in a *gouty* or *rheumatic* patient, or occurring in patients subject to those affections.

Short, *long*, *abortive* refer to the duration; *apex*, *base*, *central*, to the seat; *massive*, to the character and extent; *drinker's* pneumonia, to the habits of the patient; *relapsing*, *remittent*, or *intermittent* and *wandering*, to the course of the disease. Most of these so-called varieties have been already referred to in other places. But two or three have sufficient clinical importance to be a little further considered.

Apex pneumonia is usually attended with more severe symptoms, with higher temperature, and greater delirium; and is more common in children.

Louis taught that apex pneumonia in the adult was evidence of pre-existing tuberculosis, and this opinion has been held by many authorities since, to which has been added the statement that pneumonia at the apex is more likely to resolve incompletely and leave some chronic lesion behind than pneumonia at the base. I do not think this can be proved by figures, and even if it be the general rule, there are, at any rate, very many exceptions to it.

Wandering pneumonia.—Every pneumonia spreads to some extent, for commencing in one spot it rapidly extends until the limits of the lobe are reached, but here it as a rule stops. In some cases, however, the pneumonia still slowly creeps on, until, it may be, the whole lung is involved. This has been called *pneumonia Errans*. At other times the pneumonia does not spread in direct continuity, but starts afresh and independently in some distant part of the same lung or on the opposite side, and this is distinguished as *pneumonia Migrans*. In either case the result is to prolong the attack considerably and also to increase the risk of it.

Remittent, *intermittent*, and *relapsing* forms are also described. Most creeping pneumonias are remittent, i.e., although the temperature may drop, it does not fall completely to the normal. The difference between intermittent and relapsing pneumonia is rather one of terms, for in each the temperature is supposed to have fallen to and remained at the normal level for some days.

Relapses in the strict sense of the term are rare. Wagner¹ met with 2 cases only in 1100. They must occur as in typhoid, after a distinct afebrile period, and

¹ D. Arch. f. kl. Med., xlii. 410.

during convalescence. They are to be distinguished from *recurrent*, i.e., second or third attacks. French writers speak of *pneumonie à recidive*, i.e., subsequent attacks, and *pneumonie à rechute*, i.e., relapse. As already stated, subsequent attacks of pneumonia are by no means rare; indeed, pneumonia is prone to recur, but relapses are uncommon. I am inclined to think that they are most frequent where pneumonia has developed in the course of bronchitis. According to Germain See¹ they occur usually about the fifteenth or sixteenth day of illness, i.e., at the end of the week of convalescence. They belong to the category of short or abortive pneumonias and are of good prognosis. Their access is often marked by no rigor, but by rise of temperature only. The difficulty in these cases is, of course, to be sure that the fresh access of fever is not due to some complication, but the diagnosis is, as a rule, determined by the appearance of the ordinary physical signs of consolidation.

The pythogenic or septic pneumonias much resemble typhoid fever in general symptoms, and may be confused with it. The hepatisation is often patchy or ill-developed, and the physical signs, therefore, often indefinite; in these respects agreeing with the pneumonia which is so frequent and fatal a complication of influenza.

Perhaps the three most important varieties of pneumonia are the following:—*Pneumonia in drinkers, in the aged, and in children.*

The *Pneumonia of drinkers* has been already dealt with.

In the aged, pneumonia tends to assume the asthenic form, with low wandering delirium, and no very marked febrile symptoms, so that if the chest be not carefully examined the nature of the affection might be overlooked. Pneumonia in the aged is very fatal, and death is usually brought about by cardiac failure.

In children, pneumonia is characterised by a stormy access, high fever, extreme rapidity of pulse and respiration, and the predominance of nervous symptoms. The access is frequently marked by fits instead of rigors, the temperature runs up rapidly to a considerable height (105° or 106°), the child becomes very delirious, unable to recognise any one, and rapidly becomes drowsy or half unconscious. Vomiting is frequent, and often severe and persistent.

The symptoms seem to point to some acute cerebral affection, and meningitis is often suspected, but meningitis rarely has so acute an onset, and the altered pulse-respiration ratio suggests from the first the correct diagnosis. The altered pulse-respiration-ratio is of the greater importance from the fact that the physical signs are often absent or indefinite for some time, and may not become conclusive for two or three days. Usually when the physical signs are fully developed the nervous symptoms become less extreme.

It is not uncommon, as in other acute fevers in children, to get an erythematous rash which may lead to the suspicion of scarlet fever, but the suspicion is, as a rule, quickly set at rest, for the erythema is not punctuate nor persistent.

The fever ends as usual with a crisis, and convalescence is rapid. The prognosis is good, and the mortality between 2 and 3 per cent. only.

Apex pneumonia is commoner in children than it is in adults, but in both is attended with more severe general symptoms than pneumonia of other parts.

In very young children the diagnosis from broncho-pneumonia is often very difficult, and what has been thought to be croupous pneumonia during life turns out *post-mortem* to be catarrhal (cf. Broncho-pneumonia).

¹ *Maladies spécif. non-tuberculeuses des poumons*, 1885, p. 207.

DURATION OF PNEUMONIA.—Pneumonia ends by crisis in two-thirds of the cases and by lysis in the remaining third, but in the majority of cases the difference between crisis and lysis is not more than from twelve to twenty-four hours. The diagram, constructed to show the critical days, may be used also to show approximately the total duration of the affection.

It is there shown that the pneumonia has run its course in 73 per cent. of the cases by the end of the 7th day, and in 95 per cent. by the end of the 10th. These figures refer, of course, only to those cases which did not end fatally. It is also shown that 56 per cent. of all cases ended on one of three days, viz., the 5th, 6th, or 7th.

Cases of shorter duration than this require to be more specially considered. The fever ended in 9 per cent. on the 4th day, in 5.6 per cent. on the 3rd day, and in 1 case only out of 250 on the 2nd. Death may certainly occur within a very few hours. Jürgensen records two cases, fatal in eight and in thirty-four hours respectively, and Wilson Fox another, fatal in twelve hours.

Instances of *one-day pneumonia* have been recorded, but it is obvious that the difficulties of diagnosis are very great. Jürgensen¹ stated that he only knew of one case, viz., that recorded by Leube. Since then two cases have been published, one by Moellmann,² and one by Weil.³ In the last the temperature ran up suddenly to 104°, but became normal after twenty-four hours, then rusty sputum made its appearance, and definite physical signs developed.

Cases of long duration are recorded,⁴ and I have notes of one in which it was thirty-eight days before the temperature became normal. I could not find any cause for this prolonged fever, but it was impossible, as it is in most of these cases, to exclude some complication which would yield no definite physical signs, as, for example, a deep-seated empyema. It is certainly the general rule that when pneumonia is protracted beyond the fortnight some complication is present, unless a relapse have occurred or the pneumonia be of that wandering kind, which spreads from one part of the lung to another.

PROGNOSIS OF PNEUMONIA.—In discussing the symptoms, complications, and associated diseases, many important points in prognosis have been incidentally mentioned. In this section it will be necessary only to deal with prognosis in a general way.

In the prognosis of pneumonia three separate questions are involved—(1) the risk to life during the attack, (2) the prospect of complete recovery, and (3) the effect of the attack upon the duration of life.

Pneumonia at any age is serious and threatening; but in the healthy, temperate, and young the prognosis, as a rule, is good, however severe the attack may at the time appear to be. As soon as the fever is past, convalescence is rapid, and recovery complete; while, except for the possibility of another attack, which, however, is not great, the prospects of life and health are unaffected. The prognosis in pneumonia is therefore chiefly concerned with the risk to life during the attack, but, however useful general statements may be, each individual case must be considered upon its own merits.

The three most important points in the prognosis are the age; the previous health and habits; and the occurrence of complications.

The mortality tables show, after five years of age, a steadily increasing risk as life advances.

¹ *Loc. cit.*

² *Ibid.*, 1879, p. 666.

³ *Berl. kl. Woch.*, 1889, Nos. 16, 18, 20.

⁴ *Cf. Chronic Pneumonia.*

The prognosis is rendered more grave by any previous ill-health, whether the result of disease, or of debilitating or vicious habits; among the latter drink is the most important.

It is further aggravated by the existence of any complication, of which the most serious are old or recent affections of the heart or lung; or by the presence of some acute disease (like a fever), of which the pneumonia has been itself a complication.

Besides these general considerations unfavourable symptoms may develop and give the danger signal. Of these the most important are the following:—

1. *High fever.*—High temperature is characteristic of pneumonia, and, strange to say, the maximum of safety seems to be with the temperature ranging about 104° , the danger increasing not only when the temperature rises above this, but also when the average lies much below it, for many cases of severe pneumonia have a temperature which does not rise above 101° or 102° at any time.

Persistent high temperature at a level of 105° or 106° with but slight daily remissions makes the case, of itself, a grave one; while actual hyperpyrexia, especially if it occur about the time when the crisis is expected, is almost invariably fatal.

If, at the crisis, the temperature fall but no general improvement take place, the prospects of recovery are very small.

2. *Rapidity of pulse and respiration.*—A rapid pulse is of worse augury than a high temperature; rapidity of respiration worse than either.

In the adult, a pulse above 120 or a respiration rate above 50 is grave, and with a pulse above 130 or respirations above 60 few recover. In children, however, the pulse may reach 160 or more, and the respirations 60 or even 80, and yet the cases do well.

3. *Delirium.*—If in the adult this be due to previous alcoholism, it usually foreshadows a fatal result, but, when due to asthenia, the prognosis is given by the asthenia rather than by the delirium. Wild or active delirium is always of grave omen on account of the exhaustion it produces, if for no other reason. In children delirium is often extreme, but does not affect the prognosis.

4. *General muscular tremors* always show grave nervous prostration, and, if not due to alcoholism, they indicate the approach or existence of dangerous asthenia. These cases usually pass rapidly into the typhoid state and die.

5. *The typhoid state.*—This, though of all conditions the most grave, is not necessarily fatal, for even apparently desperate cases sometimes struggle through.

6. *Changes in the sputum.*—Prognosis is bad, if the expectoration contain much blood, whether bright or dark red (prune-juice); if it develop a gangrenous odour; if it become copious and bronchitic in character; or if the patient suddenly cease to cough any up.

Death is usually ushered in with the signs of gradual failure of the heart. The pulse grows weaker and weaker, becomes rapid, irregular, and intermitting; the respirations shallow; the cough ceases or is ineffectual; the body becomes bathed in a clammy sweat; the extremities grow cold; the cyanosis increases, and is replaced at the last by an ashy pallor, which is especially marked in children; the secretion is no more coughed up and rapidly accumulates in the tubes; both pulse and respiration grow slower and slower, and finally cease.

This stage takes some hours to run its course, and the patients die with the signs of gradually increasing suffocation. Sudden death is rare, and is then due to cardiac syncope or to clot on the heart or pulmonary vessels.

Death happens most frequently at the end of the first or beginning of the second week, *i.e.*, between the 4th and the 11th day. Death on or before the 4th day is, however, rare, while after the 11th it is by no means uncommon.

Fatal cases have been recorded even within a few hours of the initial rigor—8 hours (Jürgensen), 24 hours (Mendelssohn), and 34 hours (Jürgensen).

Even in convalescence all risk is not past, especially in the aged, for patients sometimes seem as if they had strength enough to get over the attack but not strength enough to get well; while others die suddenly, sometimes of cardiac syncope, but more often of pulmonary embolism from the detachment of a clot which has formed during the illness, in the right side of the heart. This is the most distressing of all events, for death then comes as a sudden surprise when all seemed well.

Mortality.—The general mortality is stated by Huss to vary from 1·5 to 2·3 per 1000 persons living.

The mortality or fatality of pneumonia averages, on the whole, about 20 per cent. of those attacked, but it varies much in different places, and, in the same place, at different times, as well as in relation with the age, habits, amount of consolidation, and complications.

At St. Bartholomew's Hospital the mortality of 925 cases for nine years was 22·7 per cent., and at St. Thomas', of 708 cases, 20·2 per cent.

The variation in different places is illustrated by the following figures given by Jürgensen:—Vienna, 24·5; Bale, 23·1 and 25·7; Tübingen, 12·1; Kiel, 15·4; Stockholm, 10·7.

The rate also varies greatly in the same place in different years.

Thus Bamberger found as the average of three consecutive years a rate of 11·2, and the next year it rose to 18·15. Brander of Copenhagen found in one year a rate of 5·4, and on the next of 31. Such extreme variations are, of course, very rare. The mortality for seventeen consecutive years at St. Bartholomew's Hospital is given in the following table:—

1875 . . . 26·0	1881 . . . 14·5	1887 . . . 22·0
1876 . . . 36·8	1882 . . . 20·4	1888 . . . 20·0
1877 . . . 30·75	1883 . . . 29·2	1889 . . . 12·3
1878 . . . 18·4	1884 . . . 22·6	1890 . . . 12·5
1879 . . . 18·5	1885 . . . 21·0	1891 . . . 13·3
1880 . . . 15·6	1886 . . . 20·0	

Sex.—The St. Bartholomew's statistics show that of the fatal cases, 71·3 per cent. were men and 28·7 per cent. women. It is commonly stated that though the proportion of men to women attacked is nearly 3 to 1, the relative mortality is higher in women.

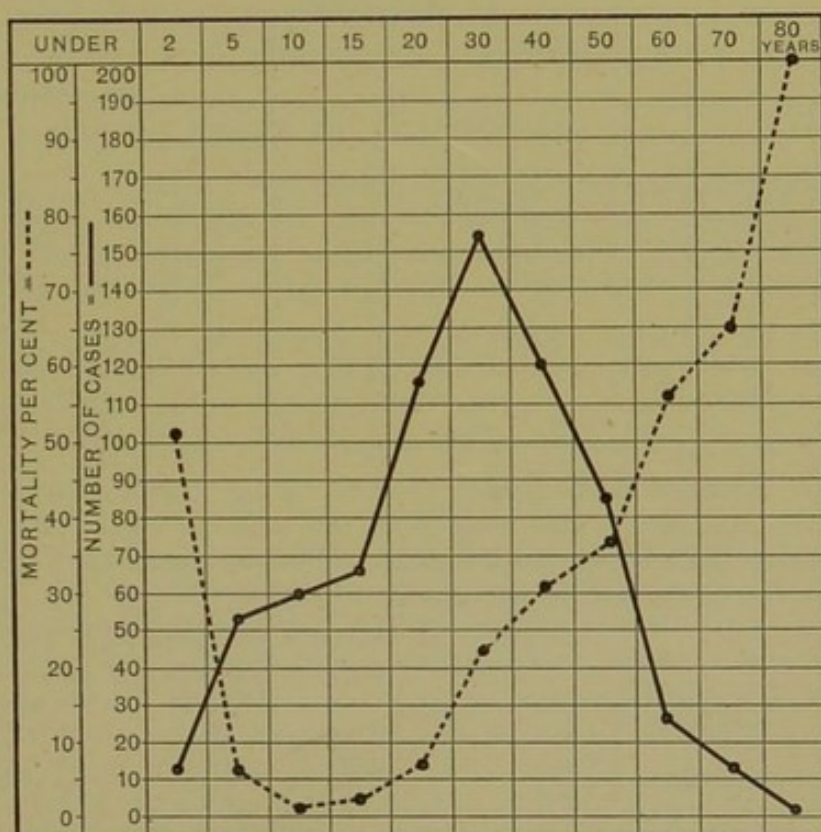


Fig. 75.

Diagram, showing the relative mortality of pneumonia at the different age-periods. Number of cases, dark line; percentage mortality, dotted line (Hadden, Mackenzie, and Ord, *St. Thos. Hosp. Rep.*).

This is not supported by my own statistics, which show a percentage of 23·5 for men and 22·0 for women, and with these the St. Thomas' statistics agree, viz., 21·4 ($\frac{11}{12}$) for men and 16·3 ($\frac{27}{16}$) for women, as well as those of Coupland and Sturges, 19·4 for men and 16·7 for women. On the other hand, Huss gives the mortality for men as 14·2 and for women as 19·9, and the Collective Investigation Records state it as in the proportion of 3 women to 2 men.

Age.—Age affects greatly the death-rate, for pneumonia is rarely fatal in children above five years of age, but from adult life onwards the fatality rises rapidly and steadily with every year.

Mortality in relation to number of persons attacked.						
	Huss.	Coupland and Sturges.	St. Bartholomew's Hospital.			
			Period.	Males.	Females.	Combined.
Up to 10 years	...	5·0	5-15	2·5	2·0	2·25
10-20	6·0	5·4	15-20	10·0	3·3	6·6
20-30	6·0	12·6	20-30	20·0	11·3	15·6
30-40	12·0	25·8	30-40	44·0	22·0	33·0
40-50	20·0	39	40-50	50·0	25·0	37·5
50-60	21·0	37·7	50-60	63·0	45·0	54·0
60-70	...	35·3	Over 60	60·0	50·0	55·0
Above 70	...	57·7				

Extent and Seat.—Other things being equal, the mortality varies with the amount of the lung involved, and, as would be expected, it is at its highest where both lungs are attacked. Pneumonia is also, speaking generally, less fatal at the base than at the apex.

Base 14·5. Apex 21·8. Both lungs 36. (Huss.)

		Males.	Females.	Persons.	
Left lung	{ Base, . . .	14·2	11·6	12·9	} 19·0 } St. Bartholomew's Hospital.
	{ Apex, . . .	13·0	37·5	25·2	
Right lung	{ Base, . . .	20·8	11·9	16·4	
	{ Apex, . . .	23·2	13·6	18·4	

Complications of any kind greatly increase the mortality.

Huss states the death-rate of the uncomplicated cases at 5·79, and of the complicated at 19·29. Rychner's figures are somewhat higher, viz., 9·5 and 61·1.

It is impossible to state this numerically, for complications or previous disease are so often found *post-mortem* when not suspected during life. Thus, out of my 100 fatal cases some important complication was found in no less than 40.

The preceding facts rest, to a very great extent, upon hospital statistics, and it must be borne in mind that they may yield results higher than would be true for the general population, and especially for the well-to-do, considering that hospitals draw their cases largely from the poor or destitute, and from those whose health has often been ruined by want, vice, or drink.

DIAGNOSIS.—In most cases the diagnosis is free from difficulty. There are the ordinary signs of acute fever, the localising symptoms pointing to the chest, and the physical signs of consolidation of the lungs, so that the diagnosis is clear, viz., acute, inflammatory consolidation of the lung, in other words pneumonia. Most of the difficulties in diagnosis have been referred to in previous sections. Here it will be necessary to do little more than sum up what has been already said.

Before the physical signs have made their appearance, or if they are indefinite, some other fever may be suspected, especially one of the common exanthemata in children, or even acute meningitis if there be much delirium; in adults influenza may be thought of, or typhoid fever, and in very bad cases the question even of typhus may arise. As a rule in all these cases the pulse-respiration ratio will determine the diagnosis at once, for in none of the affections mentioned does it alter in the way so characteristic of pneumonia.

If the onset be not so sudden as usual, the question of typhoid fever or of bronchitis may present itself.

The physical signs, again, may be indefinite or misleading; for example, when the tubes are plugged, and the diagnosis from pleuritic effusion will be difficult; but in this, as in other cases, the course of the disease will soon clear away all doubt.

If the patient be in the typhoid state, other affections in which that condition is likely to arise will suggest themselves, *e.g.*, typhus, typhoid, or some other specific or septic fever in a grave form.

The character of the delirium may suggest delirium tremens.

Acute alcoholic delirium develops in two ways. In the one the patient has been constantly drunk, and has in fact drunk himself into the delirium. In the other, the patient has been a constant soaker, never perhaps actually drunk, yet rarely quite sober, and would not have developed the delirium except for an accident, such as a fracture or some acute illness, for instance, pneumonia.

The symptoms are the same in either case, *viz.*, those of delirium tremens, but the prognosis differs widely. In the former case a few days' simple treatment will suffice for cure, but in the latter the patient will have a hard struggle for life, and will probably die.

It would be well if the two forms were always described by different names, and the former called delirium tremens, the latter traumatic delirium. It is to the second form that the acute alcoholic delirium of pneumonia belongs.

Delirium in alcoholics may set in so suddenly, coincident as it were with the onset of the pneumonia, as to lead to the erroneous diagnosis of simple delirium tremens and the overlooking of the pneumonia. The error could easily be avoided by taking the temperature, for in delirium tremens the temperature is rarely raised much, if at all, and it may be laid down as a general rule, that if, in a case which seems to be delirium tremens, the temperature is high, the delirium is due to some cause other than drink alone.

Some cases of phthisis begin with, or are complicated by, acute attacks, which may resemble pneumonia; the fever, however, is longer continued, is of a more hectic type than is usual in pneumonia, and, instead of resolution, signs of excavation follow.

Broncho-pneumonia in children is often puzzling. In some instances it begins with symptoms as acute as those of croupous pneumonia, which indeed I believe these cases to be. In the other group, *viz.*, that which follows bronchitis, the fever is more irregular and longer continued, and relapses are frequent.

The acute inflammations of the lung which may arise in the course of pyæmia or other septic affections are clinically pneumonia, although their cause, course, and consequences are different.

It is asserted (Besançon and Griffon) that the serum of pneumonia has agglutinative properties, and that this is of diagnostic value.

In all cases alike, whatever difficulties there may have been at first are removed as the case progresses, and the diagnosis is made clear by the course the disease takes, so that, as a matter of fact, there are but few cases in which the diagnosis is doubtful for more than a short time.

TREATMENT.—Pneumonia being a germ disease, the methods of treatment are preventive, antitoxic, and symptomatic.

1. Preventive.—We know so little at present about the conditions that determine an attack of pneumonia, that we can do little in the way of prevention.

2. Antitoxic, the object being to check the production by the germs of the poisonous substances upon which the symptoms depend, or to neutralise them if they are produced.

The *serum treatment* of pneumonia has not, so far, proved of practical use, and there are theoretical reasons why it is not really so promising as at first sight it might seem (*cf.* Etiology).

It is doubtful whether we can by any means at present at our disposal cut the disease short, but it is equally certain that we can, by bad treatment, prolong its duration and increase its mortality.

3. Symptomatic.—The third method of treatment, and the only one open to us at the present time, is to watch the symptoms as they arise, and to so modify and control them as to prevent their exercising a pernicious effect.

The study of the natural history of pneumonia shows that it is a disease which in the healthy and young tends to get well of itself. This has led to the adoption of what is called the *expectant method* of treatment, as opposed to the more active and violent measures formerly in vogue. We now endeavour to assist nature to throw off the disease rather than lay heavy hands on what was thought to be the disease itself, lest by that means we do a fatal violence to the patient.

Although the general impression that pneumonia has become a much less grave disease under the expectant line of treatment is without doubt founded on fact, still it is impossible to prove this absolutely by figures, and still less are statistics competent to decide between rival treatments of a more special kind. Pneumonia differs so much at different times and in different places, and according to the age, habits, and health of the persons attacked, that it becomes impossible to get a sufficient series of cases which differ only in respect of the treatment adopted. Experience shows that the statistical method applied to the results of the treatment of pneumonia is as unsatisfactory as it is in the case of typhoid fever.

Symptomatic treatment implies that for a mild case of pneumonia in a young and healthy person, where no symptoms are in excess, but little special treatment is required, and that such treatment only becomes necessary where the symptoms are severe or out of the common.

In an ordinary case the patient should be kept entirely in bed, but he has rarely either inclination or power to leave it. The room should be kept cool and fresh, at a temperature not above 60° Fahr.

The diet should consist chiefly of milk and beef-tea, with which one or two raw eggs may be beaten up. It is important to remember that harm may be done by giving more milk than can be completely digested. The thirst causes a craving for fluid, and milk is often taken in quantities too large to be properly digested. It then lies in the stomach and undergoes fermentation, thus producing much discomfort, or even troublesome vomiting. It is best where the thirst is extreme either to dilute the milk with some effervescing water, or, if sufficient food be taken, to slake the thirst with water acidulated with lemon juice or with phosphoric acid. A prejudice seems to exist against giving water in fevers, for which there is no good reason.

Stimulants are, in mild cases, unnecessary. The usual indication for their administration is given by the pulse and heart.

A simple purge is generally required at the commencement, and nothing is better than a dose of castor oil or a small pill of calomel and colocynth.

If the skin be very hot and dry, a diaphoretic mixture will do good, and in children a hot bath often gives great relief. Where a diaphoretic is not indicated, an acid draught with some quinine or strychnia will help to clean the tongue and improve the appetite.

The pain in the side, if not relieved by counter-irritation or hot poultices, will be quickly removed by a few leeches.

As soon as the fever is past and appetite returns, solid food may be given and a tonic administered.

It is a good rule not to allow the patient to leave the bed until the temperature has been normal for at least ten days.

Special treatment will be indicated when any symptom is in excess.

Fever.—A temperature of 103° for the few days that a pneumonia lasts will do but little harm of itself, and need not be treated. There are two classes of cases in which the temperature will call for treatment; in the one it runs up suddenly to a considerable height, and in the other it continues at a high general level without much remission throughout the twenty-four hours.

Hyperpyrexia in the strict sense is even rarer in pneumonia than it is in rheumatic fever, but it is still more dangerous. It requires the immediate use of the cold bath if life is to be saved.

Cold baths have been used as a routine treatment even in ordinary cases with a temperature that cannot be called abnormally high. Patients have been plunged into water at almost a freezing temperature many times each day as long as the fever lasted, and that without the evil effects that might have been anticipated. Jürgensen records the case of a child of four years of age who had sixty baths in three days, and recovered; but in spite of such energetic bathing the temperature was not reduced below the mean of 105.3° . It is thus clear that the cold bath may be employed without any great risk when it is necessary, but its effect is transient. The bath, as ordinarily administered to an adult, is attended with a good deal of fatigue, and since the result desired can be arrived at in other ways, *e.g.*, by *sponging*, *packing*, or *cradling*, the cold bath is best reserved for exceptional cases. Cradling is the simplest in practice, and is certainly very efficacious. The patient is stripped, or, at any rate, covered only with a light night-dress; a cradle is put over him, extending from the chin to the feet, and over it is laid a single sheet. In this way the temperature may be considerably reduced and kept down.

When the skin is dry and pungently hot, wet packing is useful. Better still for children is a *hot bath* at a temperature of 105° to 108° , a few handfuls of mustard being mixed with the water. After being removed from the bath, the patient is laid, without being dried, between blankets; a powerful reaction soon sets in with profuse sweating, and continues for some hours, during which time the child, who has been restless before, often sleeps quietly and wakes up much refreshed. I have given such a bath to children daily in the evening, or even twice in the day, and always with the greatest benefit.

Antipyretic drugs have two disadvantages, the first that their action is so transient, and the second, that they are so likely when freely used to produce faintness or collapse. The routine use of antipyretics, as of cold bathing, rested upon the assumption that the temperature was the chief cause of danger. Now that this theory is shown to be one-sided, antipyretics, except for very special cases, are falling into the place to which unbiassed experience had already relegated them. Still they have their use in special cases, though, for

safety and efficacy even in these cases the cold bath is generally to be preferred.

Quinine, to produce any effect upon the temperature, must be given in large doses, *e.g.*, 30 grains either in a single dose or within a few hours; if in a single dose it may cause vomiting, and then, from the uncertainty as to the actual quantity retained, the dose cannot be repeated; it is better given in smaller doses at frequent intervals, say 5 grains in the form of pill or solution every hour or half hour; even so one of the doses is not infrequently vomited, but it is easier to estimate approximately the amount retained.

Salicylate of soda is also a very safe and good antipyretic, but it often causes sickness, or at any rate nausea. Salicylic acid has been entirely given up on account of the frequency with which it causes collapse, and Antipyrin for the same reason is now but little used in pneumonia. Kairin, Thallin, Resorcin, and the other antipyretics all labour more or less under the same objections, and are practically but little used.

Alcohol in full doses is also an antipyretic, but, being a stimulant, belongs to a different group. To reduce temperature it has to be given in very large amounts, and is especially suited to those cases in which there is marked asthenia.

Cardiac failure is the great risk in pneumonia, and the knowledge of this fact yields two prime indications for treatment, first to counteract it when it develops, but more important still, to avoid everything which tends to excite it or to aggravate it. It is for the last reason that the old depressant treatment by repeated bleedings, full doses of antimony, aconite, veratria, etc., has been abandoned, and for the same reason many of the antipyretic drugs have been given up.

The causes of cardiac failure are not the same in all cases. It may be the result of previous disease; or due to acute degeneration of the muscle consequent on long-continued high temperature, or of the action of some blood poison developed by the disease itself. Each of these causes must be treated as best they can by the use of cardiac tonics and diffusible stimulants.

One further cause remains which calls for treatment of a different kind. Owing to the obstruction to the circulation through the lungs, the right side of the heart is overworked and becomes over-distended with blood. This over-distension weakens its power and in the end paralyzes its action. The usual signs of an embarrassed right side are present, *viz.*, increased cardiac dulness to the right, epigastric pulsation, distension and forcible pulsation in the cervical veins, and a certain amount of cyanosis. Under these circumstances the heart may *suddenly* fail. Its action becomes irregular and intermittent, the dyspnoea greater, and the cyanosis grows rapidly deeper.

Bleeding.—If the over-distension be not quickly relieved, death will soon follow, and there is but one way of effecting this relief, and that is by bleeding. For this purpose blood must be withdrawn rapidly and in sufficient amount. One of the big veins at the bend of the elbow is usually selected, and 20 to 30 ounces drawn. As the blood flows the heart's action becomes less laboured and irregular, the colour improves, and the breathing grows less difficult. The urgent symptoms being relieved, the improvement continues even after the flow of blood is stopped, and in a short time the patient may be out of imminent danger. In suitable cases taken at the right time I have no doubt that a free bleeding has saved many lives. Unless the bleeding be excessive no harm is likely to arise at the time, for the risk, if any, is not then but afterwards. The use of bleeding is to meet a sudden emergency, and to stave off imminent death. The loss of blood, tending to leave the patient weaker, might be expected to retard or prolong convalescence, but in the cases in which I have bled I have never seen any harm either at the time or later. Still it is only right to say that the cases were very carefully selected, as they always should be. Bleeding is contra-indicated where there is much general weakness, and especially where the heart has been gradually failing throughout. It is therefore out of the

question in weakly, unhealthy, anæmic persons, in the very young or the aged. Sturdy children, however, above 3 or 4 years old, have frequently been bled with advantage.

The typical case for bleeding is that of a young, full-blooded, healthy, well-fed, perhaps over-fed, adult with florid complexion and well-developed muscles.

Exceptions may be made to all rules, and I have bled copiously and saved the life even in a case of double aortic disease when acute congestion of the lungs set in, consequent on sudden failure of the left ventricle. The patient, who was at the time to all appearance rapidly dying, lived for more than three months after.

Free bleeding such as is referred to cannot be safely repeated; indeed, I have not seen any case of pneumonia in which it was necessary to bleed more than once. The rule should be to bleed freely or not at all, and only under the urgent conditions described. If, then, such bleeding fail, a second will not succeed. That indiscriminate bleeding is harmful there can be no manner of doubt, and this is shown by the diminished mortality of pneumonia since the routine practice of bleeding and other depressing methods have been given up.

Although bleeding was a recognised treatment from the earliest times for fevers, it was not until recent times that repeated copious blood-letting became the routine treatment for pneumonia. It reached its extreme in Italy in the early part of this century, where it was the custom to remove ten or more pounds of blood in the course of the attack; two pounds of blood being taken in three bleedings on the first day, two pounds in two bleedings on the second day, and the rest on subsequent days. With this was combined large doses of tartar emetic, even to the extent of a grain an hour, *i.e.*, 24 grains in the day. All that can be said is that it remains a marvel that the mortality was not much higher than it was admitted to be.

Oxygen.—This is a suitable place to refer to the inhalation of oxygen, for under its administration not only does cyanosis lessen, but the pulse drops a few beats and becomes more steady.

With these effects it often has also a sedative action, the restlessness quieting down and the patient falling for a few minutes asleep. The use of oxygen should not be too long delayed, for little can be expected of it if its employment be postponed until the patient is almost moribund. If commenced early enough it is certainly useful, but I have not myself seen the almost miraculous effects sometimes attributed to it, which are usually referable to the accidental coincidence of its use with the natural crisis. It may be administered almost continuously if given in a slow stream, through a funnel, but a mouthpiece is hardly ever tolerated by any patient conscious enough to object.

Stimulants.—Stimulants are not required in the ordinary pneumonia of the healthy and young, but in the aged or weakly they will be necessary from the first. In persons of alcoholic habit, or where nerve symptoms or marked asthenia develop, stimulants must be given freely, and then often form our sheet-anchor, pneumonia being a short though intense fever and the chief danger lying in the continuance of the fever. If the patient can be kept alive till the acute stage be past he will probably recover, and for this purpose nothing is more useful than alcohol either in the form of spirits or wine. It is astonishing how large a quantity may be taken during high fever without symptoms of intoxication being produced. Under its use not only do the pulse and respiration improve, but the delirium often diminishes and the tongue cleans; if, however, as Graves taught, the tongue become drier and the delirium increase, the alcohol should be reduced or stopped and some other form of stimulant substituted for it. Brandy or whisky is sometimes very distasteful, and may even make the patient sick. When this happens rectified spirit may be substituted, and if given in milk will be hardly detected. It must be remembered that rectified spirit is twice as strong as good brandy or whisky, and therefore not more than half the amount of the former is necessary.

Alcohol in full doses is also an antipyretic, and it is thus especially suitable for those cases in which the asthenia is associated with high temperature.

Ether may be used where alcohol is unsuitable, or as an adjuvant to it. It is best given in small doses frequently repeated, but its action is more transient than that of alcohol, and it is often unpalatable.

To meet any sudden emergency such as collapse, *subcutaneous injections* of ether (15 to 20 minims) or *strychnine* (3 to 5 minims of the liquor) are useful.

Musk and *camphor* have also a reputation as stimulants in asthenic cases, and may be given in from 5 to 10 grain doses in the form of pill or emulsion.

Lastly, the special cardiac stimulants, *digitalis*, *strophanthus*, and *caffeine*, are of advantage to counteract any tendency to asthenia. *Digitalis* must be carefully watched, for it may produce the same effect that it does where the heart is fatty or degenerate, and the beats may suddenly drop, without any other evidence, from 120 or more to 50 or even 40, and this especially about the time of the crisis. If the pulse drop to 80, *digitalis* should be at once suspended. *Strophanthus* may be prescribed where *digitalis* proves unsuitable, but it is not so reliable.

Citrate of caffeine is a most useful drug, both during the fever and during resolution. It is, I think, most serviceable where resolution is delayed and accompanied with great asthenia. In such cases 5 grains may be given three or four times a day for many days in succession with great benefit. For subcutaneous injection the sodio-salicylate of caffeine is the most suitable salt on account of its solubility.

Caffeine,	20 grains.
Salicylate of soda,	17½ "
Distilled water,	1 drachm.

This contains 1 grain in 3 minims, and the dose is 1 to 6 minims (Martindale).

Pain.—The stitch in the side, which is so commonly present at first, tends to disappear after twenty-four hours or so, but it may continue longer, and then, by checking the movements, further embarrass the breathing, or, by preventing sleep, weaken and distress the patient.

In ordinary cases it is relieved by hot poultices or by a blister. A subcutaneous injection of morphine or heroine has been advocated, but the best and most reliable remedy is the application of two or three leeches over the seat of pain. This is, as a rule, sufficient to take away the pain at once, and it does not return.

Poultices are often objectionable on account of their weight, and then a spongiopiline jacket sprinkled with spirits of camphor or turpentine will answer all the purposes of counter-irritation.

The use of cold applications to the chest, instead of hot, has been highly recommended, in the belief that they reduce temperature, check pain, and control the inflammation. Cloths wrung out of ice-cold water and applied to the affected side have been often used, but they soon get warm and require to be frequently renewed. Better than this is the application of an icebag to the side, or the use of Leiter's tubes, through which a constant stream of cold water is flowing. The objection to both is that the condensation of moisture round and over them makes the clothes damp and uncomfortable. In my own experience, cold applications have not been so successful or agreeable to the patient as the ordinary poultice or counter-irritation. I do not find that they in any way control the inflammation, and the temperature and pain can be more satisfactorily dealt with in other ways.

Cough is rarely severe enough to call for treatment. It is essentially conservative and directed to remove the secretion, and in itself does not require to

be checked. If it be very painful, the pain is better relieved by leeches than by narcotics.

Expectorants are seldom required, and then only during the stage of resolution. They should be of the stimulant group, such as senega and ammonia.

Narcotics are, in most cases, unnecessary, and often actually harmful, for they diminish the sensibility of the respiratory tract, and thus check cough and expectoration. The symptoms which might suggest their use are cough, pain, delirium, and sleeplessness, but these are all better treated in other ways. Narcotics of any kind have to be used with great discrimination in pneumonia, if they are not to do more harm than good. If they are necessary, bromide of ammonia, with or without chloral, is less risky than opium, and it may be combined, if thought fit, with hyoscyamus and cannabis indica. But it may happen that, in spite of all the objections urged, recourse must be had, after all, to morphine to procure rest, and though I always give it with hesitation, I must confess I have never seen any harm come of it in the cases in which I have used it.

Delirium has several causes, *e.g.*, high temperature, some severe complication like pericarditis, previous alcoholic habits, or asthenia; these must be sought out and dealt with accordingly. For most of them stimulants are most suitable and do most good, but if sedatives are necessary, some of those last mentioned may be employed.

Sleeplessness, again, is due to many causes, chief among which are pain and high temperature, which may be treated in the usual way. In children there is no better sedative for the weary restlessness of fever than a hot bath, and in the adult wet packing may have the same result. If in the adult twenty-four hours be passed absolutely without sleep, something must be done to give rest, or dangerous exhaustion is likely to set in, and this is as true of pneumonia as of other high fevers. Sometimes a cold douche to the head will evoke sleep, and if the other sedatives fail, recourse must be then had, in spite of all risks, to morphine, but, as a matter of fact, we are but rarely driven to employ this drug. It should be remembered that nothing is so disturbing as the constant flitting in and out of anxious relatives, and that the most absolute quiet and freedom from excitement are essential.

As regards the digestive tract there are rarely any symptoms which call for treatment. *Loss of appetite* may be dealt with by acid, and strychnine. *Vomiting* usually ceases after the first few hours. If it continue, it may depend upon improper feeding. Some effervescing mixture and regulation of the diet will usually control it. *Diarrhœa* also rarely requires treatment. The regulation of the food and a dose of castor oil, or better still, a few grains of calomel, will often check it. It is, however, at times very obstinate, and resists all treatment, but even in these cases it rarely seems to do any serious harm. The complications and sequelæ must be dealt with, as they occur, in the usual ways, it being borne in mind in all cases that depressing treatment must be scrupulously avoided.

Convalescents should be placed upon a liberal diet, with some stimulant and a tonic, and should not be allowed to leave their bed too soon. When they are well enough, it is wise to send them away to the seaside or the country for change of air.

HISTORY.—Pneumonia, as we know it now, evidently existed in the most ancient times, but was confused with other acute affections of the thorax. An attempt was made to distinguish between them, but without success, by the use of the terms pleuritis and peripneumonia. From the descriptions given little more can be said than that peripneumonia was the name given to the most

severe of the acute thoracic affections, and pleuritis to the less severe, especially when accompanied with pain in the side.

About the time of Sydenham it came to be recognised that no real distinction could be made, and all the acute affections of the respiratory organs were grouped under the common head of "inflammation of the lungs," pleuritis and peripneumonia being regarded as differing only in degree and not in kind.

To Laennec we owe the means of diagnosing pneumonia as we understand it now, and there has been but little that is essential added to the description which he gave of the disease.

Laennec's observations were supplemented by those of Rokitansky, who described minutely the pathological changes in the affected parts of the lungs.

Since then the discussion has turned chiefly upon the nature of the disease; whether it be a specific disease, with local manifestations in the lung, or a local disease with general symptoms. Of recent years research has chiefly concerned itself with the bacteriology of pneumonia.

38. METASTATIC PNEUMONIA—SEPTIC, EMBOLIC, HÆMATOGENOUS PNEUMONIA.

In this form of pneumonia the inflammation is caused by irritating particles

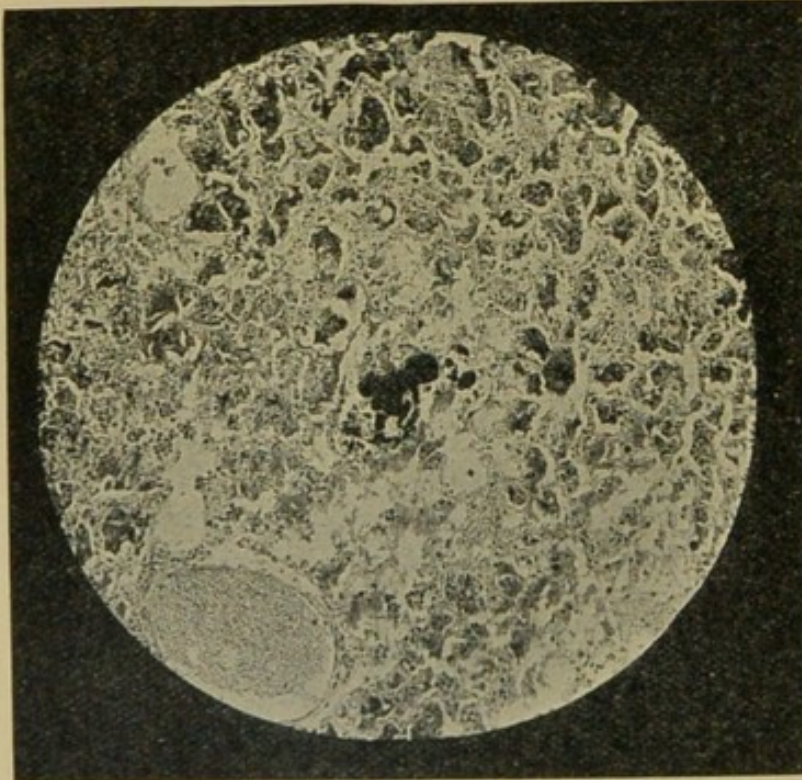


Fig. 76.

Plague pneumonia in a guinea-pig ($\times 85$) showing the inflammatory exudation in the alveoli. In the centre are several vessels (looking black in the specimen) filled with the plague bacilli. (From Dr. Klein's Collection.)

which have gained access to the lung by the blood-vessels. In many cases the branches of the pulmonary artery are found occluded by fragments (emboli) derived from clots formed elsewhere, as, for example, in the right side of the heart, in the femoral or some other vein. Simple embolism, however, does not of itself cause inflammation. This is excited not by the embolus, but by infective organisms brought with it, and it will vary in its form, *i.e.*, be simple, suppurative or gangrenous, according to the nature of the organisms present. Something depends also upon the size of the embolus; if it be big enough to completely occlude a large or medium vessel, an infarct

will be first produced, but if it be so small that it lodges only in one of the minute arteries, or in the capillaries, no infarct will form. In other cases there may be

no actual embolus, but the vessels be found stuffed with what is almost a pure cultivation of the infective organism grown *in situ*.

In any case the inflammation excited is of an intense character. The vesicles round are found filled with fibrinous and hæmorrhagic exudation similar to that of croupous pneumonia, but in strong contrast with that affection, the tissue of the lung is always involved, and the lymphatics, especially the perivascular, peribronchial and sub-pleural, are in a state of active inflammation and are filled with small cells.

The patches of consolidation produced may be numerous, discrete, and of small size, resembling those of broncho-pneumonia, or they may be of considerable size, owing to the confluence of many small ones or to previous infarction, so that the whole or the greater part of a lobe may be involved and the consolidation resemble that of croupous pneumonia.

In most cases sup-
puration occurs, the vesicles, as well as the interstitial tissue, become filled with pus-cells, and break down, and in this way an abscess is formed, which rapidly extends to the whole of the parts consolidated. The abscesses thus formed will vary, in number and size, according to the character of the antecedent consolidation, but they are very frequently numerous and of small size. Where an infarct has been first formed, the infarcted parts may necrose, become separated by a line of demarcation from the surrounding tissue, and when detached lie as sloughs in the centre of a pus-containing cavity of irregular size and shape with ragged walls. The contents of such cavities are very likely to become putrid or stinking, and the tissues around gangrenous.

The abscesses usually open into the bronchi, but they may perforate the pleura and produce pyo-pneumothorax. If the pleura be adherent, they may even burst through the diaphragm into the abdomen or externally through the chest walls.

If life be preserved, and there be a free rent for the pus, the abscess cavity may contract, and, in the course of time, if it be very small, may, it is said, be completely obliterated. Such results, though possible, are very rare, for metastatic pneumonia is generally fatal within a short time of its development, and frequently in the early stages of consolidation, before abscesses have

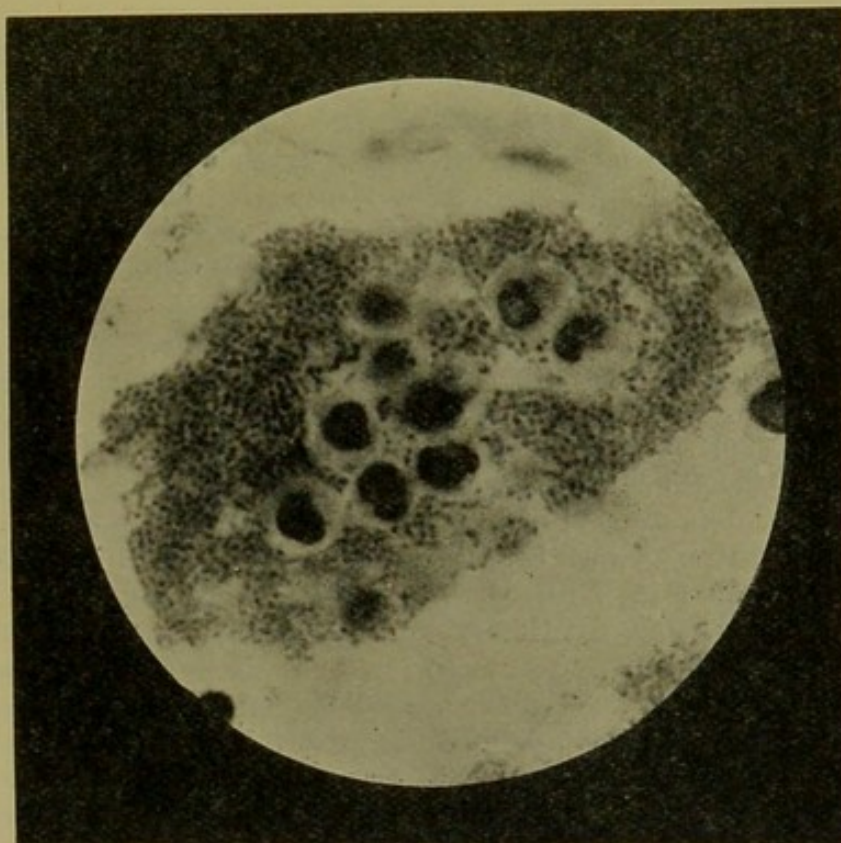


Fig. 77.

Section of the same specimen ($\times 1000$) showing the masses of plague bacilli in the exudation in an alveolus. (From Dr. Klein's Collection.)

had time to form. Metastatic pneumonia is not unlikely to pass into gangrene, which may become diffuse and rapidly involve extensive tracts of the lung. Such cases are, almost without exception, rapidly fatal.

The **symptoms** differ much in different cases, being at first those of acute inflammation of the lung and subsequently those of abscess or gangrene, as the case may be.

The **physical signs** also vary in a similar way, and, where the patches are small, may be too indefinite to base a diagnosis upon. Both symptoms and physical signs pointing to the chest are often so slight as to be easily overlooked, and this is especially likely to occur when the primary disease is one which is associated with a rise of temperature. It often happens in such cases that in the lung, as in the pleura and peritoneum, extensive inflammatory lesions are found *post-mortem* of which there has been no direct evidence during life.

Diagnosis.—Metastatic pneumonia is always a secondary affection, and when, in the course of thrombosis or of suppuration in some part of the body, chest symptoms develop, even if they be not very acute or definite, the diagnosis is not difficult. In other cases, where the source of the infection is deep-seated or not evident, as is not uncommon with inflammation in the abdomen or in the pelvis, chest symptoms may develop with such acuteness as to suggest idiopathic pneumonia; but in most of the cases the repeated rigors, frequent sweatings, irregular temperature, and the continued absence of marked physical signs, suggest a pyæmic origin, and enable the correct diagnosis to be made.

The pneumonias that occur in the course of specific fevers are sometimes placed in this, the hæmatogenous, group, and referred to the presence of the specific fever germs in the vessels of the lung. Probable as this may seem, there is, so far, but little proof of it, and there is good reason to believe that most of these pneumonias are due to no other than the ordinary cause.

The Prognosis in any case of secondary pneumonia is bad enough, but in metastatic pneumonia it is even worse, on account of the septic character of the inflammation and the risk of abscess and gangrene.

Most cases end fatally in the course of a few days. Even if life be preserved, the lungs are, as a rule, seriously injured, and the health is permanently damaged.

The **treatment** is the same as that for the other forms of asthenic pneumonia. The strength must be maintained by means of tonics, stimulants, and careful feeding. If gangrene, abscess, or other complication develop, it will require to be dealt with in the ordinary way.

39. INTERSTITIAL PNEUMONIA.

Inflammation of the interstitial tissue of the lung may be *acute* or *chronic*, and *primary* or *secondary*. In the great majority of cases it is chronic and secondary.

Acute Interstitial Pneumonia.—1. The most common form of acute interstitial pneumonia is that due to septic organisms which have gained access to the lung by the blood-vessels, and have been derived from some focus of septic inflammation elsewhere. Though primarily interstitial, the inflammation rapidly spreads to the alveoli and leads to more or less extensive consolidation. This is the condition described as *embolic* or *septic pneumonia*. Clinically it is a form of acute pneumonia, and has been already considered under that head. Embolic pneumonia frequently ends in abscess or gangrene; so, too, may acute ordinary pneumonia, though but very rarely. In the former case the interstitial tissue is

the seat of primary change, and in the latter it is involved secondarily by extension from the alveoli. The lesions, once established, are the same in either case, and their cause must be determined by other evidence.

2. In another remarkable but very rare form the primary seat of the inflammation is in the lymphatics. This is the affection described as lymphangitis pulmonaris, and in its most extreme form presents itself as *pneumonia dissecans*.

3. *Acute miliary tuberculosis* of the lung is also in many cases an acute or subacute interstitial inflammation excited by the presence and growth of the tubercle bacillus either in the blood-vessels or in the lymphatics.

Clinically the acute interstitial inflammations, even when not associated, as they usually are, with acute alveolar inflammation, present themselves under the same guise and are classed together under the group acute pneumonia. Embolic or septic pneumonia has been already considered. Acute tuberculosis belongs naturally to the general subject of tubercle of the lung. Abscess and gangrene will be treated in special sections, so that the only form of acute interstitial pneumonia which remains for consideration here is that of pneumonia dissecans.

Lymphangitis Pulmonalis — Acute Interlobular Pneumonia — Dissecting Pneumonia.—This affection, though common in cattle, is very rare in man. The symptoms presented are those of acute inflammation of the lungs, but the lesion found is an acute lymphangitis, the lymphatics in the affected part being distended with pus.

The lesion is sometimes limited to the subpleural and adjacent interlobular lymphatics, over, it may be, no large extent of surface, and it is then usually secondary to septic empyema.

The pleural surface of the affected part is marked with a meshwork of yellowish grey lines. These are the distended interlobular lymphatics, which may be followed on section some distance into the substance of the lung. When the lesion is more extensive, a larger portion, even the whole of a lobe, may be involved, and in some rare instances actually the whole lung. The inflamed lymphatics may then be traced by the same yellowish lines from the pleura to the root of the lung, and even into the mediastinum. On section they give the appearance as if the lung were riddled with small abscesses. The distension of the lymphatics may be so considerable as to strip off the pleura from the lung beneath, and, when the pleura has given way, the lung may hang free in the pleural cavity, suspended, as it were, by its bronchi. This constitutes the rare condition known as *pneumonia dissecans*.

In all cases alike the alveoli adjacent become involved in the inflammation, the exudation being of a catarrhal, fibrinous or hæmorrhagic character, and thus are formed patches of consolidation closely resembling those of broncho-pneumonia.

The number of cases of lymphangitis pulmonaris recorded in man are very few, hardly amounting to more than a score since the time when Carswell described the first case.

In many of them the affection has been a complication of an antecedent septicæmia.

Thus in Carswell's case it occurred in an adult who was suffering from chronic cystitis, and in infants it has been found associated with suppuration in the umbilical cord.

In others it has been attributed to direct extension of inflammation from the neighbourhood, though here, again, it is probably septic in nature.

Virchow,¹ Moxon,² and Cornil and Ranvier³ have observed it in connection with empyema. In Moxon's case it was associated with pus in the mediastinum. Similar cases have been recorded by Goodhart⁴ and by Wilson Fox.⁵ In the latter case the pus in the mediastinum was due to the tracking downwards of a retropharyngeal abscess, and probably preceded the lesion in the lung.

In a few instances the lesion was apparently primary in the lung; at any rate, no suppuration was found elsewhere.

Thus Damaschino⁶ records a case in a man aged 58, the victim of chronic gout, who was seized with a rigor and acute chest symptoms, and died after thirteen days' illness. The lymphatics of the whole right lower lobe from the pleura to the root of the lung were filled with pus. Wiedenmann⁷ also describes two cases in children of the same family living in the same house. The resemblance to the affection of cattle was so close that direct infection by milk was thought possible, but could not be proved.

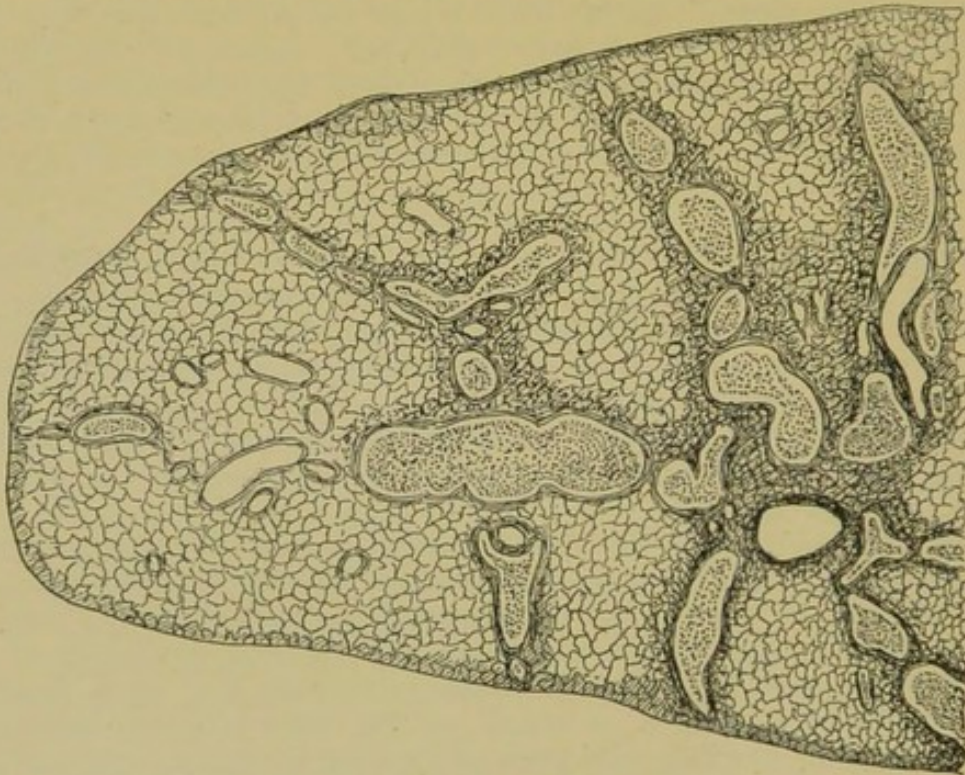


Fig. 78.

Pneumonia dissecans, showing the interlobular lymphatics distended with pus and pneumonic consolidation in the vesicles adjacent to them. (From Ziegler's *Pathol.*)

Instances in which the lesions have gone so far as to produce pneumonia dissecans are extremely rare.

Stokes⁸ records one, in which the lungs hung into the pleura like "a bunch of grapes." Rokitsky⁹ describes a similar case, the only one he had ever met with, and Wiedenmann's two children probably also belong to this group.

Many of the early cases recorded are, curiously enough, connected with cancer; of the stomach it would seem by preference,¹⁰ but also of the uterus, bones and breast.¹¹ Though in some of these cases the changes in the lymphatics are undoubtedly suppurative, in others the presence of numerous epithelial cells

¹ *Ges. abhandl.*, 469.

² *Manuel d'histol. pathol.*, ii. 126.

³ *Loc. cit.*

⁴ *D. Arch. f. kl. Med.*, 1880, xxxv.

⁵ *Path. Anat.*, iii. 72.

⁶ Blackey, Review in *Gaz. Hebdom.*, 1874, 345.

⁷ *Path. Soc. Tr.*, xxiv. 20.

⁸ *Path. Soc. Tr.*, 1887.

⁹ *L'union medic.*, 1879, p. 1046.

¹⁰ *Dis. of Chest*, 144.

¹¹ Raynaud, *Féréal. Gaz. d. Hop.*, 1894, p. 259.

makes the lesion peculiar, and somewhat similar cases are described by Wagner¹ under the name of cancer of the lung and pleura.

The affection presents itself under the clinical guise of septicæmia, of which it seems to be in most cases but a complication. When definite symptoms develop they are those of acute pneumonia, but there is no means by which the diagnosis from other forms of acute pneumonia can be made.

The disease is so rare as to be remarkable more for its pathological interest than for its clinical importance.

Most cases are fatal.² If recovery take place, it is stated that chronic induration will be left behind, but though this is theoretically probable, I do not know that the assertion rests upon any actual evidence.

Chronic Interstitial Pneumonia.—Chronic interstitial pneumonia means in plain English the presence of connective tissue in abnormal amount in the lung. The term would imply that it was the result of slowly progressive or antecedent inflammation, but it is applied to all conditions, whether inflammatory or not, in which such connective tissue growth has taken place, and it thus becomes synonymous with *fibroid induration*, *fibroid substitution* or *degeneration*, *fibrosis* or *cirrhosis of the lung*, by all of which names it has been described. Confusing as it is to have so many different names for the same pathological lesion, the confusion is still further increased by the addition to them of the term chronic pneumonia, as if chronic pneumonia and interstitial pneumonia were convertible terms.

The term cirrhosis is especially misleading, for if it mean anything more than indurative changes, it implies the existence of a primary interstitial fibrosis analogous to that described by the same name in the liver, and involving the whole of one lung or both. The very existence of such a disease, apart from tuberculosis, is, as will be seen, open to question. It would be well if this term, which has been and is the cause of so much confusion, could be eliminated from our nomenclature.

Chronic interstitial pneumonia thus comes by force of usage to mean nothing more than increase of connective tissue in the lung, whether of inflammatory origin or not, and in this general sense it will be discussed here.

Chronic interstitial pneumonia or fibrosis of the lung may be *unilateral* or *bilateral*, *partial* or *general*, *disseminated* or *localised*. It may be most developed round the air-tubes (*peribronchial*), round the vessels (*perivascular*), between the lobules (*interlobular*), or between the vesicles (*interalveolar*).

1. It is most widespread and general as a consequence of general bronchitis, and especially when this is due to prolonged irritation of the air-tubes by solid particles in the form of dust, as in the pneumo-konioses. But even when widespread, it is not necessarily uniformly distributed; on the contrary, it is usually more developed in some parts than in others, as at the apex or base.

2. In a more localised form it may follow broncho-pneumonia. It then occurs, especially at the bases of the lung, in children, and is associated with more or less bronchiectasis.

3. It may also occur in combination with other lesions; thus it may be the result of long-standing obstruction of the air-tubes by a foreign body, or of external compression of the lung by a new-growth, aneurysm or enlarged gland.

4. In another well-marked form it is *pleurogenic*, and spreads inwards along the dissepiments of the lung from the pleura, as is often seen in a case of chronic pleuritic thickening or in a lung collapsed by a long-standing pleuritic effusion.

¹ *Arch. d. Heilk.*, 1863.

² Zeigler, *Path.*

5. The commonest cause of fibroid change in the lung, whether partial or general, is chronic tuberculosis. Fibroid induration forms a very large part of the lesions of chronic phthisis, and it is probable that in most cases of diffuse interstitial induration, of which the nature is obscure, the original exciting cause has been tuberculosis.

6. Fibroid induration may also follow injury or destructive disease of any kind, mechanical or pathological, *e.g.*, contusion, laceration, tubercle or syphilis. It is the usual way in which the so-called healing of such lesions takes place.

7. It may follow lobar pneumonia, but very seldom.

8. It may possibly also arise as an independent idiopathic disease.

The pathological lesions are simple.

The fibroid parts of the lung are shrunken, hard, and cut like gristle. If near the surface, the pleura is also thickened and adhærent, and then the corresponding parts of the chest wall are retracted.

The changes consist in the multiplication of the elements of connective tissue. The interstitial tissue is thickened, indurated and pigmented, being of a black or slaty gray colour.

When the change affects the walls of the bronchi especially, as in the pneumo-konioses (*q.v.*), the condition is often described as peribronchitis fibrosa.

The changes are best studied in the walls of the alveoli. These become thickened owing to infiltration with small cells, the cells being at first round, but afterwards flattened between the bundles of newly-formed connective tissue. The alveoli are at first compressed, but end by being completely obliterated, the walls coming into contact and fusing together. Sometimes outgrowths or buds are formed from the walls which project into the alveoli, and finally fill them. In these ways parts of the lung may be converted into a solid mass of fibrous tissue, in which almost all trace of lung structure is lost. The fibrous parts are

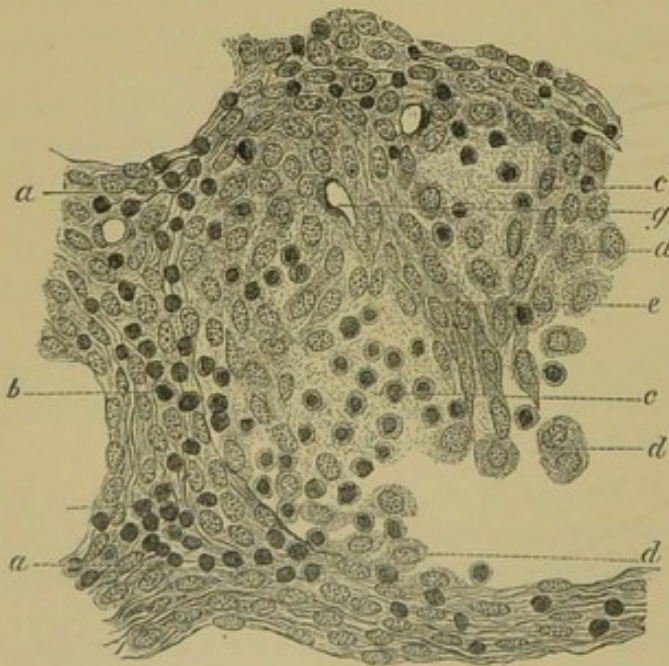


Fig. 79.

Intra-septal and intra-alveolar growth of connective tissue. *a*, alveolar septum showing fibro-cellular thickening and round-cell infiltration (*b*); *c*, cellular exudation into alveolus; *d*, formative cells within alveolus; *e*, strand of spindle-shaped fibroblasts; *g*, newly-formed blood-vessel within alveolus. (From Ziegler's *Pathol. Anat.*)

more or less pigmented, the pigment lying chiefly round the vessels and bronchi.

Although there has been a great disappearance of small arteries and capillaries, the larger arteries persist and remain pervious. The bronchi are for the most part obliterated, but the larger ones persist and frequently become dilated.

The coarse pathology or general appearance of the lung will vary according to the conditions under which the fibroid induration has developed, and the further description will find its proper place in the sections in which the different chronic affections of the lung are dealt with (Pneumokonioses, Chronic Tuberculosis, Bronchiectasis, etc.).

Physical Signs. — The physical signs vary according to the nature and extent of the mischief. If the lesion be small or deep-seated, there may be no physical signs at all, and such lesions are often found *post-mortem* which had yielded no evidence of their presence during life. If the induration be of greater extent, the part of the chest corresponding with it will be somewhat contracted and the movements defective. The percussion will vary, but is usually more or less impaired, and may be even dull; but if the parts around be emphysematous, it may be boxy or even hyperresonant. The voice and breath sounds vary according to the condition of the tubes, being enfeebled or exaggerated according as the tubes are much obstructed or not. If bronchiectasis is present, the signs of cavity may be obtained.

Symptoms.—The symptoms also vary like the physical signs, a slight amount of induration producing no symptoms at all and the larger amount causing more or less shortness of breath. Extreme cases lead to great dyspnoea and cyanosis, symptoms which are liable to sudden aggravation by the bronchitis to which these patients are especially liable. Fever is, as a rule, absent, so that elevation of temperature indicates some complication.

In the end, when the lesion is extensive, the nutrition fails, the patients grow thin and feeble, and pass into the condition which has been described as *fibroid phthisis*. This is, as a matter of fact, in most cases chronic phthisis, *i.e.*, chronic tuberculosis with fibroid induration.

Prognosis.—The prognosis varies with the extent of the lesion. Fibroid induration is, of course, irremovable, and the cure of chronic interstitial pneumonia therefore impossible; but unless the affection be progressive, or bronchiectasis be present, the duration is long. In the absence of bronchiectasis, the chief risk lies not in the affection itself but in the increased liability to chest complications, and the greater danger arising from them when they occur.

The **diagnosis** depends upon the physical signs and the chronic symptoms referred to, coupled with the evidence or history of some long-standing affection capable of causing the lesions, and among these, as will be seen, chronic tuberculosis plays a very important part.

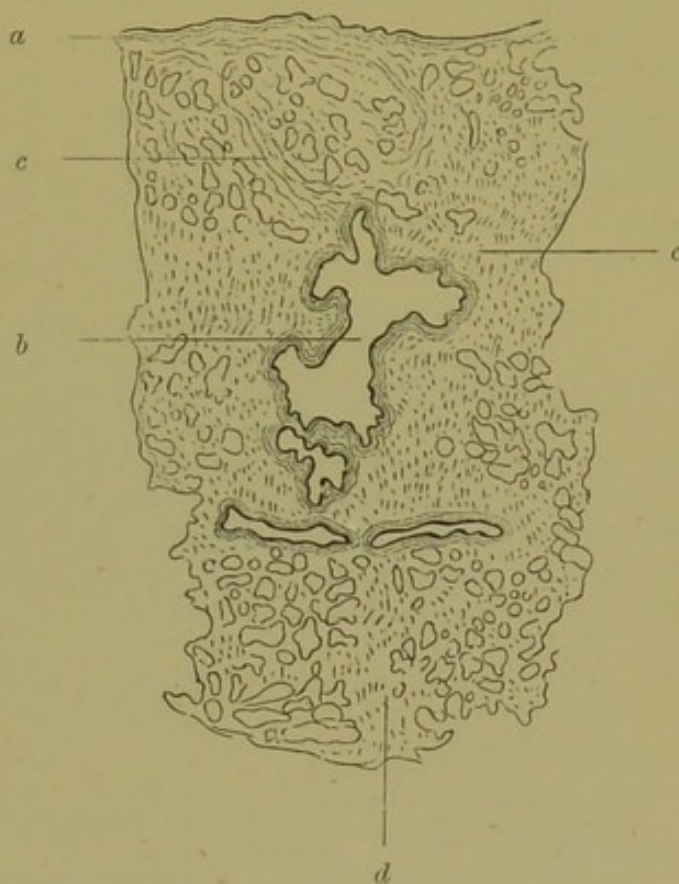


Fig. 80.

Chronic interstitial pneumonia (Hamilton, fig. 36).
a, thickened pleura; b, bronchiectatic cavity;
c, thickened lobular septum; d, thickened septum,
running inwards.

40. CHRONIC PNEUMONIA.

Chronic pneumonia is a clinical term which embraces various pathological conditions. It is not a disease but a set of symptoms, and although it is true that in many cases pathological changes of a fibroid or indurative kind accompany these symptoms, still they are not constant or necessary. Much of the confusion that prevails in this subject is due to the assumption that chronic pneumonia and interstitial pneumonia are convertible terms, though they are not, the one being a clinical condition and the other a pathological lesion.

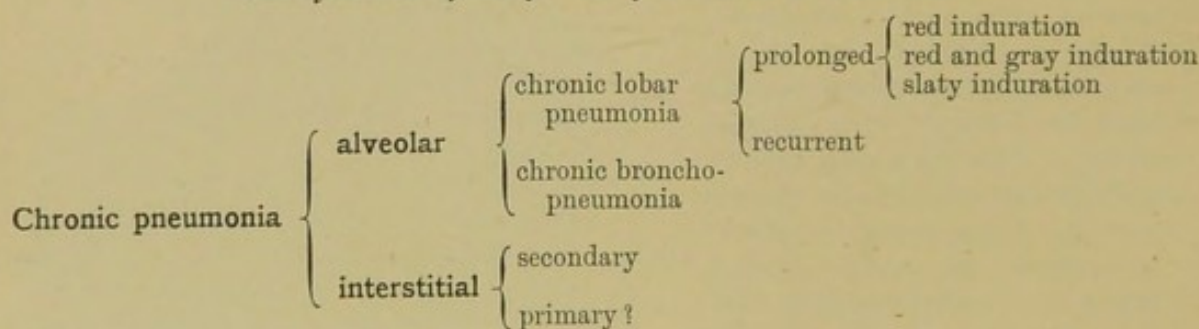
The term chronic pneumonia is applied to two different conditions.

1. To an inflammation which has continued for a long time and has become, as it is commonly called, chronic, *i.e.*, long-standing; and

2. To the pathological results of an affection possibly long past, and which may not have been, strictly speaking, at any time inflammatory.

Chronic pneumonia includes at least two groups of affections of the lung; the first, that of chronic or long-standing alveolar pneumonia in either of its forms, *viz.*, chronic lobar pneumonia and chronic broncho-pneumonia; and the second, that of chronic interstitial pneumonia.

Classification of the forms of Chronic Pneumonia.



Chronic broncho-pneumonia forms part of the chapters on broncho-pneumonia and bronchiectasis. Chronic interstitial pneumonia is in nearly every case secondary, and, so far, has been already considered. Two questions only remain to be discussed—(1) What are the lesions of chronic lobar pneumonia? (2) Is there such an affection as primary or idiopathic chronic interstitial pneumonia? And these two questions are more closely connected than might be anticipated.

Chronic lobar pneumonia.—After acute pneumonia resolution may be delayed, especially in the feeble or aged. In many of these cases the physical signs are simply slow to disappear and convalescence is tardy. Sometimes contraction of the side, corresponding with the part affected, occurs. This contraction, when not the result of the pleurisy with which the pneumonia has been associated, is due to collapse and shrinking of the lung and to persistence of the alveolar exudation. The lung is then found in the condition described by Charcot¹ as *red*, or *red and gray induration*. Microscopical examination shows nothing more than the persistence of the exudation, and the colour depends upon the amount of blood contained in the part and upon the changes the exudation has undergone.

In either of these conditions resolution may ultimately be complete and recovery perfect.

¹ *Rev. Mens.*, 1876, p. 776.

When recovery is incomplete, the parts usually remain contracted. In most cases this is due to chronic pleurisy, but in others to some indurative change in the lung itself, corresponding with the third form, described by Charcot as *gray* or *slaty induration*.

This induration is of two kinds: the one consists in fibrotic thickening of the interstitial tissue, and the other in the organisation of the intra-alveolar products. Both forms are rare. They are usually found associated together, but each may exist alone.

Of the first form there is nothing special to say; it is similar to that met with in so many chronic affections of the lung.

In the second form the exudation becomes infiltrated with small round cells, which gradually are replaced by spindle-shaped cells lying between the connective tissue fibrils. Attachments are formed with the alveolar walls, and along these blood vessels pass into the developing connective tissue in the alveoli. In the end complete union with the walls takes place and the alveoli are obliterated.

The organisation of the intra-alveolar products, which was pointed out by Virchow, and frequently described since, has been well figured by Coupland¹ and Green.² (Cf. fig. 79.)

When it is associated with interstitial thickening, difficulty will arise in distinguishing the intra-alveolar organisation from growths or buds of connective tissue springing from the walls. Such budding is often seen in chronic fibrosis of the lung, and especially in connection with chronic tuberculosis. When there is no interstitial thickening the nature of the change cannot be doubted.

Primary Indurative Pneumonia.—As to the relation in which the indurative changes, interstitial as well as intra-alveolar, stand to acute pneumonia,



Fig. 81.

Incompletely resolved pneumonia, showing the condition of "solid oedema." The vesicles are filled with serous fluid, in which a few cells are floating. The walls are a little thicker than normal, and the cells seem to be proliferating. (From a case in which the pneumonia occurred four months before death.)

¹ *Path. Soc. Tr.*, xxx.

² *Pathol.*, Ed. 3, p. 347.

great difference of opinion prevails, not, however, in respect of facts, but of the interpretation to be placed upon them.

Some authors hold that acute pneumonia cannot under any circumstances lead to induration, and that when induration is found after acute pneumonia, it is the result of some previous chronic disease or due to chronic tuberculosis. Others—*e.g.*, Charcot, Marchand, Ziegler—regard induration as a well-established, though a rare, result of acute pneumonia. Others, again, maintain that the inflammation of the lungs which ends in induration is a disease *sui generis*, and not related in any way to the ordinary acute pneumonia.

As these differences of opinion cannot be settled by pathological observation alone, clinical evidence becomes of importance, and this, so far as it goes, does not appear to lend support to the view that the condition has its origin in acute pneumonia.

In Coupland's case a man aged 41, who had suffered for ten years with winter cough, was attacked with subacute inflammation of the lung. He had much cough and shortness of breath and some pain in the side. The temperature varied between 100° and 103°, and he died of exhaustion after ten weeks' illness. In the consolidated parts of the lung, the alveolar exudation was found undergoing organisation; there were several small cavities, some with putrid contents, and also several secondary broncho-pneumonic abscesses, but no tubercular lesions old or recent. This is the usual clinical history of the affection, and is repeated in more or less the same form in most of the cases recorded.

The patients, though generally subject to some chronic pulmonary complaint, such as winter cough or bronchitis, have often been in good health until attacked by some chest mischief; usually of not very sudden access, of subacute course, and with rather indefinite physical signs and symptoms. The pyrexia is irregular and moderate, not rising as a rule above 102° or 103°. Rigors occur, but are repeated and usually not severe. The expectoration is not characteristic; it may be rusty and towards the end become putrid. The physical signs are at first indefinite, and suggest bronchitis rather than pneumonia, but gradually the signs of consolidation develop. The consolidation may ultimately involve the greater part or whole of a lobe, or even of a lung. Towards the end signs of excavation or of gangrene may develop.

The affection is of long duration, lasting some weeks or even a few months. The patients die gradually of exhaustion, with considerable dyspnoea and cyanosis, and with the signs of cardiac failure.

This clinical picture is unlike that of acute pneumonia, and the evidence, upon which the belief in antecedent pneumonia rests, is in most cases unsatisfactory.

If, then, as some hold, the affection is independent of pneumonia, the question arises whether it stands in any relation to tuberculosis. In favour of this view is the fact, that in a few instances the tubercle bacillus has been actually found, as for example in Talma's¹ case and in one of Heitler's. On the other hand, it has often been proved absent (Pal,² Heitler,³ Auld,⁴ Kidd⁵); and in some of the earlier cases, before the tubercle bacillus was recognised, the ordinary tubercular lesions and caseation are specifically stated to have been absent. On the whole the arguments against tuberculosis are strong.

If this affection be not the result of pneumonia, and not due to tuberculosis, nothing seems to remain but the conclusion that it is an independent disease to which the name *primary indurative pneumonia* might be given. This

¹ *Ztschr. f. kl. Med.*, x. 310.

² *Wien med. Woch.*, 1888

³ *Wien med. Woch.*, 1884, Dec. 13, and 1886, Mar. 13.

⁴ *Lancet*, 1890, i. 792.

⁵ *Ibid.*, 741, and *Path. Soc. Tr.*, xxxvii. 124.

term would embrace all cases alike, whether the lesion were primarily interstitial, primarily intra-alveolar, or both interstitial and intra-alveolar combined.

Buhl's desquamative pneumonia is a very indefinite disease, and inasmuch as Buhl regards caseation as the ultimate result, it is evident that the probabilities are in favour of its being a form of tubercular broncho-pneumonia, and of its standing in no special relation to the affection under discussion.

41. BRONCHO-PNEUMONIA—LOBULAR, DISSEMINATED, PATCHY PNEUMONIA.

Forms of Lobular or Patchy Pneumonia.—Lobar pneumonia is a very distinct and characteristic disease, and it is the typical form of acute inflammation of the lung met with in the adult. Lobular or patchy pneumonia, however, is by no means so definite, for under this term are often included many affections which, for the sake of clearness, it would be better to keep separate and distinct. In other words, lobular pneumonia is rather a lesion than a disease.

Embolic pneumonia.—Thus, when the infection is carried to the lung by the blood-vessels, the patches of inflammation are often multiple and widely disseminated, and as the terminal blood-vessels correspond in distribution closely with the ultimate divisions of the small bronchi and air vesicles, the resulting consolidation is lobular in form.

This condition is commonly described under the heading of *embolic, metastatic or septic pneumonia*.

Deglutition-pneumonia.—Another form of pneumonia which is often included under this heading also is that which is described as *deglutition-pneumonia*, that is to say, inflammation of the lung excited by particles of food which have passed into the air-tubes in swallowing.

In a healthy person, if a particle of food "goes the wrong way" it excites violent cough and is immediately expelled. In conditions of disease, where the general sensibility is impaired or the local sensitiveness of the larynx affected, this cough may not be excited and the body be not expelled, but make its way to the smaller air-tubes, choke them, and thus produce inflammation.

There are two groups of conditions in which deglutition-pneumonia is likely to occur. Thus, if the patient be more or less unconscious—*e.g.*, a child who has fits or tubercular meningitis, or an adult with apoplexy or some other form of coma—particles of food are very likely to pass into the larynx, and the pneumonia thus excited is a not uncommon cause of death.

The same thing may happen in the anæsthesia of chloroform or æther, as the result of vomiting.

In another group of cases there is some neuro-muscular affection of the parts about the glottis itself, as, for instance, in bulbar paralysis or in the local paralyses which follow diphtheria.

In still another group of cases the glottis may be prevented from closing either by inflammatory infiltration round it or by some destructive disease—tubercular, syphilitic or malignant.

In all cases of this kind nothing has so much diminished the risk of pneumonia as the introduction of the practice of nasal feeding.

Aspiration-pneumonia.—Aspiration-pneumonia forms another group of cases closely allied to the preceding, from which it differs chiefly in the fact that the particles introduced are not so gross as in the former instance, but such as are more likely to be carried readily by the air.

Under this head are commonly included those cases in which disseminated inflammation of the lung has followed the sucking into the small air-tubes of discharges of various kinds derived from lesions in the larynx, or in some of the larger air-tubes, as in tubercular or malignant disease, or from a disintegrating bronchial gland which has discharged into the air-tubes.

Aspiration-pneumonia is interesting because it is capable of experimental study. By varying the substances aspirated, from inert powders to actively infective substances, every form of lobular pneumonia can be studied, from simple inflammatory lesions up to the most intense forms which end in abscess or gangrene.

All these different forms of lobular pneumonia may occur in the adult as well as in the child, and indeed are commoner in the adult because the causes which excite them are commoner in them. They are all ordinarily included

under the term broncho-pneumonia, and in a pathogenic sense this is, generally speaking, correct, because the inflammations are in the great majority of cases due to some irritant introduced through the bronchi or air-tubes.

When the term broncho-pneumonia is used at the bedside as the diagnosis of a disease, it is ordinarily taken to mean that the case is one of *acute inflammation of the lung in a little child*, which is not of the lobar form. Certainly some distinctive term is required to denote this clinical group of cases, and it would be well if the term broncho-pneumonia could be restricted to that use, and the terms lobular, patchy or disseminated pneumonia used as the general descriptive term for the pathological lesion.

The ordinary broncho-pneumonia of children is usually described as following bronchitis, or as occurring in the course of those infective diseases which are so liable to be complicated with bronchitis, *e.g.*, measles, whooping-cough, diphtheria, typhoid fever, etc. But it arises also spontaneously, and that not infrequently, in cases in which there has been no antecedent bronchitis at all.

The broncho-pneumonia of little children therefore falls into two groups, according as it has been preceded by bronchitis or not, and these two groups may be called *primary* or *idiopathic*, and *secondary* respectively.

It would be well to call primary broncho-pneumonia idiopathic, *i.e.*, arising spontaneously, because some writers, especially of the French school, apply the term primary to those cases which follow simple or primary bronchitis, and secondary to those which follow the secondary bronchitis which develops in the course of the infective diseases.

The following tabular arrangement will make the subject clearer:—

- Lobular Pneumonia.—1. Embolic or metastatic pneumonia.
 2. Deglutition-pneumonia (*vagus-pneumonia*).
 3. Aspiration-pneumonia.
 4. Broncho-pneumonia.
 (a) Secondary, i. to bronchitis
 ii. to specific fevers.
 (b) Primary or idiopathic (*i.e.*, arising spontaneously without antecedent bronchitis or other illness).

SUFFOCATIVE CATARRH.—While discussing the forms of broncho-pneumonia, two more affections must be referred to, *viz.*, **capillary bronchitis** and **acute congestive broncho-pneumonia**. Both these affections are sometimes described as **suffocative catarrh**, and as a descriptive term this is good enough, for in each the prominent symptom is acute suffocation of which the patients rapidly die, but the two affections should be kept distinct, for they are really two different diseases.

Capillary Bronchitis.—Severe bronchitis may affect the small tubes chiefly, or rapidly spread from the large tubes to the small ones. This occurs frequently in small children and not altogether rarely in the adult. This affection being secondary to bronchitis, stands in close relation with the secondary broncho-pneumonia into which it runs if the patient survives long enough.

Acute congestive broncho-pneumonia belongs to the other or primary group, for it often arises in healthy patients who are not suffering from bronchitis. It is very rapidly fatal, in one or two days, or even in a few hours. On *post-mortem* examination there is nothing obvious to the naked eye except general congestion of the lungs; but on microscopical examination widespread inflammatory changes in the alveoli and minute bronchioles are evident, similar to those which, when less widespread, are characteristic of croupous pneumonia.

Under the name, then, of suffocative catarrh, we have two affections included which belong to two different clinical and pathological groups—the one to bronchitis and secondary broncho-pneumonia, the other to primary broncho-pneumonia and lobar pneumonia.

Suffocative Catarrh.

Capillary bronchitis.
Secondary broncho-pneumonia.
(Bronchitis Group.)

Acute congestive broncho-pneumonia.
Primary broncho-pneumonia.
(Pneumonia Group.)

Henry W., aged 12, a strong and healthy child, went to bed well, but woke up an hour later crying with pain in the pit of the stomach and bringing up some "congealed" blood from his mouth.

He was taken immediately to the hospital, spitting up blood on the way and vomiting several times.

When seen he was found to be much cyanosed, suffering with great dyspnoea, gasping for breath, constantly coughing up bright red frothy blood, and groaning with pain over the præcordium. The pulse was 120, respiration very rapid, and the temperature 96°. Loud, coarse crepitation was heard all over the chest, but there was no dulness to percussion or bronchial breathing. The apex of the heart was displaced 2 inches outwards, the cardiac dulness increased upwards and to the right, and a loud presystolic murmur audible.

Throughout the night the symptoms grew steadily worse, the cyanosis increased, the frothy blood-stained fluid continued to be coughed up, and some was also vomited.

The dyspnoea and cyanosis at last became extreme, and the lad died of suffocation, seventeen hours only from the onset of his illness.

The temperature, which was 96° on admission, slowly rose and reached 100·6° just before death.

At the autopsy all the organs proved healthy except the heart and the lungs.

The heart showed well-marked mitral stenosis. The lungs were firmer and heavier than normal, the right weighing 19 ounces and the left 18 ounces. They were greatly congested and cedematous, a frothy sanious fluid flowing copiously from the surface on section, the bronchi also containing much of the same fluid. No consolidation was found anywhere, but the congestion and cedema were most marked in the right lower lobe, over which the pleura was intensely congested.

Microscopical examination revealed an interesting condition; the vessels throughout were greatly distended, as in the heart-lung. The alveoli were completely filled with a slightly turbid coagulable fluid, in which were contained numerous cells, both epithelial and lymphatic, as well as some blood. The small bronchi were filled with similar contents.

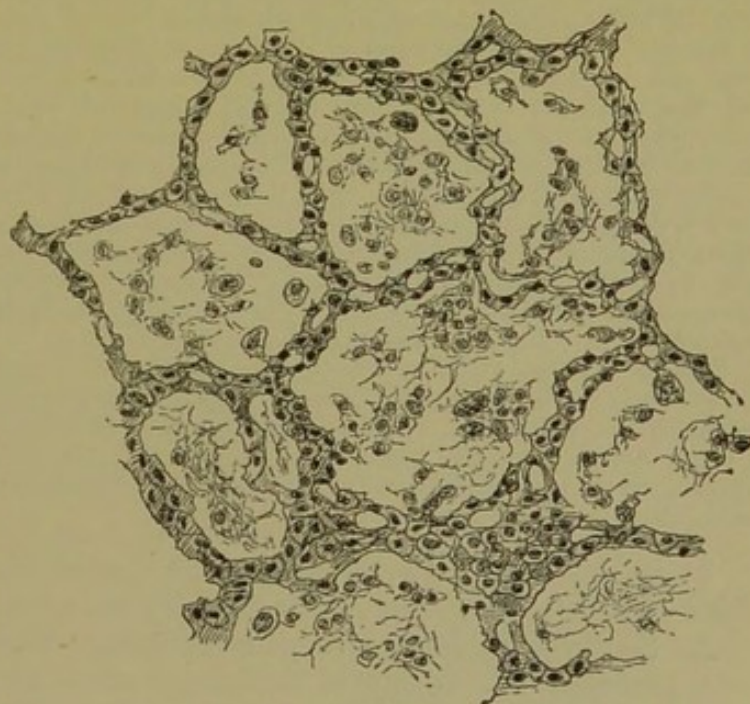


Fig. 82.

It is difficult to fix exactly the category in which this case should be placed. Clinically it was an instance of acute suffocative catarrh occurring in a heart-lung. Pathologically it may certainly be described as a case of acute congestion, but whether this is to be regarded as the initial congestive stage of acute inflammation, *i.e.*, of pneumonia, is open to question—for though the onset was very sudden there was no rigor, nor did the temperature rise in the way it usually does with the onset of pneumonia. Lastly, the age of the patient presents a difficulty, for except in young children acute suffocative catarrh is very rare.

The case may be well compared with one already described in the chapter on acute pneumonia; that in a young lady in whom there was also mitral stenosis. The onset in her case also was sudden. There was much dyspnoea and hæmoptysis, and the temperature was not high. As the general congestion of the lung subsided, the signs of right apex-pneumonia developed. It may be that in this boy also, from the predominance of the congestion and œdema in the right base, the ordinary signs of pneumonia would have developed there, had the patient lived longer.

42. THE BRONCHO-PNEUMONIA OF LITTLE CHILDREN.

THE PATHOLOGICAL LESIONS.—Broncho-pneumonia has been experimentally studied by introducing various irritating substances into the air-tubes of animals, either by direct injection or by causing the animals to inhale contaminated air. The substances chosen have been either chemical irritants or fluids containing various pathogenic or putrefactive organisms. The results have in either case been much the same, and differed only in degree according to the intensity of the irritation.

The greater part of the substances so introduced are deposited upon the walls of the large air-tubes, where they produce ordinary bronchitis. In the course of time, however, they reach the alveoli and excite inflammation there.

In the alveoli, the first effect of the irritation is an active congestion, which is followed by exudation into the vesicles, sometimes serous, sometimes fibrinous, and sometimes more or less hæmorrhagic according to the intensity of the inflammation. The affected alveoli become filled with epithelial or catarrhal cells and leucocytes, both of which may contain some of the inhaled substance. The large epithelial cells are derived from the vesicular epithelium, which is at once shed and sometimes detached in shreds; the white cells are derived from the blood vessels and the exudation may contain a good many red cells. The cells are for the most part fatty, granular and opaque. In some of the alveoli the exudation may be fibrinous, and thus the consolidation closely resemble microscopically that met with in croupous pneumonia.

The patches of consolidation thus produced may be very small, and are then spoken of as *acute miliary broncho-pneumonia*. In most cases they are of somewhat larger size, and in the ordinary form affect the groups of vesicles which constitute a lobule. If these lobular consolidations be closely aggregated or of large size, as they sometimes are, the resemblance to lobular pneumonia may become very close. These are also the lesions of what has been called *vagus pneumonia*, *i.e.*, pneumonia following section of the vagi.

The result of the inhalation depends upon the nature and amount of the substance inhaled. If the substance be not large in amount and not very irritating, small miliary patches are the result; if the amount be larger or the substance contain infective organisms, the inflammation is more intense, produces larger patches, and may, under certain circumstances, run on into suppuration or gangrene; or if, for instance, the tubercle bacillus be present, acute or sub-acute tuberculosis will be excited.

In children with broncho-pneumonia the lesions are much the same as those which have been produced experimentally in animals.

A. Secondary Broncho-pneumonia.—Where the broncho-pneumonia has been preceded by bronchitis, the alveoli may become involved in two ways—by the inflammation spreading either along the bronchial tubes to the alveoli or directly through the bronchial walls to the alveoli adjacent.

Usually where there is much bronchitis the sequence of events is this: the smaller tubes become plugged with secretion; the alveoli corresponding with them lose the air they contain and become collapsed; the collapsed vesicles subsequently become inflamed either by direct extension of the inflammation to them or as the result of the aspiration of the secretions.

It is this form which, in clinical medicine, is usually understood by the term broncho-pneumonia, *i.e.*, local patches of acute consolidation disseminated irregularly through the lung resulting from antecedent bronchitis.

In the great majority of cases (82 per cent., Holt) the patches are irregularly distributed through the whole of both lungs, but usually in greater abundance in the lower lobes posteriorly. It is rare for the anterior parts of the lung alone to be affected, and then the consolidation appears to be most frequent in the right apex.

In the lung from a case of this kind, the bronchial tubes present the ordinary lesions of bronchitis and contain much mucopurulent secretion, which may be squeezed out in drops or worm-like threads. The smaller tubes are often so completely filled with secretion, that on section they look like small abscesses, and it is this condition which has been described under the name "*Vacuoles Pulmonaires*." In some cases microscopical examination shows that these small collections of pus are really abscesses, *i.e.*, that there has been a distinctive suppurative process involving the bronchial wall and spreading through them to the alveoli beyond (*cf.* Bronchiectasis, p. 187).

The lung itself is mottled in colour; the result of being in parts collapsed, in parts emphysematous, and in parts consolidated. The *collapsed* parts are bluish-brown or mahogany-coloured, somewhat depressed below the surface and airless; they may be isolated in small patches, or involve a considerable portion of the lobe. The *emphysematous* parts are bright red and crepitant. The *consolidated* parts reddish or yellowish gray; somewhat prominent above the surface, airless, and more or less dry on section.



Fig. 83.

Section of recent broncho-pneumonia. *a*, alveolar septum with distended vessels; *b*, epithelial plates detached, partly nucleated, partly non-nucleated; *c*, do., containing granules; some surrounding nuclei (*d*); *e*, small epithelial cells detached, with visible nucleus and slightly altered protoplasm; *f*, swollen granular epithelial cells, with nucleus concealed; *g*, colourless blood-cells. (Ziegler, *Path. Anat.*)

The affection appears in most cases to develop in the following way:—The catarrh spreads from the larger tubes to the capillary bronchi, and these become

plugged with the secretion; the air in the corresponding alveoli is absorbed, and the parts collapse. Such a collapsed portion is reddish in colour, depressed below the surface, and airless, but as long as it is only collapsed it can be easily blown out again. Soon inflammation develops in the collapsed portion, the epithelial cells are shed, white blood cells emigrate, and more or less of exudation occurs from the vessels. The inflammatory products are of a catarrhal type, that is to say, the epithelial cells predominate over the white blood-cells and the exudation is serous; hence the name, *catarrhal* or *desquamative pneumonia*, by which the affection is often described. Not infrequently, however, the inflammation is of a more active character; the white blood-cells are more numerous, red

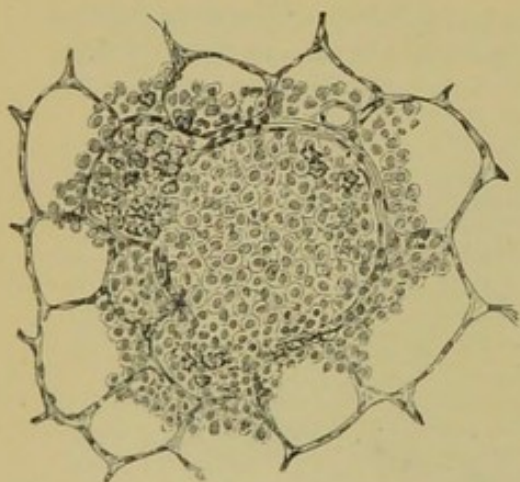


Fig. 84.

Miliary patch of broncho-pneumonia, involving a fine bronchiole, and the adjacent alveoli. Preparation from a dog's lung. Some of the cells contain granules of the substance inhaled, by which the inflammation was provoked. (Ziegler, *Path. Anat.*)

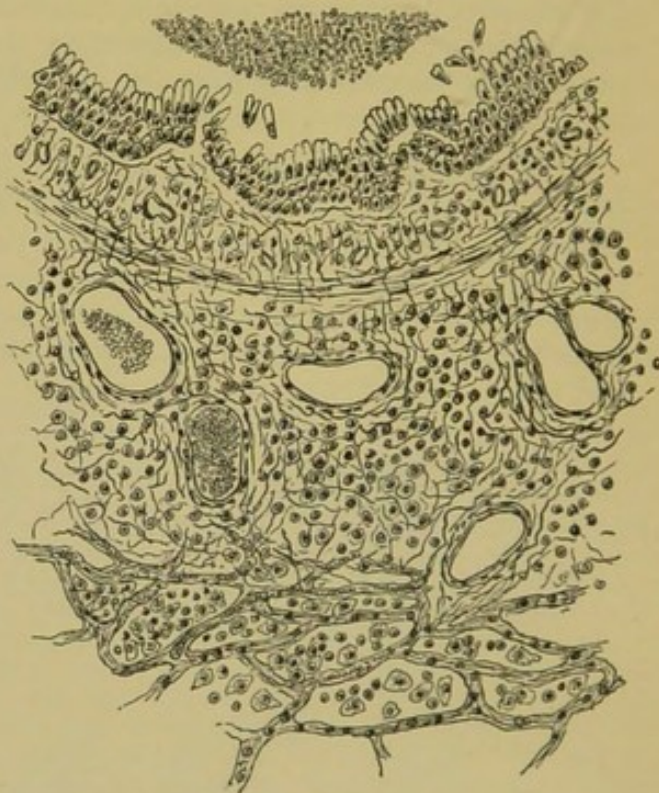


Fig. 85.

Section of a bronchus and adjacent alveoli from a case of acute bronchitis with broncho-pneumonia. The infiltration with small cells of the deeper bronchial and peribronchial tissues is well shown. The inflammation has spread through to the adjacent alveoli, which are full of cells.

blood-cells as well as fibrinous exudation are present, so that in some of the alveoli is found a consolidation which does not differ in any material respect from that found in croupous pneumonia.

The inflammation is rarely confined to a single infundibulum or group of alveoli, *i.e.*, it is rarely miliary, but usually leads to a consolidation of larger size and is called lobular. By the confluence of many such lobules the greater part or even the whole of a lobe may be involved, and in this respect, again, catarrhal pneumonia may come to resemble croupous pneumonia, but as a rule in such cases the section of the consolidation has a more gray or reddish-gray colour; the characteristic granulation of croupous pneumonia is absent, and the consolidation is not so uniform, that is to say, it is made up of patches of consolidation with portions of collapsed and congested lung between them.

Where the bronchitis is severe and the deeper bronchial tissue much involved, the inflammation may spread directly through the walls to the alveoli adjacent, producing patches of peribronchial rather than lobular inflammation.

Owing to the infiltration of the bronchial walls bronchiectasis often occurs in an acute form; the dilatation is sometimes cylindrical, that is to say, involves some length of the bronchial tube more or less uniformly, and at other times it is patchy and the dilatation described as moniliform.

The bronchial glands are frequently enlarged; but this occurs generally in persons who have been subject to prolonged bronchitis, to which it is probably to be referred rather than to the broncho-pneumonia.

B. In Primary Broncho-pneumonia, that is to say, in cases where the broncho-pneumonia has not been preceded by bronchitis, the alveolar lesions are as a rule of a more acute inflammatory character, and resemble closely those of croupous pneumonia, from which they differ chiefly in extent only, the inflammation being lobular and not lobar.

RESULTS OF BRONCHO-PNEUMONIA.—In the acute cases resolution is the rule, and recovery becomes complete. The intra-alveolar products liquefy, the cells undergo fatty degeneration, and the products are either expectorated or absorbed, the alveolar epithelium and the bronchiolar epithelium are regenerated, and in time the affected parts are restored to the normal.

In the primary broncho-pneumonias, resolution is almost as rapid as it is in the case of true pneumonia, but in the secondary broncho-pneumonia recovery is delayed by the presence of bronchitis and by the changes which have been excited in the larger air-tubes.

In all severe cases the interstitial tissue is more or less involved, and this is especially likely to occur in the more protracted or relapsing cases. The interstitial infiltration may also in time completely disappear. If not, more or less fibroid induration will be the result.

The patches of induration are for the most part small, and, like the original inflammation, irregularly distributed through the lung, so that they do not produce any serious interference with the function of the organ. Where the inflammation has been massive, the resulting induration may be considerable. This is especially the case in the lower lobe, which may thus be converted, in the whole or great part, into fibrous tissue, in which the only trace of lung tissue left is found in the saccular bronchiectatic cavities with which it is riddled, and which give it its resemblance to Gruyère cheese (see fig. 42).

As already stated, the infection which causes the original acute inflammation is very often a mixed one, and, if septic or putrefactive organisms are present, the consolidation may suppurate or even gangrene, and thus a destructive disease of the lung be produced which may end in death.

Among the specific organisms which seem to find an especially suitable soil for growth in the lesions of broncho-pneumonia is the tubercle bacillus, whether it has been introduced at the time or by subsequent infection. At any rate it is not at all rare to observe the lesions of phthisis develop after what was apparently at first nothing but a simple broncho-pneumonia, and in all the protracted or relapsing cases it is this question which is sure to arise, namely, whether the duration or recurrence of the disease is not due to the presence of tubercle. None of these lesions last described, however, have anything to do essentially with broncho-pneumonia, their association being entirely accidental.

Pleurisy, though not uncommon when the inflammatory patches are near the surface of the lung, results in little more than slight exudation, which rapidly resolves, and at the most leads to a local adhesion, which in time may also disappear.

Pleuritic effusion is rare with broncho-pneumonia, and, when it occurs, is likely to be purulent, as would be expected in children.

If a superficial patch of consolidation were to break down, it might perforate the pleura and lead to pneumothorax. This is an extremely rare complication, of which Steffen records two instances and Pepper one, but the majority of writers content themselves with simply mentioning the possibility.

Among other rare complications are purulent meningitis, otitis media, noma, acute endocarditis or pericarditis. Such lesions are accidental associations, the result of some general infection to which the broncho-pneumonia itself is due. The same may be said of abscess of the brain, of which the following is an instance.

A boy of 8 who had been subject to cough for two years, and had never been strong, was attacked with broncho-pneumonia of the right base. The attack was of no great severity, but the physical signs were well marked. Three weeks later the child was seized with fits on the left side chiefly, and in two days became hemiplegic on that side. The fits recurred, and a week later double optic neuritis was found. Ten days after the first fit the child became comatose and died. At the autopsy the base of the right lung was found collapsed with some diffuse suppuration in it, and a sloughing partly gangrenous patch in its centre. The central part of the right hemisphere of the brain was occupied by a large recent abscess.

ETIOLOGY.—The etiology of broncho-pneumonia is to a great extent the same as that of bronchitis.

It prevails especially in cold, damp, changeable weather, *i.e.*, chiefly during the winter and spring.

All conditions of debility increase the liability. Thus it is common in the ill-fed, ill-kept children of the poor, in those who have been weakened by long confinement within doors or to bed, and in those who are the subjects of constitutional diseases such as rickets, syphilis or tubercle.

Undue prevalence of broncho-pneumonia is often explained by the prevalence of one of the epidemic infectious diseases, of which broncho-pneumonia is the common complication, such as measles, whooping-cough, influenza or diphtheria.

The relative frequency of Primary and Secondary Broncho-pneumonia is thus stated by Holt:—

Primary— <i>i.e.</i> , without previous bronchial affections,	.	.	.	154	}	433
Secondary—						
1. To bronchitis of larger tubes,	.	.	.	41	}	279
2. To some infectious disease—						
Measles,	.	.	.	89	}	238
Whooping-cough,	.	.	.	66		
Diphtheria,	.	.	.	47		
Scarlet fever,	.	.	.	7		
Influenza,	.	.	.	6		
Varicella,	.	.	.	2		
Erysipelas,	.	.	.	2		
Acute ileo-colitis,	.	.	.	19		

Broncho-pneumonia, whether primary or secondary, is met with chiefly in the early years of childhood, and becomes less and less frequent as the fourth and fifth year approach, and is quite rare after that age.

Of 426 cases (Holt):

53 per cent. occurred during the 1st year.				
33	"	"	"	2nd "
11	"	"	"	3rd "
2	"	"	"	4th "
1	"	"	"	5th "

As broncho-pneumonia diminishes in frequency, lobar pneumonia increases in frequency, so that the two vary inversely.

Statistics showing frequency of broncho-pneumonia at different ages in children and its relation to lobar pneumonia at the same ages (Holt):

	Broncho-pneumonia.	Lobar pneumonia.	Proportion.
Under 6 months, . . .	73	11	6·7 to 1
From 6 to 12 months, . . .	96	29	3·3 to 1
In 2nd year, . . .	73	40	1·8 to 1
In 3rd year, . . .	19	23	'8 to 1
In 4th year, . . .	0	6	...
	261 (75 per cent.)	109 (25 per cent.)	

At St. Bartholomew's Hospital, during the ten years from 1887 to 1896 inclusive, there were admitted 412 cases of broncho-pneumonia, of which 82 per cent. were under 5, and 11·6 under 10 years of age. At the same time there were admitted 1786 cases of croupous pneumonia, of which 14 per cent. were under 5 and 16·4 per cent. under 10 years of age.

The actual numbers of cases under 5 years of age were—

Croupous pneumonia, 250, with a mortality of 7·2 per cent.
Broncho-pneumonia, 331, " " 37·5 "

These facts raise some interesting questions.

1. Why is it that bronchitis, which so commonly leads to broncho-pneumonia in very young children, is so much less likely to do so as the children become older?

2. Why is it that acute inflammation of the lungs in little children tends to take the lobular or broncho-pneumonic form, and but rarely the lobar form?

It can hardly be said that children are less susceptible to bronchitis after the age of four years than before. Yet broncho-pneumonia becomes much less frequent after that age.

Nor does it seem reasonable to assert, if lobar pneumonia be due to a specific infection, that children under four are less susceptible than children above that age, especially as the pneumococcus is found almost as frequently in the broncho-pneumonia of little children as it is in the lobar pneumonia of the adult.

If, then, the common inflammation of the lung in little children is of the lobular form, and in the older child as in the adult of the lobar form; it seems probable that the explanation lies not in a difference of cause, but in the difference of age; in other words, that the form inflammation takes in the little child depends upon some peculiarities in its lungs, as compared with the adult. The question then arises whether there are such peculiarities in the child's lung as seem adequate to explain the difference.

Now the respiratory mechanism in children presents many peculiarities. The chest-walls are elastic and yielding owing to the incomplete ossification of the ribs, and the thorax approaches the cylindrical in form; the type of respiration is diaphragmatic rather than costal, and remains so up to about the sixth or

seventh year; very slight causes easily produce irregularities of respiration and may even lead to local defects of expansion in different parts of the lung.

In structure also the lungs of children present many notable differences from that in the adult. Thus the trachea and main bronchi in the young child are relatively much larger; the other bronchi are also larger and occupy greater relative space; the air-cells are smaller and occupy less relative space; the interstitial tissue of the lung is more abundant; the epithelial lining of the small bronchi and alveoli is relatively more bulky, the cells are of relatively larger size, and they respond more actively to slight irritations, under which they proliferate readily and are easily shed.

With all these peculiarities in the child's lung, it can hardly be a matter of surprise if inflammation in it should assume a peculiar form.

In fact, the conclusion to which these considerations lead, and which I do not hesitate to adopt, is this, that the same exciting cause—*e.g.*, a pathogenic organism such as the pneumococcus—which in the adult and older children excites a lobar inflammation, in the young child usually causes a lobular inflammation.

If this be so, the distinction drawn between primary or idiopathic broncho-pneumonia and secondary or catarrhal broncho-pneumonia becomes of importance, both on pathological and clinical grounds; for while the secondary broncho-pneumonias stand, as is generally accepted, in close relation with bronchitis, the primary broncho-pneumonias stand in still closer relation with lobar pneumonia, of which they are but another anatomical form.

I propose to deal first with secondary broncho-pneumonia, because it is the generally recognised and familiar form, and that upon which all the text-book descriptions are based. I shall then deal with primary broncho-pneumonia, and give the reasons for separating the two forms.

SECONDARY BRONCHO-PNEUMONIA.

Clinical History.—The child will have been suffering, as a rule, from evident bronchitis prior to the attack, and will present the ordinary symptoms and signs of that affection. Possibly even extensive collapse may have previously developed at the base of the lungs posteriorly, sufficient to yield an impaired percussion note.

The onset of the broncho-pneumonia is usually indicated by general aggravation of symptoms, by further rise in the temperature, by acceleration of the pulse- and respiration-rate, by an increase in the dyspnœa, by the cough becoming less paroxysmal and more painful, and, possibly, by the occurrence of delirium if there has been none before.

General Condition.—At this stage the child is extremely restless owing to the fever as well as to the dyspnœa and cough. It tosses itself about from side to side, and if it fall asleep it is but for a minute or two at a time. It often cannot lie down, but has to be propped up with pillows.

The expression is anxious and distressful, the cheeks are flushed, the eyes suffused, the *alæ nasi* dilating, the dyspnœa considerable, and the respirations rapid and panting, and in infants often irregular.

Physical Signs.—The physical signs are those of bronchitis, and are present on both sides of the chest, though probably more marked on one side than the other.

In infants, the upper parts of the thorax are prominent, and the lower somewhat contracted. Inspiratory recession is generally present to some extent, and in rickety infants may be extreme, especially at the lower part of the thorax.

The percussion note is usually hyperresonant everywhere, except, it may be, at the bases behind. Rhonchus, sibilus and crepitation are heard more or less over both sides.

In all these signs there is nothing characteristic or different from what is met with in bronchitis, for the patches of consolidation are as a rule small, and so surrounded by emphysematous lung that they give rise to no alteration in the percussion note, while bronchial breathing and bronchophony, when present, are often masked by the rhonchus and sibilus.

Even when the consolidation is extensive enough to produce impaired percussion, bronchophony and bronchial breathing may be absent owing to the plugging of the tubes with secretion.

But, on the other hand, it sometimes happens that even when the consolidation cannot be made out by percussion, bronchophony and bronchial breathing may be heard distinctly here and there, and the crepitation acquire a sharp, clear, ringing quality which is very characteristic.

The physical signs vary greatly from time to time, and that within very short periods. Rhonchus, sibilus and crepitation vary, of course, as they do in bronchitis; even bronchial breathing and bronchophony may come and go, and dulness, which may be present one day, may not be detected the next.

Collapse, when associated with patches of consolidation, may greatly increase the extent of dulness at the time, and with the exaggerated voice and breath sounds may give all the signs of an extensive or lobar pneumonia. If in such a case the dulness suddenly or very rapidly disappear, this can only be due to the disappearance of the collapse, the vesicles having become expanded again and filled with air, thus masking, as at other times, the patches of consolidation which they surround.

This change in the percussion I have seen several times at the base, and in the most marked case of the kind I have met with, the whole of one side posteriorly was dull at the time of examination; yet within twenty-four hours the dulness had completely disappeared, although the evidences of consolidation still persisted in patches.

I have seen collapse cause displacement of the heart. This occurred in a child with extensive collapse of the left base. The heart's apex was felt half-an-inch outside the left nipple; it receded into its normal place as the collapse passed off.

When consolidation develops, it is stated not to become evident to physical examination until three to eight days after the commencement of the attack, but this is an assertion which it is obviously even more difficult to prove in broncho-pneumonia than in croupous pneumonia.

The younger the child the more difficult it is to depend upon the physical signs alone for diagnosis.

Respiratory System.—The respirations are short, panting and very rapid, running up to 60, 80, or even 100 in the minute; their rapidity increases as a rule as the disease progresses. They are often irregular, but in small children the regular rhythm of breathing is easily disturbed from very slight causes. Irregularity of breathing, therefore, in the early stages is of little significance. In the later stages, when the respirations have become less energetic and more shallow, they are often irregular, and may even be suspended for a few seconds at a time. Irregularity of this kind is a bad sign, for it is often only a form of Cheyne-Stokes breathing, and indicates failure of the respiratory centres or of the heart.

The dyspnoea is usually out of all proportion to the physical signs. It is liable to exacerbations at night and in the morning. It may be paroxysmal, the paroxysms being generally produced by the accumulation of secretion in the tubes, but they are occasionally of laryngeal origin and due to laryngeal catarrh or spasm.

The cough is short, hard and hacking, very frequent and often painful, but less paroxysmal and violent than in bronchitis.

Expectoration is rare in children, for the secretion, if coughed up, is swallowed. If there be any sputum it is bronchitic in character, and may be streaked with blood or even rusty.

If the cough be paroxysmal and end with vomiting, the possibility of whooping-cough must not be overlooked, for with the onset of broncho-pneumonia the characteristic paroxysms usually diminish and the whoop disappears, to return when convalescence is established. It not unfrequently happens, when the cause of the attack has been obscure, that it is made evident by the occurrence of the whoop during convalescence, without there having been anything previously to suggest that affection.

Circulatory System.—The pulse is extremely rapid, and often reaches 140 or 150; it may even run up to 200 and be almost uncountable. The pulse-respiration ratio may be as 1 to 1.5 or 2, but this may occur in children with bronchitis as well as with lobar- and broncho-pneumonia; so that too much stress must not be laid upon it in the diagnosis between these affections. The pulse, however, often remains high in broncho-pneumonia after the temperature has fallen, while in croupous pneumonia the fall in the pulse rate at the time of the crisis is almost constant, and may be the first indication of the coming of the crisis.

If the pulse beats become indistinct and running, and still more if they become irregular, the prognosis is bad. During convalescence, however, an intermittent pulse is often observed in young children after broncho-pneumonia, as after any other severe illness; it suggests the need of care, but indicates no special danger.

The sounds of the heart are weak, short and flapping, and its action irregular, but, as with the respirations, irregularity has not so great a significance in young children as in the adult.

Dilatation of the right side of the heart is frequent in severe cases.

General dilatation of the heart may be found during convalescence, when the lung symptoms have disappeared, as in convalescence from other severe diseases. It is often present without obvious cardiac symptoms, and may be discovered only on physical examination. It is associated with general feebleness and anæmia, and is easily increased or produced by exertion. It is not specially important, but indicates the need of care and rest.

Cutaneous System.—The skin is usually moist and sweating. It feels warm, though rarely as pungently hot as in lobar pneumonia. During the course of the disease transient sweatings, like transient flushings, are not uncommon, but towards the end, when cyanosis is extreme, the body may be bathed in a cold clammy sweat, which is of bad omen.

Herpes is almost unknown, but excoriations often develop round the mouth and nose, and ecthymatous spots are not uncommon on the body.

Specific rashes should always be looked for. The most frequent to be found is, of course, that of measles, in which affection bronchitis and broncho-pneumonia may sometimes develop at the very commencement, and long precede the appearance of the rash.

With the development of broncho-pneumonia, or of any other acute disease, the specific rashes tend to fade greatly, a fact which accounts for the popular explanation that the complication is due to the rash having struck inwards.

The Temperature.—The fever is of a remittent or hectic type, and presents very irregular variations. Though it may reach a considerable height every day, even 104° or 105°, it does not remain at so high a level for long, perhaps not for more than an hour or two, and falls then to 100° or 101°, to rise again the next day.

The temperature is usually at its maximum in the evening, but the highest point may be reached at any time of the day or night. In this respect great differences exist between different cases, and, indeed, in the same case at different times (see charts, pp. 341 to 346).

As a rule, the more persistently high the temperature remains, the more extensive is the lung mischief likely to prove, and the more severe the case. High fever is especially liable to occur in the broncho-pneumonia of measles.

In fatal cases the temperature may run up to a great height just before death, even to 107° or 108° , but it may also fall rapidly, even to a point much below the normal.

The fever lasts rarely less than fourteen days and ends by lysis.

As broncho-pneumonia is especially prone to relapse, the disease may in this way be greatly protracted even over many weeks, and the occurrence of relapse is indicated by the return of fever, but the relapses are often of shorter duration than the original attack, though they may recur very frequently.

Digestive System.—The appetite is impaired or completely lost, and thirst is considerable. Vomiting is a common symptom, but often due only to the coughing. Food is frequently refused, especially when swallowing increases the dyspnoea and cough, as it often does; and infants may decline the breast because of the difficulty in breathing whilst sucking. These difficulties in little children may necessitate nasal feeding.

The tongue is usually moist, but it may in bad cases be dry and cracked, and the mouth be covered with sordes. In infants thrush, and in older children aphthous stomatitis is likely to develop.

Diarrhoea is frequent; it has been attributed, as in bronchitis, to the swallowing of the expectoration, and the irritation caused by its decomposition in the intestines; but it may be an early symptom, and produced by the same chill which excited the bronchitis, *i.e.*, the chill may cause acute catarrh of the stomach and intestines, as well as of the bronchi.

Urinary System.—The urine presents no special characters. It sometimes contains a little albumen, but this is, in most cases, due to the fever, or, where there is much cyanosis, to the obstruction to the circulation through the lungs.

Acute nephritis has been described, but it is very unusual, and probably an entirely accidental complication.

Nervous System.—The nervous symptoms are rarely so marked as in croupous pneumonia, and the confusion with meningitis is less likely to arise. A little delirium is common enough, but it is not often extreme. Fits occur frequently at the onset, but rarely during the course of the disease. Towards the end, when the cyanosis is extreme, they may recur, and are then usually the precursors of death.

Access.—The access of the affection varies somewhat in different cases. With *acute bronchitis*, broncho-pneumonia often sets in suddenly, especially in weakly, feeble children, so that the two affections appear almost to begin together. In subacute and chronic bronchitis, it appears later. In *measles* it usually follows the eruption and develops in the second week, but it may precede the rash. In *whooping-cough*, it is most likely to arise during the paroxysmal stage, but the liability continues throughout the disease, and long into convalescence. In *diphtheria*, it develops when the obstruction is at its worst; where tracheotomy has been performed, the risk continues for two or three weeks at least after the operation.

The onset is sometimes marked by an attack of laryngismus stridulus, and when laryngismus stridulus proves fatal after the spasm is past, the result is not uncommonly due to the bronchitis and broncho-pneumonia it has led to.

Duration.—The duration is rarely less than a fortnight, and may be very much longer.

Thus in 66·6 per cent.¹ the duration was between seven and twenty-one days, and only in 11·5 per cent. less than seven days, while in 21·9 per cent. the illness lasted for more than twenty-one days.

I have met with several cases of five to eight weeks' duration, and in one or two instances the illness lasted for three months or more.²

As the disease runs its course, the children rapidly lose flesh and strength, become very pallid and flabby, and look extremely ill. The marasmus often lasts long after the fever is past, and may be one of the most troublesome complications during convalescence.

Prognosis and mortality.—The general prognosis is bad, for the rate of mortality is high.

Thus, as the result of many collated statistics, Jürgensen places the average mortality at 48 per cent. and Ziemssen at 52 per cent.

Of 412 cases at St. Bartholomew's Hospital the average mortality was 33·4 per cent.; for male 32·6 per cent. and for female 35·2 per cent.

The general prognosis is greatly modified by age.

Thus under 1 year the mortality is placed at	50 per cent.	Ziemssen.	66 per cent.	Holt.
" 2	"	"	55	} 44 " "
" 3	"	"	33	
" 4	"	"	16	

In most statistics allowance must be made for the sources from which the cases are drawn; for in hospitals the patients consist for the most part of the under-fed weakly children of the poor, and just as the children of the well-to-do are less liable to broncho-pneumonia, so are they also less likely to die from it.

Something also depends upon the sanitary conditions under which the children are placed. Thus the presence of broncho-pneumonia varies greatly in different infant-institutions, just as ophthalmia does, and therefore depends obviously to some extent upon preventible causes.

In all cases alike much depends upon the muscular power of the child, inasmuch as death results more from the failure of the respiratory muscles than of the heart. Thus the prognosis is least favourable where the child is weakly, ill-fed, or badly nourished, and that whether too thin or too fat; if it be the subject of rickets, syphilis, or tubercle; or if it be suffering from some previous disease of the chest.

The prognosis, again, is worse when the broncho-pneumonia develops in the course of some exhausting disease, especially one of the infective fevers, for instance, measles, whooping-cough or diphtheria.

Bartels states that broncho-pneumonia in the course of measles is invariably fatal in children under twelve months of age; and that of the total deaths from measles, 80 per cent. are due to broncho-pneumonia.

Table showing the mortality of broncho-pneumonia in its primary and secondary forms (Holt):

Primary,	49·4 per cent.
Secondary to bronchitis of larger tubes,	65·5 "
" to measles,	62·9 "
" to whooping-cough,	81·8 "
" to ileo-colitis,	94·7 "
" to scarlet fever,	} 100·0 "
" to diphtheria,	
" to varicella,	
" to erysipelas,	
" to influenza,	16·6 "

¹ Holt.

² Cf. case.

In any given case the gravity may be measured by the amount of bronchitis, the extent of collapse of the lung, the muscular strength of the patient, and the degree of cyanosis.

Rapidity of the pulse and respiration is not of the same importance in the child as it is in the adult, but irregularity of either in the later stages is of grave omen, while Cheyne-Stokes breathing, if it develop, is, I believe, invariably fatal.

Marked predominance of nervous symptoms, again, is unfavourable. Their importance, however, varies, according as they ushered in the attack or developed subsequently in the course of it. Thus, fits at the commencement of the attack are of little significance; during the course they are rare, but serious; while at the end they are of grave import, and if associated with cyanosis or with unconsciousness, are usually precursors of death.

It is important to bear in mind, in any bad case of bronchitis or broncho-pneumonia, how rapid the changes in the condition of the patient may be. Improvement in broncho-pneumonia is rarely so sudden or so unexpected as it often is in bronchitis, but changes for the worse may occur with startling rapidity. Yet hope should never be abandoned, for it is astonishing how tenacious of life children are, and the gloomiest prognostications are often falsified by experience.

Other things being equal, the longer the case lasts, the less the chance of recovery, but even in the most protracted cases recovery may ultimately be complete.

Of 231 fatal cases collected by Holt:

25	per cent.	were fatal during the first week.
55.5	„	between the 7th and 21st day.
and 19.5	„	21st and 60th day.

I have seen complete recovery take place after three, four, and even five months' continuous illness.

If no complications arise, convalescence, though it may be greatly protracted, is often in the end complete. Recovery is in all cases slow, and it may be months before the child has regained its former health. During this tedious convalescence the child is ever liable to a recurrence of the disease, and from mere debility is a bad subject for any of the common ailments of children, so that many are carried off by some intercurrent malady.

When once perfectly recovered they may grow up quite strong and enjoy long and active lives.

Diagnosis. — The diagnosis of broncho-pneumonia rarely presents any serious difficulty. The three diseases from which the diagnosis has chiefly to be made are bronchitis, croupous pneumonia, and tuberculosis.

From *bronchitis*, the diagnosis is clear when signs of consolidation are present. When they are not, a sudden aggravation of symptoms and increase of fever mark, as a rule, the onset of broncho-pneumonia. At the time of examination the amount of bronchitis may obviously be insufficient to account for all the symptoms, for it often happens that when broncho-pneumonia sets in the signs of bronchitis become less marked.

From *croupous pneumonia*, the secondary broncho-pneumonia is distinguished by its more gradual onset and termination, by its greater duration and tendency to relapse, by the markedly intermittent character of the fever, and by the irregular distribution of the affected parts. The chief difficulties arise when the consolidation is extensive or there is much collapse. Where the physical signs are not conclusive the course of the case will usually decide its nature.

Tuberculosis of the lung may excite both bronchitis and broncho-pneumonia. The question then is not whether there is consolidation or not, but whether the cause of the lesions is tubercle.

This it may be impossible to determine except by the course of the case. Where the attack is greatly prolonged and there are frequent relapses, the presence of tubercle will always suggest itself.

From *pleuritic effusion*, the diagnosis, if not to be made by the physical signs and by the course of the case, may be assisted by an exploratory puncture with the needle, but this question is not likely to arise except where there is marked dulness at the base behind.

The predominance of nervous symptoms may suggest the diagnosis of *meningitis*, but this difficulty is not so likely to occur as in croupous pneumonia. The diagnosis is usually easily determined by the pulse-respiration ratio; for on the one hand the respirations, though they may be somewhat rapid and irregular in meningitis, rarely present the perverted ratio to the pulse, which is the rule in pneumonia, while on the other hand, except where the case is one of general tuberculosis, the pulmonary symptoms in meningitis are rarely so marked, and may be entirely absent.

Treatment.—Bacteriological investigations show that broncho-pneumonia is most frequently associated with, and caused by, organisms which are extremely common in the mouth.

Thus Marfan showed that as the result of the inoculation of saliva in 127 cases, a pneumococcal infection was obtained in 15·5 per cent., streptococcal in 5·5, and infection with Friedländer's bacillus in 4·5.

This leads to the inference that the source of infection is usually the mouth, and suggests a common-sense prophylactic measure, viz., a careful *mouth-toilet* in all young children, especially when suffering from any epidemic disease like measles, influenza, etc.

Much may be done by proper care and judicious management in a child already suffering from bronchitis to prevent the collapse of the lung, which is the common precursor of broncho-pneumonia. The child should not be allowed to lie long in one position, especially on the back, but be shifted from side to side, or best of all, if an infant, be carefully nursed; for while the child will sleep well on the lap or arm, the frequent changes of position which the fatigue of nursing necessitates are good for it. A crying fit now and then is the best thing possible, for it expands the lungs, and often, by exciting a fit of coughing, gets rid of much secretion from the tubes.

The chest should be freely poulticed or rubbed with some counter-irritating liniment, *e.g.*, Lin. Terebinth. acet.; or a spongiopiline or cotton-wool jacket may be used sprinkled with spirits of camphor.

When broncho-pneumonia has developed the treatment will vary somewhat with the amount of bronchitis present, with the height of the fever, and with the general condition and strength of the patient.

The fundamental principles which underlie the choice of remedies are to avoid depression and to maintain the strength.

The strength must be kept up by feeding, stimulants and tonics.

The food should vary according to the age of the child, but should consist chiefly, if not entirely, of milk, with which may be beaten up the yolk of an egg or two.

Stimulants are generally necessary in bad cases, and are best given in the form of brandy added to the milk, 10 to 30 drops in the hour. If the food

be refused, it must be administered by the nasal tube, the only care requisite in forcible feeding being not to give too much at a time, lest vomiting or diarrhoea be excited.

The bronchitis often diminishes as the broncho-pneumonia develops, but if it continue, it must be treated on the ordinary lines; the child being put in a half-tent, a steam-kettle started and the usual remedies employed.

For medicine, it will be sufficient in ordinary cases to give a draught containing some sweet spirits of nitre and acetate of ammonia, with the addition of some tincture of squill and ipecacuanha wine, or a mixture containing a little carbonate of ammonia, senega and cascarilla. When the fever has subsided a tonic such as the syrup of the phosphate of iron will be found very useful.

If the secretion accumulate in the tube and suffocation threaten, an emetic may be indicated. Of these, ipecacuanha is the favourite, but it often fails in little children. Apomorphia is more reliable, and of this one-fiftieth of a grain may be safely given to an infant of one year old.

With the view of exciting active respiratory movements in such cases, the patients have been plunged into hot and cold water alternately, or, while in a warm bath, douched with cold water, but I do not think such violent measures useful or desirable.

The inhalation of oxygen may be employed with advantage, and under its use the colour often strikingly improves, and the restlessness often subsides, so that the child falls into a quiet sleep—but its administration in such cases must be frequent and continued for some time.

The fever is not often persistently high, and therefore rarely calls for special treatment. If the temperature be high and the skin dry, a hot mustard bath gives great relief. The bath should contain about one ounce of mustard to the gallon and be of about 106° – 110° . The child should be kept in it for 10–15 minutes, and then placed without drying between warm blankets. The result of this will be prolonged and copious sweating, during which the temperature falls and the child goes to sleep.

Antipyretics are rarely necessary, either in the form of drugs or of cold water. Cold or tepid baths and the application of cold compresses, or an ice-bag to the chest, have been advocated, but with less reason than in croupous pneumonia, and their use is limited.

Antipyrin has been given, but it is of little or no use, and has dangers of its own in consequence of the depression it often causes. The same applies to phenacetin and other antipyretics of this class.

Complications must be treated as they arise.

During convalescence, good feeding, change of air, and tonics are indispensable. In many cases no progress seems to be made until the child has been sent away to the country or seaside, and kept there for two or three months. The greatest care is always necessary to avoid colds, for the chest remains delicate for many months, so that the bronchitis is very likely to return, and with it the risk of broncho-pneumonia.

Secondary Broncho-pneumonia in the Adult.

As already stated, broncho-pneumonia as a clinical term is almost now confined by usage to the pneumonia of little children; the patchy, disseminated pneumonia of the adult arises under other conditions and is called by other names.

In the adult, simple bronchitis rarely leads to broncho-pneumonia except when the muscular powers are greatly reduced. It is therefore among the aged, feeble,

and cachectic that the affection is most frequently met with, especially after long confinement to bed. In such cases hypostatic congestion will be present, and this may end in pneumonia either of the croupous or catarrhal form. This group of cases is usually described under the title of hypostatic pneumonia and not of broncho-pneumonia.

When broncho-pneumonia arises under these conditions it is often very insidious in access and marked throughout by no definite and characteristic symptoms; the temperature is but little raised and the pulse and respirations but slightly accelerated. The tongue may be coated or somewhat dry. The patients are clearly ill, but it is not till physical examination has been made that the cause is apparent; and sometimes the lesion is not discovered except on *post-mortem* examination.

In all cases alike the prognosis is bad and the mortality high.

Broncho-pneumonia plays an important role in phthisis, the lesions of tubercle being largely made up of catarrhal pneumonia which undergoes the characteristic caseous degeneration, but the consideration of this form belongs to the subject of phthisis and is dealt with there.

As already described, the subacute and chronic forms of broncho-pneumonia constitute, in part, the lesions found in the lungs in connection with the inhalation of dusts.

The most acute and destructive forms are met with as the result of the passage of infective or irritating substances into the air-tubes.

These various forms are dealt with more fully under their respective headings.

PRIMARY BRONCHO-PNEUMONIA—IDIOPATHIC.

(Broncho-pneumonia without antecedent Bronchitis.)

The two groups of broncho-pneumonia, *i.e.*, the primary and the secondary, differ in a very pronounced way clinically. Thus in the ordinary secondary, broncho-pneumonia which follows bronchitis, the child is—

1. Generally weakly, often rickety, perhaps tubercular, liable to attacks of diarrhoea, and subject to bronchitis, from which it is suffering at the time of onset.
2. The temperature is of a hectic or irregularly remittent type, remains high for many days, and terminates by lysis.
3. Recovery, when it takes place, is slow and tedious, and there is a great tendency to relapse.
4. The mortality is considerable, especially in very young children.
5. The symptoms are severe, but local rather than general; what symptoms there are, depend upon the cyanosis, which is to a greater or less degree always present.
6. The diagnosis has to be made from capillary bronchitis rather than from anything else.

It is in the temperature chart especially that the chief characteristics of the cases are seen.

This is the common form of broncho-pneumonia, upon which the usual clinical and pathological descriptions are based, and of which the four cases following are good examples.

CASE 1.—T. S., 1 year and 5 months old, was in good health until attacked by measles, five weeks before, after which he caught cold and had some diarrhoea. He was admitted with broncho-pneumonia, and a temperature of 104.4° . There were the signs of general bronchitis, with a few small patches of dulness, one at the apex and another near the heart on the left side, and others at both bases behind; there was considerable recession of the lower parts of the chest, so that the dulness at the bases was probably due in great part to collapse. In various parts of the lung there were patches over which small, sharp, crackling crepitations were heard. The child was somewhat cyanosed, and though evidently very ill, was at times bright and intelligent. The temperature was of a distinctly hectic type, oscillating daily between 103° or 104° and 99° . The chart is characteristic, and shows the main features of the case, which continued without much alteration of physical signs or conditions until death occurred, rather more than a fortnight later, when the patient died somewhat suddenly. There was no *post-mortem* examination.

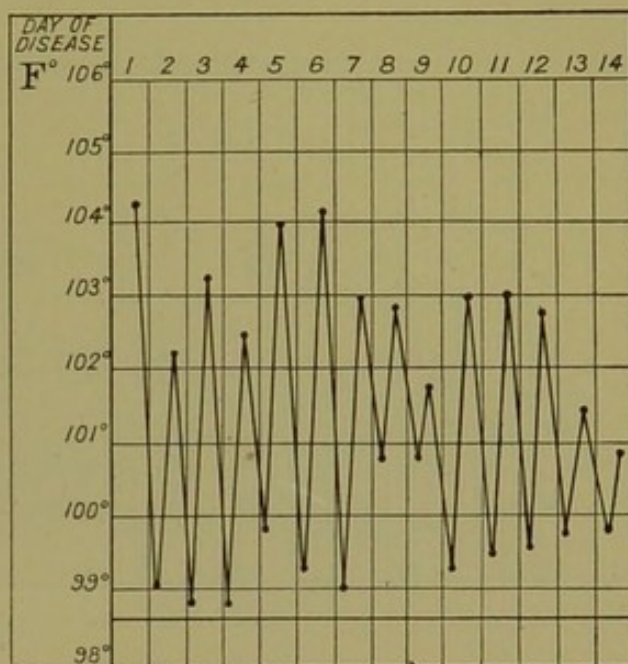


Fig. 86.—Case 1.

CASE 2.—D. R., aged 1 year and 8 months. The child was a healthy baby; had whooping-cough at 12 months of age, from which she completely recovered; and measles seven weeks before, since which time she had never been quite well. Four weeks before admission, that is, three weeks after the commencement of measles, the child developed bronchitis, and it was for this that she was brought to the hospital, having at the time a temperature of 103.6° . There was impairment of percussion more or less over the whole of the right lung, where the breathing was greatly exaggerated, and in places almost bronchial in character; elsewhere on both sides of the chest the ordinary signs of bronchitis were present. The course of the case need not be minutely described. It was a very long one, and was characterised by a succession of relapses, the temperature rising considerably during each relapse, and the physical signs constantly changing; a patch of dulness, of bronchial breathing, or of crepitation appearing first in one part, then in another, the whole case being gradually drawn out for a period of three months. Ultimately the patient completely recovered.

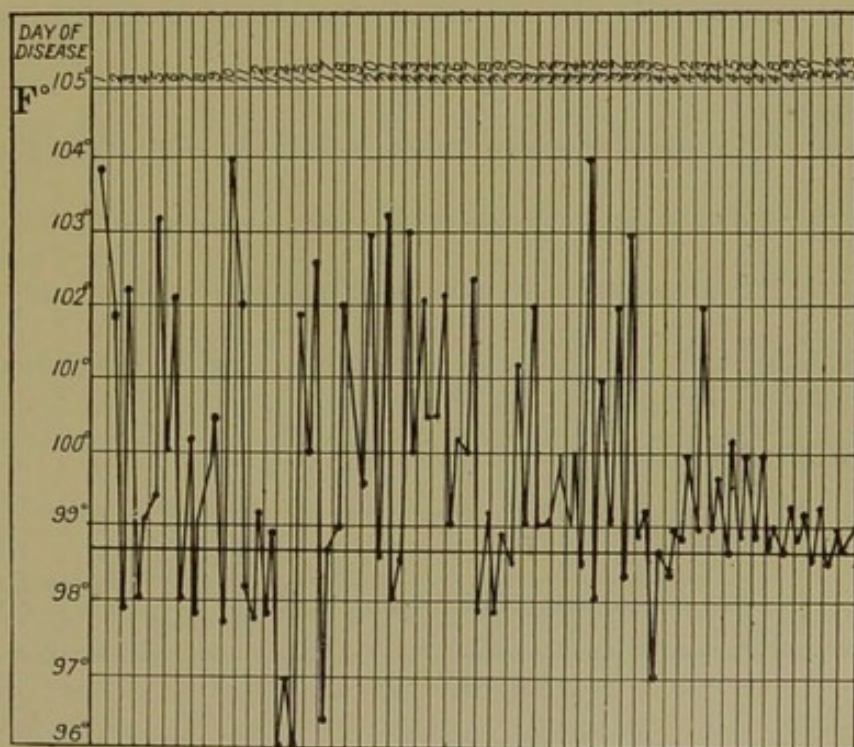


Fig. 87.—Case 2.

CASE 3.—L. C., 2½ years of age, was well until attacked with whooping-cough two months before admission. For the last three weeks the patient had had a good deal of cough and the breathing had been rapid. Two days before

admission the patient had become suddenly worse, and was then admitted with a temperature of 103° and all the signs of broncho-pneumonia. The whole of the right side was impaired to

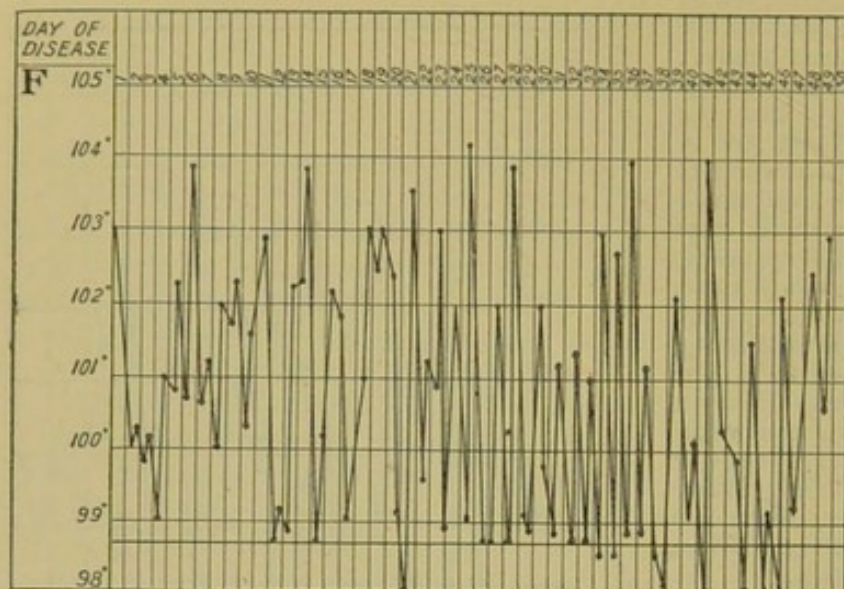


Fig. 88.—Case 3.

a good many fresh patches in the left lung. So the case went on with one relapse following another for a period of three months. In the end the patient recovered completely.

In the next two cases, although the affection proved ultimately to be broncho-pneumonia, the onset was of a more sudden character; but with this exception they did not differ in their clinical course from those preceding.

CASE 4.—A. D., aged 3, was in good health until October 3rd, when after being at school she became restless and feverish and complained of being giddy; she

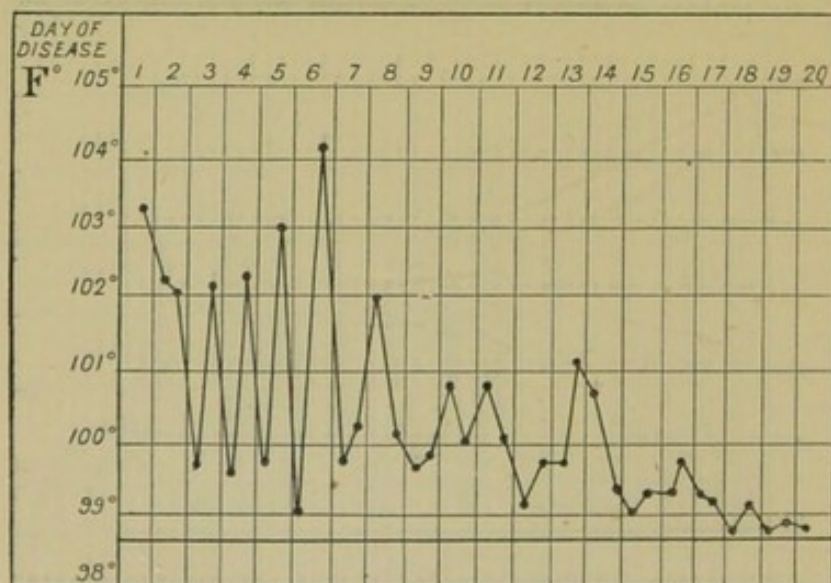


Fig. 89.—Case 4.

then got some bronchitis, with very rapid breathing, and on the 6th was admitted with a temperature of 103.6° , pulse 96, respirations 60. The only physical signs were those of bronchitis, and there was no definite consolidation to be found anywhere. The fever was of a markedly hectic character, varying from 103° to 99.5° daily for about a week; after this it became less hectic for a period of fourteen days longer, and finally subsided: the patient gradually recovered completely.

CASE 5.—D. T., 1 year and 5 months of age, was attacked with whooping-cough on January 1st, having been previously in

good health. The attack proved to be a severe one, so that she would have attacks every hour or so. She vomited a good deal, had frequent epistaxis, and rapidly lost flesh and strength. On January 15th breathing became rapid, and she was found to have bronchitis. By the 20th the symptoms were much worse, and there was marked recession of the lower part of the chest. On January 21st she was admitted, with high temperature, respirations 56, pulse 150, tempera-

ture 101°. The chest was hyper-resonant, except in the right axilla, where there was a considerable patch of dulness, over which the breath sounds were bronchial. Besides this there were signs of general bronchitis over both sides. The next day the temperature rose to 106°, and remained very high for the whole of that day, in spite of frequent sponging. Pulse 180, respirations 76. In the course of the next two days the signs of bronchitis cleared off to a great extent, but the dulness remained the same. The temperature was very high on January 24th: temperature 104°, pulse 200, respirations 80. The condition remained much the same till January 27th, when the temperature went up to 106°, and was reduced by bathing; but it quickly went up again to 104°. That night a crisis occurred, a fall of 10·5° taking place in a few hours; and when the temperature fell to 98°, the pulse was still 148 and the respirations 76. This continued for three or four days, but on January 30th the temperature began again to rise, and continued high until February 14th—that is, eighteen days longer. Signs of bronchitis appeared in the chest again, with some patches of consolidation on the left side as well. The respiration and pulse still remained very rapid, the respirations averaging 40 to 50, and the pulse 160. On February 14th the temperature began to fall, and on the 16th was subnormal, from which time the patient made an uninterrupted recovery, the respirations, however, remaining rapid for another week, and the pulse also.

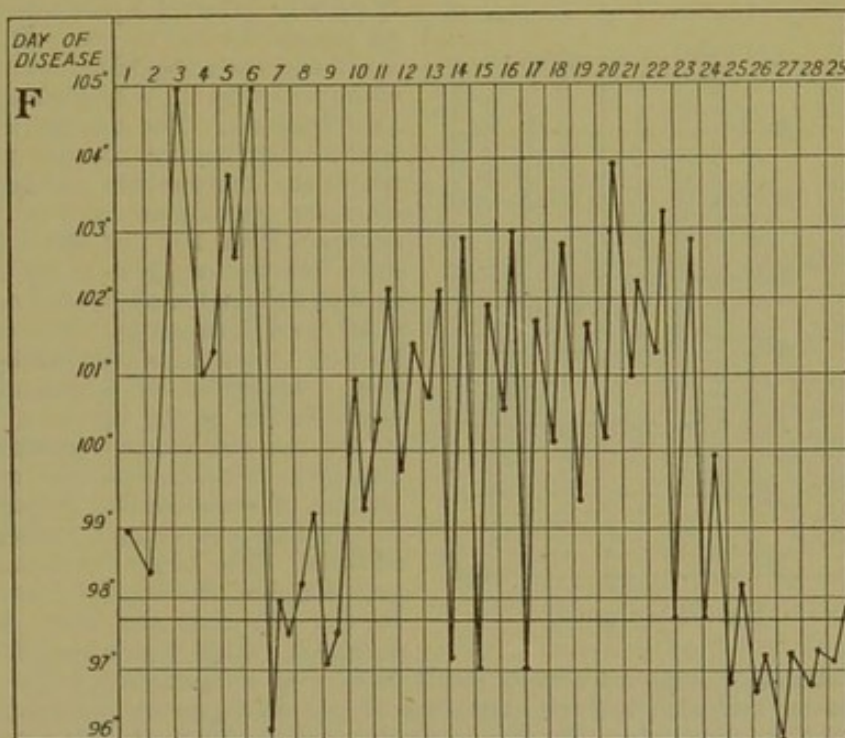


Fig. 90.—Case 5.

The first attack appeared to be broncho-pneumonia, but with considerable consolidation on the right side. The second attack presented the ordinary features of broncho-pneumonia.

Primary broncho-pneumonia stands in strong contrast in almost every respect to secondary broncho-pneumonia.

1. The children are often robust and healthy until suddenly attacked with severe symptoms.

2. The fever commences abruptly and is often ushered in by convulsions or nerve symptoms; it is of shorter duration, and terminates, as it began, suddenly, often by well-marked crisis. The temperature is of a higher range and less remittent type.

3. There is no great tendency to relapse.

4. Recovery is rapid.

5. The mortality is small.

6. There is no antecedent bronchitis, and often no coincident bronchitis; and though, occasionally in the later days of the disease, signs of bronchitis may present themselves, they are obviously secondary.

7. The general symptoms are often much more severe than the local ones, so that the pulmonary affection may be more or less masked.

8. The nervous symptoms may be marked, and the patients be unconscious or delirious, and it is in this group of cases that the difficulties of diagnosis from meningitis most frequently present themselves.

The following cases are examples of this form of broncho-pneumonia.

CASE 6.—H. B., aged 9 months, was a weakly child, and had had one or two slight attacks of bronchitis, from which he had completely recovered. He was in his usual health till March 31st, when he woke up one night crying and in a cold sweat. Until his admission on April 5th he had continued very ill, having a good deal of wheezing on the chest, very rapid breathing and a good deal of fever. On admission the child's temperature was 103° , pulse 200, respirations 70, with a good deal of wheezing over the whole chest, with crepitation in nearly all parts, and extreme recession of the lower part of the neck. The only part where there was any impairment of percussion was at the left base behind, and here the breathing was exaggerated and was almost bronchial. The child was very ill, somewhat cyanosed, and with considerable dyspnoea. The condition remained much the same, without any marked change in the patient, till April 9th, when the temperature suddenly fell, just as it does in croupous pneumonia, and in the course of a few hours dropped from 104° to 98° . This was accompanied with great prostration, from which the child did not rally, but died of exhaustion on the following day. From the sudden onset and termination the case presented the clinical features of croupous pneumonia. The physical signs, however, were those of a general bronchitis, with possibly consolidation at the left base.

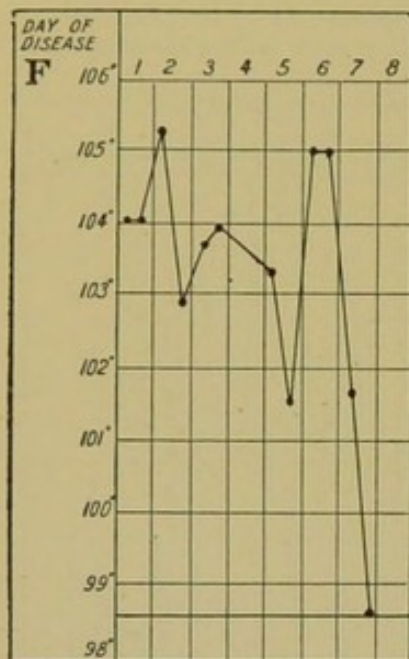


Fig. 91.—Case 6.

contained a large number of small patches of consolidation. The posterior surface of the lung was covered with a thick exudation of lymph, a quarter of an inch thick over the lower part, which extended along the diaphragmatic surface and reached above to the middle of the upper lobe. In this lung there was a good deal of mucopurulent secretion in the smaller bronchial tubes throughout. The pathological lesions found were those of ordinary broncho-pneumonia.

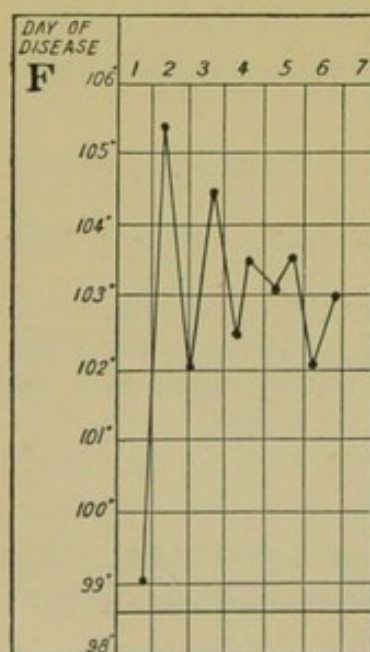


Fig. 92.—Case 7.

CASE 7.—D. D., aged 7 months, was a weakly child, but had been in fair health until February 12th, when it became restless and fretful. It vomited and had some wheezing on the chest, with a good deal of cough. On the 13th it was admitted, and had then a considerable amount of dyspnoea; the temperature was 105° , pulse 50, respirations 60, and there was a good deal of recession of the lower part of the thorax. The physical signs were those of general bronchitis, but in the right middle lobe there was an area of impaired resonance. The child remained in much the same condition until January 20th, when it seemed to be somewhat better, but it died somewhat suddenly that day without any obvious cause. There was no *post-mortem* examination.

CASE 8.—W. T. W., aged $3\frac{1}{2}$ years, had always been a fairly healthy child, except for two attacks of bronchitis during his second year. He was well till December 25th, 1891, when his digestion was upset by some plum pudding. On December 28th he complained of thirst, lost his appetite, was drowsy, and had some cough. On January 1st he became delirious and had a slight fit. On admission he was found to have the signs of some general bronchitis, which, however, rapidly subsided as the signs of consolidation appeared at the right apex, where there were some impairment of percussion and well-marked bronchial breathing. The pulse was 120, the respirations 64. The temperature averaged about 103° , occasionally reaching 104° ; it was hectic in character, but retained a high general level. He remained very ill, but

gave no special cause for anxiety, and on January 7th appeared to be somewhat better in the morning, but in the middle of the day gave a cough, was seized with dyspnoea, and died in a few minutes. There was no *post-mortem* examination.

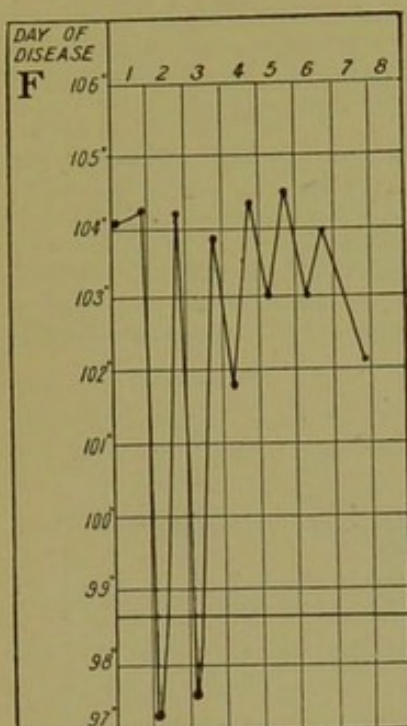


Fig. 93.—Case 8.

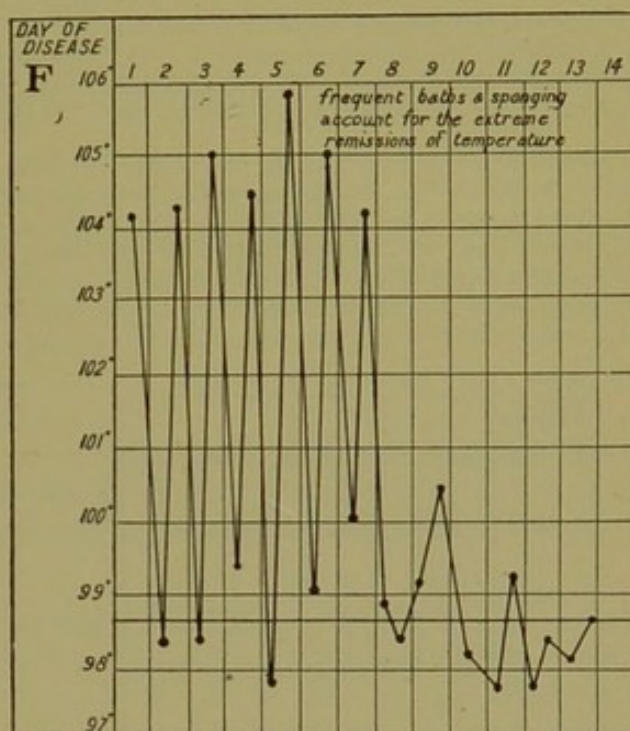


Fig. 94.—Case 9.

CASE 9.—F. R., aged $4\frac{1}{2}$ years, was in good health till October 12th, when he was suddenly seized with shivering and coughing, and on the 13th he was very feverish, and vomited. The respirations were 40, pulse 150; the temperature was so high that the patient had to be bathed and sponged on several occasions. The physical signs were those of broncho-pneumonia, without any very definite local signs of consolidation. The patient was very ill. The fever was very high, but it was intermittent in character, and it terminated suddenly with a well-marked crisis. At the same time a brother, aged 2, was also in the hospital with broncho-pneumonia, after which an empyema developed, and the child died.

To compare with this last series of cases we may take another series, in which, on account of the more marked physical signs, the diagnosis of croupous pneumonia was made.

CASE 10.—E. K., $1\frac{1}{4}$ year old, a healthy and strong child, was well till January 20th, when he suddenly became very restless, had a kind of fit, vomited several times, and his breathing became short. He was admitted into the hospital on January 25th—that is to say, on the sixth day of the disease. His pulse was 150, respirations 60, and his temperature very high. The whole of the lower half of the left lung was almost dull, the resonance greatly impaired, and there was well-marked bronchial breathing. There was nothing remarkable about the case, except the severity of the fever. On the tenth day the temperature fell suddenly by a well-marked crisis. Recovery was then rapid, and in five days all physical signs had disappeared.

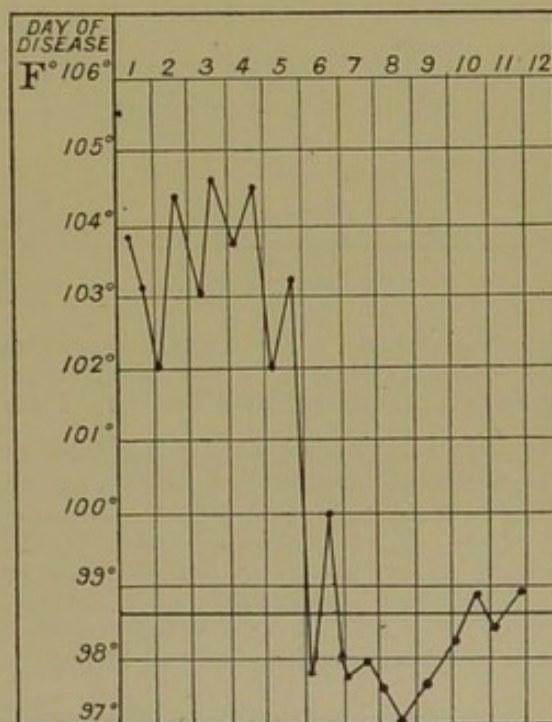


Fig. 95.—Case 10.

CASE 11.—G. K., aged 8, was well till October 5th, when he was suddenly attacked with pain in the right side, frontal headache, vomiting, and pain in the chest. Temperature 103.8° , pulse 130, respirations 72. On October 9th the whole right lung was found to be solid from top to bottom, over which bronchial breathing was audible. There was only one part which maintained its resonance, and that was a small portion in the region of the nipple in front; no crepitation was present anywhere. On the left side the breathing was normal, except that it was somewhat exaggerated and puerile. The temperature fell abruptly on October 10th—that is, on the sixth day of the disease—and the patient made a very rapid recovery.

CASE 12.—F. J., aged 5, and fairly robust, was well until January 30th, when he was suddenly seized with sickness and shivering and seemed very ill. On the 31st he was admitted into the hospital with well-marked signs of left base pneumonia, the whole lower part being dull and presenting well-marked bronchial breathing. The respirations were 160, the pulse 150, the temperature 103.8° . The case presented no particular features, except the extreme rapidity of the pulse, which retained about an average of 160. On February 5th, the seventh day of the disease, the temperature fell suddenly, falling 9° in sixteen hours. The temperature chart is

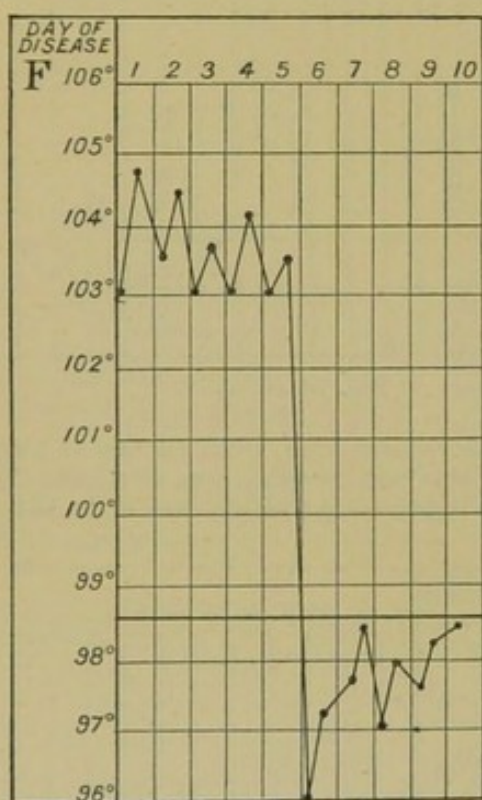


Fig. 96.—Case 12.

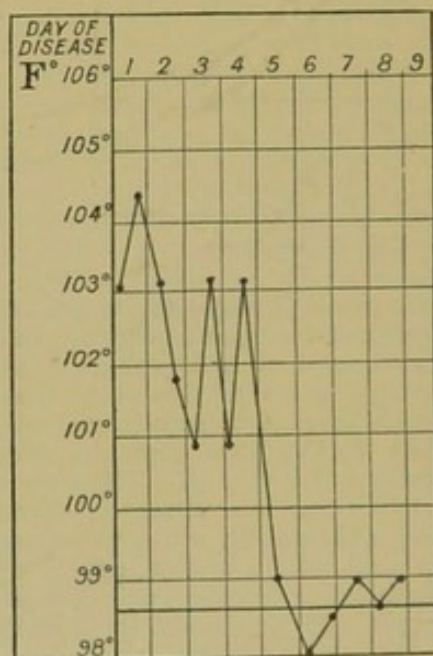


Fig. 97.—Case 13.

very characteristic. The fall in the pulse was almost as remarkable as the fall in the temperature, dropping from about 160 to 60. The child made a rapid recovery, except that it was left with some contraction of the affected side.

CASE 13.—A. C., aged $5\frac{1}{2}$, had been a fairly healthy child, except that it had had two attacks of bronchitis at the ages of $3\frac{1}{2}$ and $4\frac{1}{2}$, but for the last two months it had been a little ailing from some indefinite cause. It was then a little restless at night and was troubled with dyspepsia. A week before admission it suddenly became delirious and vomited, complained of pain which it referred to the abdomen, and had several shivering fits. On admission it was found to have a temperature of 104° , pulse 160, respirations 56; there were marked physical signs of consolidation at the right apex, but no general bronchitis. The case ran its usual course and terminated by crisis on the tenth day. Recovery was rapid and complete.

The cases that I have quoted show that there are three clinical varieties of broncho-pneumonia:

1. That in which the disease is of gradual onset, and preceded by some affection of the air-tubes; the temperature hectic in character; the course prolonged and interrupted by frequent relapses; and the termination by lysis.

2. That in which the history and symptoms are similar, but the disease is of sudden rather than gradual onset.

3. That in which there is no antecedent affection of the air-tubes; the affection is of sudden onset and short duration; the temperature persistently high, without any marked tendency to relapse; the termination is by crisis; the clinical symptoms are those of croupous pneumonia, but the lesions those of broncho-pneumonia.

From the last group, lobar pneumonia differs only in the extent of the consolidation—that is to say, in little more than physical signs.

In other words, the secondary and primary broncho-pneumonias are clinically and pathologically different diseases; and while the secondary stands in close relation with bronchitis and similar affections of the air-tubes, the primary stands in close relation with lobar pneumonia.

Whether there be a real specific difference of this kind between the two forms can only be decided by bacteriological investigation. Unfortunately in most of the investigations made so far, the clinical distinction between these two forms has not been made, so that the results obtained, though of interest and importance in a general way, do not throw much light upon the special question at issue.

The Bacteriology of Broncho-pneumonia.—Our knowledge of the bacteriology of broncho-pneumonia is still very incomplete, though it is rapidly extending. This at any rate must be borne in mind, that, as von Besser has shown, many organisms find their way into the smaller air-tubes of a healthy person without exciting inflammation at all, and, among them, are even virulent pathogenic organisms, such as *staphylococci*, *streptococci*, and *pneumococci*, all of which, with many others as well, are capable of exciting pneumonia both in the lobar and lobular forms.

Speaking generally, broncho-pneumonia is shown to be associated with several forms of pathogenic organisms, *e.g.*, *streptococci*, *staphylococci*, Friedländer's bacillus, the tubercle bacillus, and others, but prominent among them is the pneumococcus, which, taking all cases together, is present, either alone or in association with some of the others named, in about 50 per cent.

The observations of Netter¹ and Marfan² show the presence of

Netter (42 cases).			Marfan (53 cases).	
Pneumococcus	alone	in 10 cases	} 19	{ 40 per cent.
	with others	in 9 "		{ 45.23 "
Streptococcus	alone	in 8 "	} 23	{ 32 "
	with others	in 15 "		{ 54.76 "
Staphylococcus	alone	in 5 "	} 13	{ 20 "
	with others	in 8 "		{ 30.95 "
Friedländer's bacillus	alone	in 2 "	} 6	{ 8 "
	with others	in 4 "		{ 14.28 "

Weichselbaum³ in 15 cases found—

The pneumococcus in 7.
streptococcus in 6.
Friedländer's bacillus in 1.
streptococcus and staphylococcus in 1.

Horton-Smith⁴ in 11 cases found the pneumococcus alone in 5, and associated with other organisms in 3, *i.e.*, in 8 out of 11. In 3 cases only was it absent.

¹ *Arch. d. méd. Exper.*, June 1892.

² In Charcot's *Méd.*, p. 931.

³ *Med. Jahrb.*, 1886.

⁴ *St. Bartholomew's Hospital Report*, 1897. In this paper will be found a complete bibliography of the subject.

Pearce¹ in 46 cases found—

The streptococcus	alone in 16
pneumococcus	„ 12
Staphylococcus pyogenes aureus	„ 6
Staphylococcus pyogenes albus	„ 1
colon bacillus	„ 5
A mixture of organisms	„ 6

When broncho-pneumonia occurs in the course of some infectious disease like measles, diphtheria, etc., the further question will arise whether the inflammation is then excited by the specific organism of the primary disease, or by the common organism, *i.e.*, whether the infection is homologous or heterologous. The question cannot yet be answered, for in most of the common infectious diseases the specific organism has not been isolated; but in a few, *e.g.*, diphtheria, influenza, the infection is often, though not always, homologous.

After typhoid fever the infection appears to be usually heterologous, and so, probably, after scarlet fever. As regards measles and whooping-cough, the question must for the present remain unanswered.

In 62 cases of broncho-pneumonia following diphtheria Pearce² found—

Klebs-Löffler bacillus	in 52.
Staphylococcus pyogenes	in 27.
Staphylococcus pyogenes aureus	in 1.
Staphylococcus pyogenes albus	in 1.
Pneumococcus	in 1.

Kanthack and Horton-Smith make the following classification of the broncho-pneumonias from the bacteriological point of view.

After simple bronchitis—

- (a) Pyococcus { Pneumococcus
Streptococcus
Staphylococcus } or mixed.
- (b) Tubercular.

In the course of infectious diseases—

- | | | |
|-------------------|--|-----------------------------|
| 1. Diphtheria | { Diphtheria bacillus.
Pyococcus.
Diphtheria bacillus and pyococcus. | |
| 2. Influenza | { Influenza bacillus.
Pyococcus.
Mixed influenza bacillus and pyococcus. | |
| 3. Tubercular | { Tubercle bacillus.
Pyococcus.
Tubercle bacillus and pyococcus. | |
| 4. Typhoid | Pyococcus | |
| 5. Scarlet fever | „ | } probably most frequently. |
| 6. Measles | „ | |
| 7. Whooping-cough | „ | |

There are only two sets of observations known to me in which the primary and secondary broncho-pneumonias have been separated, and the results of the bacteriological examination kept distinct.

Mosny³ examined 4 cases of primary broncho-pneumonia and found the pneumococcus in 3 and the streptococcus in 1; of 13 secondary cases the pneumococcus was present alone in 1, and associated with the streptococcus in 1. The streptococcus was found alone in 5, and associated with other bacilli in 5 more. Friedländer's bacillus was present in 1, and an unknown bacillus also in one.

The most complete series is recorded by Holt, as investigated by Dr. Martha Wollstein⁴ under his direction.

¹ *Boston Med. J.*, 1897, ii. 562.

³ *Études sur la broncho-pneumonie*, 1891.

² *Ibid.*

⁴ *Dis. of Childhood*, 1897.

Of 19 primary cases the pneumococcus was found in 17.

Alone in 9.	}
With streptococcus in 7.	
With staphylococcus in 1.	
The streptococcus alone in 1.	
The staphylococcus alone in 1.	

Of 14 secondary cases the pneumococcus was found alone in 2 and with others in 9 = 11.

It was absent only in 3, in all of which the streptococcus was present; alone in 1, associated with the streptococcus in 1, and with the tubercle bacillus in 1.

So far as the bacteriological evidence goes, it seems to show that in secondary broncho-pneumonia the streptococcus is the most frequent organism, and in primary broncho-pneumonia the pneumococcus, and that in the latter the pneumococcus is almost as common as it is in lobar pneumonia of adults.

Thus bacteriology gives confirmation of the conclusion to which clinical investigation has led, namely, that primary broncho-pneumonia is of pneumococcal origin, or, as it may be stated in other words, that the pneumococcus, which in the adult produces lobar consolidation, in the infant and young child produces lobular or disseminated consolidation.

It is now well known that the microscopical distinctions often drawn between the minute lesions of lobar and lobular pneumonia cannot be regarded as absolute, and that, for example, even the fibrin network which is so common in acute lobar pneumonia is not uncommon in acute broncho-pneumonia, while it may be absent in lobar pneumonia.

The conclusions to which this evidence points are obviously these:

1. That the primary and secondary broncho-pneumonias have a different bacteriological origin.

2. That secondary broncho-pneumonia is for the most part due to streptococcus infection, derived from some source connected with the air-tubes, throat, or mouth.

3. That primary broncho-pneumonia is of pneumococcal origin.

4. That pneumococcus inflammation occurs with almost equal frequency in the child and the adult.

5. That pneumococcal inflammation takes a different form in each; in the adult, producing massive consolidation, and in the child, disseminated patches of consolidation; in other words, that there are no real pathogenic distinctions between lobar pneumonia of the adult and primary lobular pneumonia of the child.

It has already been shown that the child's lung has many peculiarities, which seem to give adequate reasons why inflammation of the lung should take a different form in the child from that in the adult.

If these conclusions be correct, it would be best to reserve the term broncho-pneumonia for those affections of the lung which follow antecedent affections of the bronchi, but for the present the two forms of broncho-pneumonia must be dealt with under the one heading, as we are not yet ripe for their clinical separation, though, as I have endeavoured to show, the proper scientific place for the primary or idiopathic form of broncho-pneumonia is not with broncho-pneumonia at all, as it is commonly understood, but with croupous pneumonia.

The division of broncho-pneumonia in the manner which I have suggested certainly removes some of the difficulties which are felt at the bedside and some of the confusion which exists on the subject; and even if it should seem that we may have gone somewhat beyond the facts at present at our disposal in en-

deavouring to connect these varieties with different bacteria, there is no doubt in my mind of the importance of the clinical distinction between the groups to which I have drawn attention.

If, as I have pointed out, the primary and secondary broncho-pneumonias are distinguished from each other not only in clinical history, course, and results, but also in bacteriology, the case for the recognition of these two forms seems to be clearly made out.

The Table here given represents in a diagrammatic form the views I have been expressing.

	Clinical History.	Bacteriology.	
Broncho - pneumonia, lobular, patchy, disseminated.	1. Gradual onset, tendency to relapse. Fever - hectic, markedly remittent, of long duration, with gradual fall.	Streptococcus chiefly and others.	Secondary broncho-pneumonia after bronchitis, measles, whooping-cough, diphtheria, etc.
	2. Gradual onset with sudden aggravation, in other respects the same as above.	Streptococcus, etc., associated probably with pneumococcus.	
	3. Sudden onset, no tendency to relapse. Fever high, not markedly remittent, of short duration, with sudden fall.	Pneumococcus chiefly.	
Croupous pneumonia, lobar massive consolidation.	Similar in all respects to the last.	Pneumococcus chiefly.	Primary broncho-pneumonia. .. Croupous Pneumonia

History.—Leger (1823) is said to have been the first to distinguish between the pneumonia of the adult and that of the child. Earlier writers included these and many similar affections under the common name of peripneumonia notha, spuria, pituitosa; others described them as acute asthma, paralytic asthma, paralysis of the lungs, etc. Laennec himself did not distinguish between pneumonia and broncho-pneumonia, but included the latter under the head of suffocative catarrh.

Gerhardt (1834) wrote a full clinical account of pneumonia in the child before the age of six years, and in the adult. Barthez and Rilliet (1838) extended Gerhardt's account, and to the description given by them little has been added by subsequent writers. They introduced the terms lobar and lobular. The name broncho-pneumonia was invented by Seifert, while Buhl, who described the epithelial changes in the alveoli, suggested the name desquamatic pneumonia.

The discovery of the close relation in which broncho-pneumonia stood to collapse raised the question as to the inflammatory nature of broncho-pneumonia, but this was quickly set at rest by the observations of Legendre and Bailly, Damaschino and others. In Britain Gairdner's name is closely associated with this affection.

Of later years the chief advance that has been made consists in recognising the important part which broncho-pneumonia plays in many other diseases of the lungs, *e.g.*, inhalation-pneumonia and phthisis. At the present time it is the bacteriological side of the subject that is attracting attention.

43. PULMONARY INFARCT—PULMONARY APOPLEXY—EMBOLISM AND THROMBOSIS.

Embolism, infarct and pulmonary apoplexy are not convertible terms, and, however close their relation to each other may be, deserve separate consideration. Infarct is but one form of pulmonary apoplexy, and not the most serious, nor do the ordinary causes of hæmoptysis usually lead to infarct. Infarct, again, is not caused by embolism only, nor does embolism always lead to infarct.

44. PULMONARY INFARCT.

This is the name given to the condition in which a part of the lung is found solid, the consolidation being due to the stuffing full of the air-vesicles with blood.

Infarcts may be of any size; the smaller and most characteristic are as large as a walnut or a small orange. They are pyriform or wedge-shaped, with the base towards the pleura, and they correspond with a group of lobules.

When the lungs are removed from the body the parts around retract, so that the infarct stands out somewhat prominently above the surface. The parts affected are dark purple or nearly black in colour, but they become red on section and exposure to the air. They are sharply defined from the surrounding lung, are firm, airless, and sink in water.

The surface of the section has a granular appearance, resembling that of croupous pneumonia, except in colour, and due to similar causes, viz., the stuffing of the vesicles with solid substance and the retraction of the elastic walls on section.

Microscopical examination shows the vesicles to be filled with coagulated blood. The alveolar epithelium is granular, and in parts detached, but otherwise the lung tissue is not found to be obviously altered. The vessels surrounding the infarct are greatly distended and filled with blood.

Pathological results.

—If the infarct be small and no complication arise in it, the blood effused will in time undergo the usual changes and be removed partly by expectoration and partly by absorption, so that in the end complete restitution to the normal may take place. If the infarct be larger, the vesicles affected may collapse as the blood is absorbed and induration follow, so that a more or less pigmented cicatrix will be left in the site of the infarct.

Under certain conditions the infarcted parts may become the seat of inflammation, and thus pneumonia, abscess or gangrene develop, but these results are accidents, the work of infective organisms, and not necessary part of infarction. Infarcts vary greatly in size. They may not be larger than a pea or small nut, or they may involve a whole lobe or even more than one lobe. They may be single, as most of the large ones are, or multiple; but if multiple they are usually of small size.

Causes.—The two common causes of infarct are embolism of the pulmonary artery and mechanical rupture of small vessels from over-distension. Besides

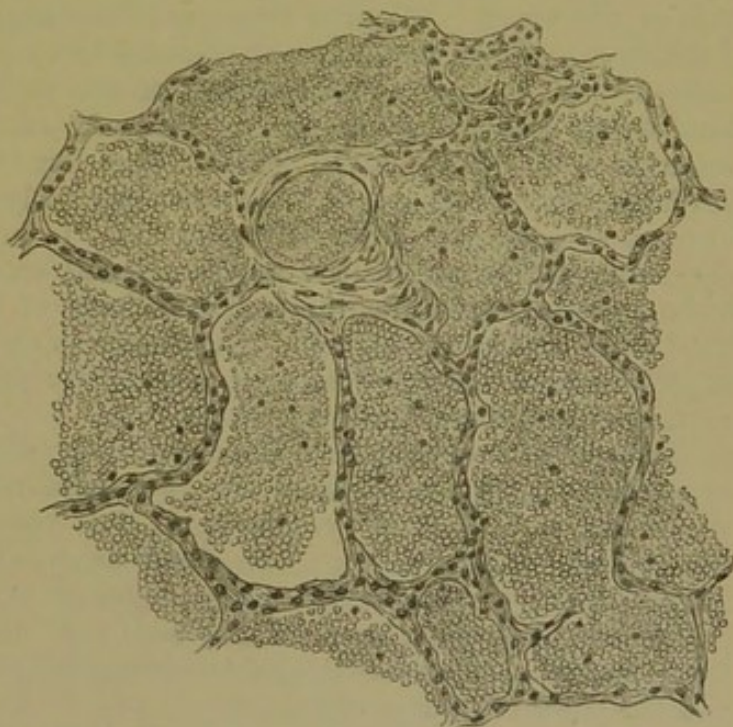


Fig. 98.

Section of hæmorrhagic infarct, showing the vesicles stuffed full of blood.

these may be mentioned as occasional causes, injury, acute inflammation, septic diseases and specific fevers.

Injury.—When the lung is bruised, hæmorrhage takes place into the vesicles and infarction occurs, but it is not so circumscribed, nor is a contusion of the lung commonly called an infarct.

Acute Inflammation, when intense, may be associated with the effusion of much blood (acute hæmorrhagic pneumonia). But on section the affected parts do not show the same dark colour, and microscopical examination establishes easily the nature of the case, if any doubt existed. This condition, again, is not included under the term infarct as ordinarily used.

In some of the *specific fevers* infarcts are not uncommon. They are usually of small size and may be multiple. They often produce no definite signs during life, and may be discovered unexpectedly on *post-mortem* examination. They occur mostly in the more severe or malignant types of fever, especially in scarlet fever, variola, diphtheria, cholera and typhus. They are not necessarily associated with the hæmorrhagic form of fever, though they may occur with it, for in this form the hæmorrhage is more likely to take place from the surface of the bronchi than into the lung vesicles.

In *septic diseases*, again, infarcts are common, and usually pass on into pneumonia, abscess or gangrene.

Many of these cases are no doubt embolic, and derived from clots in the right side of the heart, but in some the obstruction is due to the growth of septic organisms in the capillaries and small vessels, as may be easily demonstrated in anthrax. *Cf.* fig. 76.

a. Mechanical rupture of vessels.—Infarcts in the course of *morbus cordis* are no doubt often due to embolism, the emboli being derived from the right side of the heart. Whether they are all embolic in origin is open to discussion. I think not; for in many no embolus can be demonstrated, and there is reason to believe that the mechanical rupture of the capillaries and small vessels in the lung would sufficiently account for them, an event which we know is of frequent occurrence in the bronchial tubes. This form of infarct is met with in cases of disease of the left side of the heart, especially with mitral affections, but also in cases of weak and failing left ventricle. In brown induration of the lung the enormous dilatation of the capillaries and small veins is the most striking feature in the morbid anatomy, and evidence of their rupture is common both in the tissues and in the alveoli. The small black lumps which are so frequently expectorated in mitral diseases are certainly produced in this way, and so also may be some of the larger infarcts.

Infarcts of this kind from backward congestion may be large or small, single or multiple. The small ones are very common, and frequently recur without producing any definite symptoms, so that patients often complain of spitting these dark lumps without attaching any importance to them. The larger infarcts usually occur in the later stages of mitral disease, when the signs of pulmonary congestion have already been well marked for some time; then often without any obvious cause the breathing may become accelerated and shorter, and the signs of infarct appear.

Sometimes there is a succession of infarcts, and when one is subsiding others are observed to develop.

Alarming as these cases are, the prognosis is not really so grave as it often appears to be.

A woman was under treatment with general œdema and the usual signs of cardiac congestion. She then began without any obvious cause to spit blood and to suffer more from her breathing. Slowly the signs of consolidation appeared at the one base of one lung, and extended until the whole of the lower lobe was involved. This gradually subsided, when a similar condition developed on the opposite side. Infarct was clearly the cause of her trouble. Her condition seemed desperate, but after a few days she began to improve, and in the end all the symptoms disappeared, though she continued to spit up much blood-stained sputum for more than three weeks. In about a month she had recovered and reached an even better state of health than before her illness. She presented at no time any of the sudden symptoms of embolism.

In this and similar cases it seems to me that the explanation which refers the infarct to mechanical rupture rather than to embolism is most consistent with the clinical facts.

b. Embolism.—Although infarcts are often caused by emboli—embolism does not as frequently lead to infarction as seems commonly supposed—embolism of the common pulmonary artery, or of its main trunk or other side, is fatal too soon for infarction to occur, and even in the smaller branches embolism without infarction is not uncommon.

The symptoms and physical signs of infarcts vary greatly according to their size, number, nature, and cause; but chiefly according to the amount of circulation-disturbance they produce, and the suddenness of its onset.

The breathing becomes short and accelerated, and even considerable dyspnoea may develop. There is generally a frequent cough and a varying amount of hæmoptysis. If the infarct be small, the blood will be retained some time in the lung, and when expectorated will be dark in colour. If the infarct be large, the sputum will be more abundant and more blood-stained, and has been compared in appearance to red-currant jelly. It may sometimes look almost like pure blood. As the infarct subsides, the blood expectorated loses its bright colour and passes through the usual changes of brown and yellow to green.

The actual amount of blood lost is not large—a few ounces at most, and anything that could be called copious hæmoptysis is rare.

With an embolic infarct some hours (eight or more, according to the size of the infarct) elapse from the time of the embolism before the sputum becomes blood-stained. With the mechanical infarct blood appears in the sputum much quicker.

Pain in the side is often complained of. It is like the stitch of pleurisy, and is increased by respiration. It is connected with a superficial infarct, and is due to pleurisy over it.¹

The temperature is hardly, if at all, raised. A high temperature, *e.g.*, 103° or more, would be an indication that inflammation had set in.

Small infarcts yield no physical signs, though the seat of them may sometimes be suspected by local signs of congestion. Large infarcts give the signs of consolidation with plugged tubes.

The percussion note will then be impaired and may be dull, but an infarct must have a superficial area of at least two square inches to be detected by percussion.

The voice and breath sounds are often not increased, and may even be absent when the tubes are firmly plugged, so that by physical signs the diagnosis from pleuritic effusion may not be easy.

In the adjacent parts there are generally the signs of bronchitis with abundant crepitation, *i.e.*, of collateral congestion.

An infarct, once started, can be sometimes observed slowly to extend, and so may come to involve a whole lobe or more.

There is a tendency for infarcts to recur, *i.e.*, the condition, under which the first infarct developed, being persistent, may give rise to others, as is so often seen in mitral disease. The prognosis varies greatly in different cases, but apparently desperate cases may not prove fatal, and even end in complete recovery.

¹ Gerhardt, "Hæmorrh., Infarct.," *Volkm. Samml.*, No. 91, p. 9.

45. PULMONARY EMBOLISM.

When an artery is plugged, the circulation through the parts beyond is stopped, and the blood both in the corresponding arteries and veins becomes stationary. Soon the veins become greatly distended, while the capillaries and vessels in the surrounding parts dilate. Owing to the free anastomosis which exists between the small branches of the pulmonary and bronchial vessels the circulation through the affected parts may, if the obstructed artery be not large, become completely restored, and no anatomical lesion result. Under certain circumstances hæmorrhage takes place into the vesicles corresponding with the obstructed artery, and an infarct is produced. The exact source from which the hæmorrhage in these cases comes has been a subject of much dispute.

Cohnheim¹ maintained that the blood came from the distended veins, and was due to backward congestion from the heart, but it has been experimentally shown² that when the pulmonary and bronchial vessels are both tied, and the veins alone left unobstructed, no hæmorrhage occurs. Hence it is concluded that the theory of the venous origin of the bleeding is not true, and recourse is had to the original view, advocated by Virchow,³ that the blood comes from the congested vessels of the surrounding parts; but considering the free anastomosis that has been referred to, it is not unlikely that the bleeding really comes from the vessels of the affected part itself. Whether, in either case, the rupture is due to mechanical causes, or is preceded and determined by a change in the nutrition of the vessels, is not agreed upon, but in many cases in which the vessels have been carefully examined no lesion has been discovered.

The subsequent course which the embolic infarct follows will depend chiefly upon the nature of the embolus itself; if it be simple—*i.e.*, non-infective—the blood may be removed and the parts in time become functionally active again; if it be infective—*i.e.*, septic—pneumonia, abscess or gangrene, as the case may be, will follow. The part which embolism plays in the production of these lesions is considered when treating of metastatic pneumonia, abscess and gangrene of the lung.

Recovery after simple embolism may be incomplete and lead to indurative changes, and thus a puckered pigmented cicatrix be left to mark the site of the lesion.

Panum⁴ observed in some of his experiments with finely divided substances, numerous small indurated patches which bore a close naked-eye resemblance to tubercle.

The emboli are for the most part clots or portions of clot which have been formed either in the right side of the heart or in one of the systemic veins.

Any foreign body which gains access to the vessels will act as an embolus.

Experimentally in animals all sorts of substances have been employed, but naturally in man other causes than clots are very rare. Cancer emboli⁵ have been recorded, and also hydatids and phleboliths.

The clots which constitute the emboli vary greatly in size, from minute fragments only sufficient to plug one of the smallest arteries to masses large enough to completely occlude either the pulmonary artery itself or one of the main branches. The largest masses are derived, as would be expected, either from the heart itself or from one of the larger veins, as, for example, the femoral

¹ *Allgem. Pathol. and Embol. Proc.*

² Litten, *Ztschft. f. klin. Med.*, i. 139.

³ *Ges. Abh.*

⁴ *Virch. Arch.*, xxv. 439.

⁵ Virchow, *Krankh. Geschwülste*; and Paget, *Path. Soc. Trans.*, xxvii.

or internal iliac; the smaller may be derived from the smaller veins, or be fragments from larger clots.

When clots, wherever formed, soften and break down, the embolisms are often recurrent, and as they are infective, and carry with them pathogenic organisms, they excite inflammation, suppuration or gangrene wherever they stick. Many of the forms of acute pyæmia are simply instances of multiple or recurrent infective embolism.

Emboli of cardiac origin.—These are for the most part small, being most frequently minute vegetations detached from the valves or endocardium, but inasmuch as endocarditis of the right side is not common, pulmonary embolism from this cause is rare. The emboli are generally derived from clots of larger size, situated in the right auricle, especially in the appendix or at the apex of the right ventricle between the columnæ carneæ. When the clots here formed disintegrate, numerous emboli result, and, being usually infective, they produce the clinical symptoms of pyæmia.

When the right auricle and ventricle are greatly dilated, as in the course of long-standing obstruction or of gradual failure, clots of much larger size may form. Cardiac clotting of this kind is met with in the later stages of chronic mitral disease or emphysema, towards the end of cancer and other wasting diseases, or in the course of asthenic fevers, such as protracted cases of typhoid fever, and it is by no means an uncommon cause of death after acute pneumonia. Such clots are not uncommon *post-mortem*. Their toughness and want of colour show that they have been formed before death, and have been of long standing. They are generally flattened, and represent the shape of the cavities at the end of systole. They often line the whole ventricle, and extend into the auricle or into the pulmonary artery. These large clots form on both sides of the heart, but are more common on the right side. Parts of them are often broken off, but they may be detached *en masse*.

One case¹ of detachment *en masse* I have recorded, in which the clot formed in the left ventricle, and, being detached, produced an embolism of the aorta itself, with sudden death.

If detached from the right side, sudden death would result from embolism of the pulmonary artery, and this is a fact which should be borne in mind, where embolism of the pulmonary artery has been found without the discovery of any source in the veins from which the clot could be derived.

Emboli of venous origin.—The veins most frequently the source of embolism are the femoral and its branches, or the pelvic. In the femoral the source is usually obvious on account of the œdema of the leg which has preceded the embolism.

Dugnet² describes a case of sudden death with the signs of embolism without any previous signs of thrombosis in the veins, and yet the source of the clot was discovered *post-mortem* in the femoral vein, which had been only partially obstructed, so that no swelling of the leg had occurred.

In the *pelvic veins* the source is often not so clear. After parturition, when so many of these cases occur, the clotting, which is a normal process so long as it is confined to the uterine vessels, may extend from them into the internal iliac or even the vena cava. In this way massive clots are sometimes formed which may yet not be large enough to completely occlude the vessel, and so make their presence evident.

Clotting in the *veins of the arm and neck* is comparatively rare, but owing to the difficulty in keeping these parts still it is especially risky. On the other

¹ *Path. Soc. Trans.*, xxxiv. 62.

² *Union méd.*, 1882, 469.

hand the *veins and sinuses of the skull* are common seats of thrombosis, and especially the jugular sinus in connection with disease of the middle ear and mastoid cells. The clots derived from these last sources are rarely of large size enough to produce definite signs of embolism, and what is met with are disseminated patches of pneumonia or small abscesses, which may yield little if any evidence of their presence during life, and be found unexpectedly after death in cases which have presented themselves simply as pyæmia.

No doubt the abscesses found in the lung after injuries to the external parts or bones of the skull are similarly embolic in origin.

The pulmonary artery has been in some rare cases the seat of the primary thrombosis, the result of some local lesion in it, *e.g.*, atheroma; but this will be more naturally dealt with under the head of thrombosis of the pulmonary vessels.

The **symptoms** of embolism depend upon the size of the embolus, *i.e.*, upon the extent to which the pulmonary circulation is affected. A single minute embolus may lead to no symptoms at all, for the collateral circulation may be amply sufficient to compensate readily for the obstruction at the time and to quickly restore the circulation through the obstructed parts, so that no secondary lesion develops. A large embolus, on the other hand, sufficient to plug the main pulmonary artery, will cause the most urgent symptoms, and may be fatal in a few minutes.

Virchow (*l.c.*) and Paget (*Med. Chir. Tr.*, vols. 27 and 28) were the first to draw especial attention to embolism of the pulmonary artery as a cause of sudden death.

Similar acute symptoms may be produced by minute embolisms, but only when they are in large numbers, and obstruct most of the small vessels or capillaries of the lungs. The best instance of this is fat-embolism.

Emboli of medium size will cause more or less marked symptoms at the time, and commonly end in infarct.

Emboli of any size, if not simple, *i.e.*, if infective, will lead to inflammation, abscess or gangrene, as the case may be, and will be rapidly followed by the signs characteristic of these affections.

Embolism of the pulmonary artery or of one of its main branches.—The clot in this case is of large size, and is generally derived from the heart itself or from one of the large veins, *e.g.*, the femoral or internal iliac. The patient, often without the least warning, is suddenly seized with intense dyspnœa. The respiratory movements are violent and rapid, and the air passes in and out of the chest with much noise, but without obstruction. In spite of this, cyanosis develops and rapidly deepens, the defective aëration of the blood being obviously due not to want of air, but to deficient circulation. The right heart becomes greatly dilated and the veins of the neck distended. The pulse grows more and more weak and irregular, and in a short time the patient dies of suffocation.

Death may be almost instantaneous. The patient, who has been perhaps sitting up, or has moved for some purpose, is seized with what looks like a sudden faint, becomes ghastly pale, gives a few gasps for breath, and is dead. This, which is the result of all the clinical forms of embolism, is evidently due to cardiac or cerebral syncope from the shock.

In other cases the struggle lasts for some hours, and is horrible to witness. The agony is intense, and the face has a look of the greatest horror, the eyes being wide open and protruded, with dilated pupils. An ashy pallor soon succeeds the cyanosis. The respirations and pulse become weaker and more irregular until they cease, sometimes the breathing and sometimes the heart stopping first.

The physical signs in the most acute cases are practically none beyond the noisy respiration and the dilatation of the heart and veins. In the more prolonged cases the lungs become congested, œdematous exudation takes place into the tubes, and large bubbling crepitation is heard over the whole lung, and ends in the coarse tracheal death-rattle.

In the grave cases the consciousness is always obscured, but at first it is probably due to the agony the patients suffer rather than to real unconsciousness. Subsequently, as the cyanosis becomes extreme, the sensorium is dulled by the carbonic acid poisoning. In the end the patients become really unconscious, and at this time, as in other forms of carbonic acid poisoning, may be attacked with convulsions. It is only during this later stage of cyanosis that Cheyne-Stokes breathing may be observed.

When one pulmonary artery only is plugged, the symptoms may be severe at first, but then gradually decrease in intensity as the circulation adjusts itself, and the same is true when smaller branches are obstructed. The embolised lung is often reduced in size; in other words, the distension of the blood-vessels is necessary for the normal full distension of the alveoli.

Embolism of a medium-sized vessel generally leads to infarct, which manifests itself by the physical signs already described.

The sputum, when infarction occurs, is more or less blood-stained; in other cases it may be simply œdematous in character, and in the most acute forms there may be no expectoration at all.

The temperature is not raised at first, and may even be depressed below normal on account of the shock; later it may rise a little, but a high temperature is almost always an indication that inflammation has occurred, though the absence of high temperature does not preclude the existence of inflammatory lesions.

A rigor sometimes occurs at the time of the embolism, and that too with an embolus of simply non-infective nature; but with septic or infectious emboli, rigors are common, and are then usually repeated as fresh embolism occurs.

In valvular disease of the heart, the temperature is often raised without apparent reason. This is much more common with aortic than with mitral disease, but may occur with either. In some cases the explanation is probably to be found in the occurrence of minute embolisms, but in other cases neither the physical signs nor the course of the case suggest this cause or any other.

In all cases the symptoms in the main depend upon the suddenness with which the circulation is interfered with, but the physical signs vary with the duration of the symptoms and require time to develop. In this respect there is a great contrast between embolism and thrombosis, extensive thrombosis often producing less disturbance than a more limited embolism. The same contrast is seen between pneumothorax and pleuritic effusion, and for the same reason, the former being so much more sudden in its development than the latter.

It has been experimentally shown that the gradual obliteration of the pulmonary vessels may be carried on until three-fourths of the entire pulmonary circulation is arrested without causing any serious fall in the carotid tension, or causing death, though the sudden arrest of the circulation in a much smaller number of vessels may be quickly fatal.¹

The prognosis, at the time, varies with the size of the vessel plugged, that is, with the urgency of the symptoms; subsequently, it depends more upon the nature of the embolus and the chances of its recurrence. With small embolisms and infarcts, if they be not numerous or infective, recovery is common and often complete, and even larger embolisms, which have been ushered in with the gravest symptoms, sometimes end in recovery.

¹ Lichtheim, *Störungen d. Lungen Kreislaufs*.

Vawdrey Lush¹ records a case of a woman of 50 who had three severe attacks of embolism and recovered. Humphry² also records a remarkable case of recovery after embolism, in which the patient died later of another affection when the plugged pulmonary artery was found reduced to a fibrous cord.

Minute embolisms, i.e., emboli which are only large enough to plug the capillaries or very small vessels of the lung, produce no symptoms at all unless they are very numerous, and not even then unless they all occur at the same time. This has been often demonstrated experimentally by means of finely-divided substances such as mercury or fine ground charcoal, but the condition rarely arises naturally, and almost the only instance of it is met with in fatty embolism.

Fat-Embolism.—This was first described and studied experimentally by Virchow, who showed that when fluid fat was injected rapidly into the veins the animals died asphyxiated. Zenker in 1862 was the first to describe the condition in man, following a severe crush to the abdomen in which the liver was ruptured. Since then many cases have been recorded, mostly in connection with fracture of bone; others as the result of bruising or crushing of fatty parts, such as the subcutaneous tissues.

Reiter³ collected all the cases recorded up to 1886—211 in number.

Nearly 60 per cent. followed fracture of bones.

14	inflammation of bone (acute and chronic).
11	chronic suppuration of soft parts.
7	injuries of soft parts.
2	lesions of the adipose tissues in the abdomen.
7	the causes were miscellaneous.

The miscellaneous cases include phthisis, diabetes, empyema, and *post-partum* states.

In *post-partum* cases⁴ the fat is, no doubt, derived from the involution changes in the uterus; in empyema from the fatty disintegration in the pus.

Morbid Anatomy.—The lesions found in the lungs are very tiny pin-point infarcts. Large infarcts are rare.

A case is recorded by Eberth⁵ in which three-quarters of the right lung was infarcted and almost all the capillaries were filled with fat, the patient dying with profuse hæmoptysis.

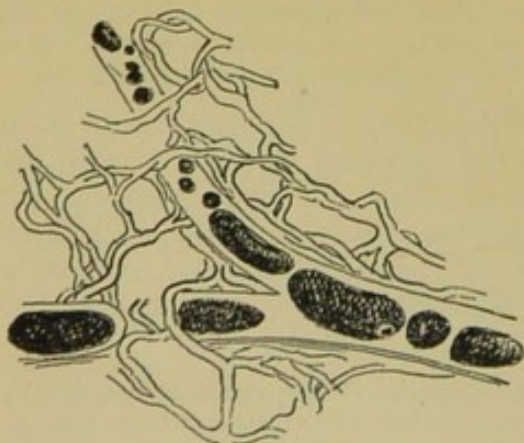


Fig. 99.

Fat embolism of the lung, osmic acid preparation. (Moullin, *Lancet*, 1881, ii. 171.)

and be found in the small vessels of the brain, liver, kidney and spleen.

The rest of the lungs is congested and œdematous, but these secondary changes are only found where the fatty embolism is extensive.

There are no inflammatory changes unless the fat be septic. As no permanent lesion follows fat embolism, it is clear that the fat must be easily got rid of. It is often found in the lymphatics of the lung, and is no doubt partly removed in this way; but it also passes readily, especially if in fine droplets and still more as an emulsion, through the small capillaries, and thus reaches the pulmonary veins, whence it may be carried through the arteries over the whole body,

¹ *Brit. Med. Jour.*, 1880, i. 843; cf. also Bowen, *Liverpool Med. and Chir. Jour.*, 1889, ix. 70.

² Cf. *Lancet*, 1881, i. 16.

³ For history and literature, cf. de Groub , *Rev. de Chir.*, 1895, xv. 577.

⁴ Virchow, *Berl. klin. Woch.*, 1886, No. 30.

⁵ *Fortschr. der Med.*, 1898, xvi. 251.

In the same way it passes readily through the capillaries of the kidney into the renal tubules, and appears as fat in the urine, and forms a fatty pellicle on its surface, of which the familiar condition *post-partum* is an example. Possibly if a large amount of fat passed into the arteries in this way, serious symptoms might be produced by the obstruction to the vessels in the brain or heart. This might even lead to sudden death, and be the explanation of some of the cases of death from so-called "shock."

It is interesting in this connection to note the fact that in some of the fatal cases Cheyne-Stokes breathing has been observed.

Symptoms. — Experimentally it has been shown (1) that if fluid fat be injected very slowly in small quantities at a time no symptoms whatever are produced; (2) that it is only when a large amount of fat is introduced rapidly, so that a large number of the capillaries are obstructed at the same time, that grave symptoms arise, for it is not until about three-quarters of the total pulmonary capillaries are plugged that asphyxia sets in; (3) that if the fat be introduced in the form of a very fine emulsion, as in milk, it passes rapidly through the vessels and produces no capillary obstruction at all.

It is therefore probable that fatty embolism in the lung is much more common and at the same time much less important than is ordinarily supposed, and Moullin's investigations show that, if looked for, the condition may be often found *post-mortem*, when there have been no symptoms to suggest it during life.

The chief symptom is dyspnœa. This is due primarily to the infarcts, and secondarily to the collateral congestion which follows in the rest of the lungs.

In slight cases there may be no dyspnœa at all, or at the most some acceleration of respiration with a little subjective, but no obvious, dyspnœa. On the other hand, the dyspnœa may be marked when there are no physical signs to explain it. Indeed, dyspnœa, which is out of all proportion to the physical signs in the lungs and arises under the appropriate conditions for fatty embolism, may enable the diagnosis in some instances to be correctly made.

When physical signs arise, they are those of congestion, *i.e.*, bronchitis, and are produced by the exudation of serum from the congested vessels into the air-tubes.

Air Embolism.—Sudden death, after the admission of air into the veins, suggests the possibility that the fatal result is due to air embolism, *i.e.*, to the obstruction of the small vessels by air-bubbles, for air-bubbles have been frequently found in them. It is known that the presence of air in fine capillary tubes greatly increases the force required to drive fluids through them, but it has been shown that air is very readily absorbed from the small vessels, and that unless a very large number of them are concerned at the same time no marked symptoms are produced. These facts make it probable that the heart itself takes a very large share in the result, for the right auricle and ventricle are found greatly distended, and the blood in them churned up into a froth, so that the force of the ventricle is probably expended more in compressing the air it contains than in driving the blood onwards.

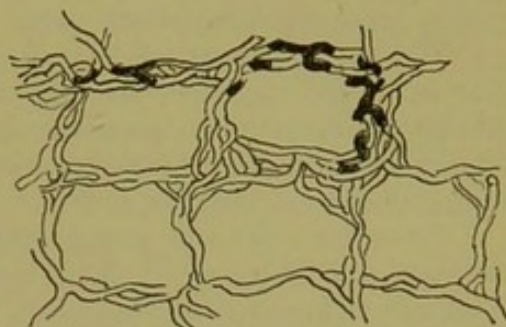


Fig. 100.

Fat embolism in capillaries of the lung.
(Moullin, *Lancet*, 1881, ii. 171.)

The **diagnosis** of embolism, as a rule, presents no difficulties. The sudden access, the symptoms, the physical signs or the want of them, coupled with the history of the case and the frequent presence of clotting in some of the systemic veins, render the diagnosis easy.

The other forms of spasmodic dyspnœa from which pulmonary embolism has to be distinguished are asthma, obstruction to the glottis or main air-tubes, diabetes and uræmic dyspnœa, cardiac dyspnœa, and pulmonary thrombosis.

From asthma and laryngeal obstruction the different character of the respiration suffices for the diagnosis.

From diabetic and uræmic dyspnœa the diagnosis would not be altogether so easy, but there is in these cases little or no cyanosis, and the clinical state suggests rather a nervous than a thoracic origin.

From cardiac dyspnœa and pulmonary thrombosis embolism would be distinguished by the suddenness of its onset, and the difficulty would be much greater to diagnose these two conditions from each other than to distinguish either of them from embolism.

Clinical forms.—The sudden onset is characteristic of all forms of embolism alike; the different clinical forms depend upon the subsequent course.

In the first and most striking form death is sudden, sometimes in a few minutes, and so rapid that no diagnosis is possible; more often intense dyspnœa betrays the cause. In the former case, the patient dies of shock; in the latter, of acute suffocation. Sudden death is, however, not as frequent with embolism as has been supposed, and even in the most acute cases life is, as a rule, prolonged for a few hours, but the dyspnœa is intense and the suffering extreme.

In a second form the symptoms, though of sudden onset, may not be very acute at first, but become so by degrees, and the patient dies with gradually increasing suffocation. This is generally to be explained as the result of extension of the clot by fresh thrombosis round the original embolus.

In another form the symptoms, though very severe at first, gradually diminish in severity and recovery takes place.

Lastly, there is the form in which the embolisms recur, it may be, many times.

Treatment.—Pulmonary embolism offers little scope for treatment. The best of all is its prevention. For this reason the greatest care must be exercised where clots are known to exist, lest by manipulation or by movement the clot should become dislodged. Thus with thrombosis of the femoral the vein and leg should be very cautiously examined, and the patient warned against moving or attempting to walk, and especially against any sudden effort. The same holds good after parturition, for in many of these cases it has been some exertion or sudden movement that has determined the dislodging of the clot.

At the time of the attack, when the symptoms are urgent and grave, the chief dangers lie in the shock produced; in the risk of paralysis of the right side of the heart from over-distension, or of the heart as a whole; and lastly, from suffocation.

Diffusible stimulants are most useful, and if the patient be unable to swallow on account of the dyspnœa, they may be administered *sub cutem* or *per rectum*. A little opium is often of great benefit, to quiet the excitement and alarm which add so much to the distress; but opium cannot be wisely given if there is much cyanosis, and the need for it is past if the patient's sensibility be already dulled by the carbonic acid poisoning.

Where the right heart is greatly distended, and the cyanosis extreme and rapidly developing, there is no more rational way of giving relief than by free venesection. Unfortunately the result is often disappointing, even where the

treatment has seemed most clearly indicated; still, I think it may be said that, where bleeding has failed, nothing else would have succeeded.

If life be prolonged, everything possible must be done to keep up the strength and maintain the action of the heart, for the longer the patient can be kept alive, the greater the chance there is that the circulation may adjust itself and recovery take place.

With this object the patient must be carefully fed and stimulants given. Strychnia, strophanthus or caffein are useful remedies, but digitalis must be employed with caution. Benefit is often derived from the application, to the præcordium, of mustard poultices or other counter-irritants.

46. PULMONARY THROMBOSIS.

Spontaneous thrombosis may occur in either the pulmonary arteries or veins, but rarely primarily in either.

In the pulmonary, as in other arteries, the determining cause will almost certainly be some local disease in the vessel itself. In most cases the vessel has become involved by extension to it of some inflammatory or other disease near it—but in some rare cases the disease originates primarily in the vessel itself.

Thus Romberg¹ describes a remarkable case in which most extensive atheromatous disease was found widely spread throughout the branches of the pulmonary artery, although the main trunk and pulmonary valves were free. Great stenosis was produced, and the patient died with much dyspnoea and cyanosis.

The clots have almost always formed originally in the heart, have spread thence by direct extension into the vessels from the right side onwards into the arteries, and from the left backwards into the veins. Instances of this are met with most frequently in cases which have been long a-dying, as in phthisis, cancer, or even typhoid fever. Such clots, though they may extend into almost every branch of the artery or vein, are rarely large enough to occlude any of the vessels completely, and they rarely produce any distinctive symptoms by which their presence could be diagnosed during life.

Among the acute diseases, there seems to be in *pneumonia* a special liability to cardiac clotting, and sometimes the clots extend as described into the vessels. But besides this, there may be in *pneumonia* a local thrombosis arising in the vessels of the inflamed part, as there may be also in other inflammations, *e.g.*, abscess or gangrene.

Local thrombosis may also result from compression of the vessels from without, as in cases where a new growth surrounds them.

From a local thrombosis, however started, the clot may extend towards the heart, but, as a rule, the extension is in the other direction, from the heart to the vessels.

The clots described have the usual characters of *ante-mortem* clots, being firm and decolorised, and are easily distinguished from the soft gelatinous purple clots of *post-mortem* formation.

Wilson Fox² gives a review of the literature of pulmonary thrombosis, and the great majority of the cases he refers to are instances of the extension of clotting from the heart.

Newton Pitt,³ from an examination of the *post-mortem* records of Guy's Hospital, concludes that thrombosis of the pulmonary vessels is not rare, indeed that it is commoner than embolism,

¹ *D. A. f. klin. Med.*, xlv., Pt. 1 and 2.

² Appendix to Art. on Embolism in *Dis. of Lungs and Pl.*, p. 223.

³ *Path. Soc. Trans.*, xlv. 48.

but he does not appear to distinguish very clearly between the clot which has extended from the heart, and that which has originated *in situ*. He usually lays emphasis on the fact to which I have already drawn attention, that embolism often occurs without infarction.

Primary thrombosis is a very rare affection. Most of the cases recorded under this name are evidently instances of embolism, for the distinction between embolism and thrombosis has not been always strictly observed, especially by the earlier writers, and it is not always easy to distinguish between the original embolus and the recent clot which forms around and beyond it.

The exciting cause is generally to be found in some local lesion of the vessels, as, for example, atheroma, calcification, syphilis, or in the extension to the vessel of some mischief from without, either inflammatory or of the nature of new-growth.

Thrombosis does not, however, necessarily follow even when a new growth penetrates the vein, but in this there is nothing peculiar to the lung.

Wilks¹ describes a case in which the clot was caused by disease of the valves of the pulmonary artery.

In Penzoldt's² case the exciting cause was found in endocarditis of the pulmonary artery.

Turner³ records three similar cases. He also describes in the same volume an interesting case in which the pulmonary artery was affected with tubercle, although there was no thrombosis. Weigert⁴ records a similar observation in the pulmonary artery, and Nugge⁵ one in the pulmonary vein.

Kingston Fowler⁶ gives a remarkable case of thrombosis of the main trunk from the valves to the bifurcation, caused by atheroma in the vessels. It seemed possible that the condition might have existed for five years before death.

Symptoms.—In most instances the lesion has been discovered only on *post-mortem* examination, and has not been diagnosed during life. The local thrombosis in pneumonia, abscess, gangrene or tumour is of little more than pathological interest. The secondary thrombosis extending from the heart is preceded by, and associated with, the signs of cardiac failure and pulmonary congestion, but it produces no definite symptoms by which it can be distinguished from these conditions. Primary thrombosis would be attended with the same signs as those of embolism, except that the onset would be gradual and not sudden. The diagnosis during life must obviously be one of the greatest difficulty.

I have seen one case of the kind in which I ventured to hazard the diagnosis of primary thrombosis, and it proved to be correct upon *post-mortem* examination. The case was very peculiar, and it is the only one of the kind I have ever seen which I was able to diagnose.

Treatment can only be symptomatic and must be directed towards supporting the strength and maintaining the action of the heart.

47. ABSCESS OF THE LUNG.

Abscess of the Lung is an indefinite affection, for the term abscess is not used with the same precision when applied to the lung as it is when applied to other parts of the body. Closed cavities containing pus are very rare in the lung, and generally prove to be pre-existing cysts, *e.g.*, hydatids, which have suppurated.

In the rare cases of lymphangitis pulmonalis, or acute interstitial pneumonia, the lymphatics, being distended with pus, present themselves as abscesses; and in extreme forms, when the pleura has been dissected off for some distance from the lung, the abscess may be of considerable size. Clinically such cases present themselves as acute pneumonia and not as abscess at all.

¹ *Path. Soc. Trans.*, viii. 121.

³ *Path. Soc. Trans.*, xxxvii. 134.

⁵ *Ibid.*, lxxvi.

² *D. Arch. f. klin. Med.*, xii. p. 26.

⁴ *Virch. Arch.*, lxxvii.

⁶ *Dis. of Lungs*, p. 511.

Most so-called abscesses of the lung are either acute suppurating pneumonias or cavities left behind by such suppuration; and they are often described as acute or chronic abscesses respectively.

Acute Abscess of the lung is met with, in its commonest as well as its most characteristic form, as the result of septic embolism. Round the embolus, a patch of pneumonia develops which proceeds to suppurate; the lung tissue becomes also involved, and either suppurates too or sloughs. In this way a portion of the lung is destroyed and a cavity produced. The walls are at first ill-defined, and consist of the remains of the inflamed lung tissue, which projects as ragged tags into the interior. Gradually a line of demarcation is formed, the dead portions of the lung are separated, and a smooth-walled cavity results, lined with a pyogenic membrane. As time goes on, more or less of fibrous tissue develops round it, and so a chronic cavity is produced with thick fibrous walls and a smooth pus-secreting lining. This is commonly described as a chronic abscess.

In the early stage there will be clinical evidence of nothing more than acute pneumonia, and the presence of abscess is often only a matter of conjecture or suspicion. The diagnosis can only be made certain by the discovery in the sputum of fragments of lung tissue.

In the later stage, if the cavity be large enough and near enough to the surface, the signs will be those of excavation; the difficulty will then be to diagnose such chronic cavities from the similar cavities produced by bronchiectasis or by chronic tuberculosis, and except by observation, or the history of the case, this will in most cases be impossible.

There are thus two forms of affection, both described by the name of abscess—

- (1) Localised acute suppurating pneumonias, of which the embolic or septic may be taken as the type.
- (2) The chronic cavities left by this or other suppuration.

There still remains a third group, often inaccurately spoken of as abscess, viz., that in which the suppurating cavity has been formed in the lung by the spreading of suppuration into it from the neighbourhood, *e.g.*, from the pleura, liver, abdomen and mediastinum.

Of the last group little need be said except that it does not at all follow that an abscess in the neighbourhood will, even if it burst through the lung, cause suppuration in it; on the contrary, it is remarkable for how long a time the pus may be expectorated without any inflammation of the lung resulting.

Abscesses following acute inflammation of the lung.—No sharp line can be drawn between the diffuse suppuration which is called purulent infiltration, or the purulent stage of pneumonia, and the local suppuration that ends in abscess. In all cases alike the suppuration is excited by specific organisms, *e.g.*, staphylococcus, pyogenes aureus or albus, etc. If this be so, it is likely that suppuration will be excited whenever the organisms of suppuration find access to the lung, and especially if there be a patch of acute inflammation already there. The organisms may reach the lung either by the blood-vessels or by the air-tubes. In the former case they are usually derived from some focus of suppuration already existing elsewhere in the body and carried into the lungs by embolisms. The parts around the embolus, whether it be large enough to cause an actual infarct or not, become inflamed, and suppuration follows. These cases present themselves as septic or embolic pneumonia, and the abscesses, being of small size, may give no evidence of their existence during life, so that their presence is usually a matter of conjecture only. They are often multiple, and then death is the invariable result. When, as in croupous pneumonia, the lung is already inflamed, and the organisms of suppuration gain access to the inflamed part, the

same changes are excited, and the consolidation either suppurates as a whole or in one or more parts. When suppuration takes place in pneumonia it is nearly always fatal—the diffuse, I believe, invariably, and the localised generally.

When foreign bodies gain access to the air-tubes, and so reach the lung, the changes they excite depend greatly upon their nature and septicity. Clean, inert substances often lie long in the lung without producing any lesion, or, at any rate, anything more than is the more or less mechanical result of their presence, viz., collapse or perhaps bronchiectasis. If, however, the substance be infective—as, for instance, particles of food or septic discharges from the mouth, pharynx, or air-tubes themselves—violent inflammation is at once excited, and this may take on an acutely suppurative or even a gangrenous character. Such cases would present the clinical features of acute septic pneumonia, and after death acute abscesses would be found. In the few cases in which life is maintained a chronic cavity or abscess is the result.

One other class of case remains for consideration, viz., that in which suppurating cavities occur in connection with a tumour or aneurysm in the mediastinum or with a new growth in the lung itself. In these cases parts of the lung are found which look like abscesses on section, and in which portions of the lung are found in a state of necrosis or gangrene bathed in pus. Where cancer is present such cavities have been often described as secondary nodules undergoing softening, but the absence of new growth in them shows them to be not of that nature, and they are really cavities of the kind described. They are correctly referred to the effects of pressure either upon the air-tubes or the vessels, a sub-acute pneumonia having developed and subsequently breaking down or suppurating. The process is almost always a chronic, or at any rate a sub-acute, one, and produces no definite symptoms, but it may explain the rise of temperature which is sometimes observed in cases of malignant intrathoracic disease, and for which no obvious explanation is forthcoming during life. The affection is of quite subordinate importance, as compared with the primary disease, that it is rarely recognised during life, and is discovered only on examination after death.

Chronic Abscess.—It is the group of cases described under this name that is responsible for much of the confusion that prevails upon the subject. The pathological condition implied is definite enough, viz., a cavity with thickened walls lined with a pus-secreting membrane, and as a rule communicating with the exterior through the bronchi. If every cavity of this kind were called an abscess there would be no confusion, but similar cavities result from bronchiectasis and from chronic tuberculosis which are not styled abscess, so that the term abscess is often used to imply that the cavity had its origin in some presumably inflammatory affection, and not in either of the two common causes mentioned. Unless, however, observation or the history of the case prove this, there is nothing, as a rule, when the chronic cavity stage is reached, by which the origin of the cavity can be determined.

Physical signs and symptoms.—From what has been already said it is evident that the signs and symptoms, as well as the prognosis of abscess of the lung, will vary greatly in different cases.

Acute abscess presents itself with the symptoms of acute inflammation of the lung, and cannot be diagnosed at first from one of the forms of acute pneumonia, though the occurrence of suppuration or abscess may be suspected if the pneumonia be embolic or septic. Suppuration in the lung gives rise to the same general symptoms as suppuration elsewhere, viz., hectic fever, sweatings, and perhaps occasional shiverings; but hectic or continued fever after pneumonia does not necessarily mean suppuration or abscess in the lung, for it may be due

simply to delayed resolution or to empyema. The diagnosis of abscess can only be made with certainty when in the purulent discharge fragments of lung tissue are found, or the signs of excavation develop in the places where consolidation only has hitherto existed; and even then the possibility of the consolidation having been an acute caseous pneumonia must not be lost sight of. In such a case the presence of the tubercle bacillus in the sputum generally makes the diagnosis easy.

The sudden discharge of pus—the so-called bursting of the abscess—is more commonly met with where a collection of matter outside the lung—as for example in the pleura, liver, or mediastinum—has burst through the lung, than in abscess of the lung itself. The account given of the development of abscess shows that in most cases the abscess is from the commencement in connection with the exterior through the bronchi. When, as sometimes happens in abscess of the lung, a sudden discharge of pus occurs, it is not due to a vent being formed for the first time, but in most cases to the enlargement of one which has already existed. The vent was either not large enough, or had become plugged by some of the sloughing or broken tissue, and in the gush of discharge such sloughs are often found. It is, however, just these cases that are most striking and most easy to diagnose, for with the sudden discharge of pus the signs of cavity make their appearance.

With large chronic cavities the discharge often occurs in gushes once or twice a day, or at irregular intervals—that is to say, it is not until the secretion in the cavity has reached a certain point that it is evacuated. These large cavities are not themselves sensitive, and it is not until the secretion flows out into the bronchi that coughing is excited; each cough expels a fresh quantity into the tubes, and so the cough continues until the whole cavity is emptied, after which there is rest until it has filled again. The recurrence of the discharge will therefore depend upon the rate of secretion and upon the size of the cavity. In such cases the physical signs vary greatly according to the amount of fluid the cavity contains at the time.

Prognosis.—The acute abscess is very often fatal, indeed, almost invariably so when multiple. If life be prolonged the abscess passes into the chronic stage. If the cavity be small it may gradually contract, and in the end become completely obliterated, leaving nothing behind it but a puckered scar. Such a result is very rare. What usually happens is that the contraction goes on up to a certain point and then stops. The amount of pus secreted becomes less and less, and may almost cease, giving rise to what is called a dry cavity.

Such chronic cavities may continue for years without symptoms and without effect upon the health, but they carry with them risks of their own. The special risks are—(1) putrefaction of the contents, and (2) hæmoptysis.

Putrid change in the contents may make the patient's life a burden by reason of the fœtor exhaled, or affect the health by absorption of the products of decomposition. Again, it may excite inflammation in the walls or round them, and thus lead to acute pneumonia of a grave kind, for such inflammations are often of a very destructive character, and, if not fatal, lead to a rapid extension of the cavity with all its fresh dangers.

Besides these septic risks there is another of a different kind as great and perhaps even greater, viz., that of hæmorrhage. Chronic cavities always have contiguous to their walls vessels of fairly large calibre, and these are liable to become involved. If ulceration be started in the walls of the cavity, it may reach a vessel, erode it, and cause its rupture, or, what is perhaps more common without ulceration, the wall of the vessel on the side towards the cavity may yield, and an aneurysm form, projecting into the cavity. In either way a serious and not

unfrequently fatal hæmoptysis may occur, and that with little or no warning. Indeed, the commonest cause of fatal hæmoptysis is not phthisis in its ordinary sense, but chronic cavities of this kind, whatever their origin be, whether tubercular or not.

If the chronic cavity be large and the discharge of pus considerable, the same results may follow, as in cases of long-continued suppuration, from any other cause, *e.g.*, exhaustion, and ultimately death from asthenia or from amyloid disease.

Treatment.—In most cases abscess forms in the lung before it is suspected or recognised. When once it has developed the hope is that the suppuration will localise itself, and the abscess pass into the chronic stage. All that can be done by treatment is to assist nature by placing the patient under the most favourable sanitary conditions, and by maintaining the strength with good food and tonics. When the abscess has become chronic the object will be to prevent putrefactive changes in the secretions, and with this view residence in the pure air of the country is most desirable. Unfortunately, nothing can be done to diminish the risk of hæmoptysis, which must remain an ever-present danger.

If the secretion has become fetid the usual antiseptic inhalations may be used, or if the cavity be within reach and its exact seat can be diagnosed, direct injections into it may be tried. These methods of treatment, as well as the more direct surgical procedures of incision and drainage, will be further discussed when the general subject of chronic excavation is under consideration.

48. GANGRENE OF THE LUNG.

Between gangrene and sloughing suppuration no definite line can be drawn; the essential distinction is, that the putrefactive organisms are present in the former which cause the characteristic fœtor.

The organisms present are very various, both aerobic and anaërobic. Thus in 12 cases of pulmonary gangrene, Guillemot¹ found, besides several anaërobic bacteria, upon which the putrefactive changes probably depend, the following aerobic bacteria in varying frequency:—*Streptococcus*, 10 times; *Bacterium coli*, 5 times; *Staphylococcus* and *Proteus vulgaris*, twice each, and undetermined bacilli, 3 times.

All the causes that lead to suppuration may also lead to gangrene. When gangrene occurs fœtor is present; but fœtor may occur without gangrene, for the putrid decomposition, to which it is due, may be produced in secretions lying within the bronchial tubes, or in chronic cavities, as in putrid bronchitis or bronchiectasis, and other forms of chronic excavation.

Gangrene, like suppuration, may occur in an acute or chronic form, and be diffuse or circumscribed.

The putrefactive decomposition introduces two new conditions—(1) An increased tendency to the spreading of inflammation, as is best seen in the walls and tissues surrounding putrid cavities; and (2) constitutional disturbance due to the absorption of the products of putrefaction. The latter shows itself in profound constitutional depression, so that asthenia is more marked with gangrene than it is even with septic suppuration which is not gangrenous.

Gangrene is rarely idiopathic, *i.e.*, it rarely arises primarily or independently in the lung, but is nearly always either the result of a secondary infection of some lesion already existent in the lung, for instance, pneumonia, or else it

¹ *Gangrene pulmonaire*, Paris, 1899.

is excited by an infective embolus brought from some part of the body which is already in a condition of gangrenous inflammation.

Morbid Anatomy.—The morbid appearances differ according to the stage and form of the gangrene. Laennec described three stages—(1) That of recent mortification; (2) that of deliquescent sphacelus; and (3) that of excavation and abscess, and this division of the subject cannot be improved on.

In *the first stage* the affected part is diffuent, in the condition, as Dittrich named it, of fœtid œdema. It has a dirty brown or greenish colour, and the sloughing tissue looks like shreds of tea soaked in dirty fluid. The colour is due to altered blood, and is of a dull reddish brown in the recent state, and black or greenish some hours after death.

When gangrene occurs liquefaction always takes place, even in previously consolidated lung, so that the parts become flabby and soft, and if they lie near the surface are sunken and flaccid. Diffuse or spreading gangrene is usually rapidly fatal, and the lung is then found in the condition described.

If the process cease to spread and become localised, it is associated with more or less suppuration. The gangrenous part becomes surrounded with pneumonic consolidation, and a line of demarcation is formed, the condition thus passing into *the second stage*, viz., that of a deliquescent sphacelus. In this stage suppuration is active, an abscess is produced, the secretion still continuing fœtid, and the walls being ragged and rough from the remnants of the lung tissue, which has not yet been separated. After a time the dead tissues are completely detached either in fragments or *en masse*, and lie loose in the cavity. Large sphaceli are rare; the largest which Lebert had met with was not bigger than a pullet's egg. The fragments slowly disintegrate and are expelled, after which the fœtor may cease and the cavity become that of an ordinary abscess, but on the other hand, it may continue to discharge fœtid pus long after all the sloughs have been got rid of.

In the end the cavity, like other chronic cavities, may contract to some extent, but only when it is very small or very favourably situated can it become entirely obliterated. When the chronic cavity stage is reached, the future history will be the same as that of chronic cavities of any other origin. As would be expected, such serious local lesions are likely to be associated with other grave changes.

The pleura is always involved, if the inflammation reach the surface. It is covered with fibrinous or fibrino-purulent exudation, and when effusion follows, as it often does, it will probably take the form of fœtid empyema. The pleura may also take part itself in the gangrene and slough, thus opening the pleural cavity and leading to pneumothorax. In other cases where the pleura is adherent, perforation may take place externally with the formation of an external abscess. This may then contain air, and so cutaneous emphysema may develop and spread over a wide extent of the body.

A case¹ is recorded in which, when such an abscess was opened, the portion of gangrenous lung was seen lying in it and was removed, the patient making a good recovery.

The bronchi, especially those nearest to the gangrenous parts, are in a state of inflammation, and are frequently covered with membrane; they may even contain a solid branched cast, and such casts have been expectorated during life.

By the aspiration of the fœtid secretions, patches of broncho-pneumonia may be excited in various parts of the lung, either on the affected or on the opposite side, and these patches may also gangrene or suppurate, so that in such cases gangrenous patches are found in all stages, some just commencing and others almost cured.

¹ *Lancet*, 1852, i. 544.

The bronchial glands are inflamed and swollen, and sometimes even suppurating.

The blood-vessels in the neighbourhood of the gangrenous part are involved. Fortunately they are generally closed by clots, but where this has not happened, or not been adequate, hæmorrhage may occur, and, the patient dying of hæmoptysis, the parts may be found full of blood. The clots are sometimes extensive and spread to large trunks. Sometimes they break down and give rise to embolisms in different parts of the body, and notably in the brain. With gangrene of the lung, as with other forms of suppuration in the lung or pleura, cerebral abscess is by no means rare.

Narther¹ met with cerebral abscess in 8 out of 49 cases of gangrene, *i.e.*, in 16 per cent.

The gangrenous cavity communicates as a rule with the air-tubes through which the secretions are discharged, but it sometimes forms other communications, as, for example, with the mediastinum, œsophagus, peritoneum or some abdominal organ, besides breaking into the pleura or externally, as already described.

The seat of gangrene may be in any part of the lung or in both lungs, and in a single place or in many places.

The combined statistics given in Wilson Fox yield the following percentages:—

One lung only, 90 per cent.	<div style="display: inline-block; vertical-align: middle;"> <div style="display: inline-block; vertical-align: middle;">} Right, 54.</div> <div style="display: inline-block; vertical-align: middle;">} Left, 36.</div> </div>
Lesions multiple and scattered, 22·5 per cent.	<div style="display: inline-block; vertical-align: middle;"> <div style="display: inline-block; vertical-align: middle;">} Lower lobe, 42·7.</div> <div style="display: inline-block; vertical-align: middle;">} Upper „ 29.</div> <div style="display: inline-block; vertical-align: middle;">} Middle „ 5·8.</div> </div>
Limited to one lobe, 77·5 per cent.	

ETIOLOGY.—Gangrene is really a rare disease, but statistics are somewhat unreliable, because the diagnosis is so often uncertain until a *post-mortem* examination has been made.

Laennec saw only 2 cases in twenty-four years in his own practice. Statistics of autopsies show a frequency in proportion to other causes of death of about 1·6 per cent. (Fischl, 1·7; Boudet, 1·6; Heschl, 1·6). If these statistics are correct, the frequency of gangrene abroad must be much greater than in this country, for Coupland was only able to find 38 cases out of the records of ten years at the Middlesex Hospital, and at St. Bartholomew's Hospital the records show only 17 cases in ten years, yielding a percentage of only 0·3.

Sex.—Gangrene is certainly commoner in the male sex in the proportion of 2 or 3 to 1.

The statistics given in Wilson Fox combined (184 cases) give a proportion of 3 to 1; Heschl's of 2 to 1; Coupland's of 4 to 1.

Age.—Gangrene is rare in childhood and not frequent in old age. The chief prevalence is during active adult life and especially between 20 and 40. Age and sex thus, so far as they go, support the theory of the infectious nature of gangrene, for they show that it is men at the most active time of life who are most subject to it, *i.e.*, precisely those persons who would be likely to be most exposed to infection.

The combined statistics of Lébert, Laurence, Huntington and Fischl yield the following results:—

—10	—20	—30	—40	—50	—60	Above 60
3	17	51	44	35	31	22
The statistics at St. Bartholomew's Hospital are as follows:—						
...	...	1	7	5	1	...

¹ *D. A. f. klin. Med.*, xxxiv. 169.

By some authors gangrene is thought to be less rare in children than is usually stated. Thus Barthez and Rilliet refer to 18 cases under 15, the youngest of their series being at the age of 2½ years. Instances have been recorded at still earlier ages, viz., at 4 months and also at a year. The question may be fairly raised whether the gangrene in many of these cases was not secondary to diphtheria.

Gangrene is doubtless due to specific infection; but whether it be due to any particular organism or to many—and what these organisms are—are questions that are still undetermined. Putrefactive organisms of all sorts and kinds have been found, but no one which can be regarded as specific. This being so, ætiology has chiefly to concern itself with the conditions under which the infection may arise.

General debility or cachexia, whether due to general causes, *e.g.*, ill-health and anæmia, to special diseases such as diabetes, or to bad habits, like drinking, all of which have been assigned as causes of gangrene, owe whatever influence they possess to the diminished vitality of the tissues they cause; in other words, to the lessened power of resistance.

In the great majority of cases gangrene is the result of direct infection from some already gangrenous part of the body, the germs being brought to the lung either by the blood-vessels or the air-tubes.

Where the blood-vessels are the channels of infection, gangrene, like abscess, is usually preceded by embolism, but embolism is not of itself sufficient to cause either the one affection or the other, unless associated with the specific organisms, and of the two results, gangrene is the less likely, unless the part from which the embolus is derived is itself gangrenous.

It is no doubt through the air-tubes that the infective organisms most frequently reach the lung, and in most cases they are obviously carried by foul discharges, foreign bodies or particles of food, which have gained access to the tubes. Thus gangrene of the lung has been found associated with ulcerations in the larynx, trachea, and bronchi, with noma and other sloughing or diphtheritic lesions in the mouth and pharynx, and it has not infrequently followed operations on the mouth and tongue.

Theoretically the existence of lesions in the lung might predispose to the infection, but, as a matter of fact, even in such cases gangrene is far from common.

In phthisis gangrene is very rare.

I do not remember to have seen it more than two or three times out of several hundred *post-mortems*, though I am unable to give the exact numbers. Boudet observed it once out of 160 autopsies. Wilson Fox,¹ however, met with it in no less than 6 cases out of 99, but such an experience must, I think, be quite exceptional.

Chronic excavation, whether the result of chronic abscess, bronchiectasis, or phthisis supplies the most favourable conditions, and yet though fetid changes in the contents are not uncommon, gangrene is very rare.

The same may be said of putrid bronchitis.

Hæmorrhage and infarct only lead to gangrene when secondarily infected. It is in connection with croupous pneumonia that the occurrence of gangrene has excited the greatest interest. With ordinary broncho-pneumonia, however, gangrene is hardly ever met with.

Grisolle out of 50 cases of gangrene found not one due to pneumonia, and in 305 cases of pneumonia found not one of gangrene. Heschl also failed to find gangrene at all out of 149 cases of pneumonia. On the other hand, Behier met with it 5 times in 114 cases of pneumonia.

¹ Wilson Fox, *l.c.*

Coupland out of 38 cases of gangrene could trace only 8 to croupous pneumonia, and Heschl found the same number of instances out of 80 cases of gangrene, and in another series met with 6 cases out of 50, while Fischl out of 80 cases of gangrene could not refer it once to croupous pneumonia.

When gangrene of the lung follows an injury, it is in most cases due to direct infection from the external wound, as after a gunshot injury; but where there has been no external wound it must arise in the same way as pneumonia does under the same conditions, viz., by infection of the contused or inflamed part from within.

The following analyses of the lesions with which the gangrene was associated is given by Coupland and Heschl respectively:—

	Heschl. ¹	Coupland. ²
Embolism and thrombosis,	20	4
Carcinoma,	10	2
Pneumonia,	14	14 6 chronic pneumonia.
Phthisis,	11	... 1 thoracic aneurysm.
Bronchiectasis,	5	2
Pleurisy,	3	... 1 suppur. bronchial gland.
Trauma,	3	... 1 cerebral hemiplegia.
Cachexia,	5	...
Carcinoma of tongue,	10	4
Carcinoma of œsophagus,	3
Undetermined,	2	29
	<hr/> 83	<hr/> 58

Symptoms.—The symptoms vary greatly according as the process is acute or sub-acute, and according to the nature of the lesion which has been produced by it. In a typical case they are first, the symptoms of asthenic fever, as in any other case of septic infection; secondly, the evidence of a recent lesion in the lung; lastly, the signs of progressive excavation in the lung with the characteristic fœtor and sputum.

In the acute form the general symptoms are those of asthenic pneumonia. The prostration is considerable, the heart's action feeble and rapid, the tongue coated and dry, the skin sweating, the face drawn and pinched. The temperature is of a hectic type, with marked and irregular oscillations, and often not very high, while the asthenia is out of all relation to the amount of the fever. In all these symptoms there is nothing pathognomonic.

The *access* is often sudden and ushered in with rigors, which may be repeated at irregular intervals.

The *chest symptoms* are of the ordinary kind, viz., hurried respiration, with cough and expectoration, but they are not characteristic, until the signs of excavation present themselves in places where hitherto those of consolidation only were present. So far, all the signs might point to acute abscess or diffuse suppuration, and, until the fœtor and the characteristic sputum show themselves, the diagnosis will remain uncertain.

The *fœtor* is extremely penetrating and nauseating. It may be so powerful as to pervade the whole ward or house in which the patient lies. It may be one of the earliest signs to appear, and may be detected even within a few hours (twenty-four to thirty-six) from the commencement of illness. Usually it does not develop for a few days, and is at first only evident when the patient coughs or expires deeply. It varies a good deal in intensity from day to day, and in those rare cases in which there is no communication between the seat of gangrene and the bronchi, it may be absent throughout. Sooner or later the sputum

¹ *Virch. Jahrb.*, 1887, ii. 247.

² Coupland, *Brit. Med. Jour.*, 1885, Sept. 5.

becomes foetid, though the foetor may have been noticed in the breath for even some days before.

The *sputum* varies greatly in amount and in foetidity. It may be scanty, and consist of only a few stinking pellets, but as a rule it is abundant and formed of the gangrenous products, mixed with more or less of mucopurulent secretion from the bronchial tubes caused by the passage of the irritating discharges through them.

In a typical case it is brought up in considerable quantity, even to the amount of a pint or more in the twenty-four hours, and often in gushes after a paroxysm of coughing, as in the case of chronic cavities.

How large a part of the sputum is formed by the bronchial secretion is well illustrated by a case of Godlee and Williams,¹ in which a quart was expectorated daily, but the gangrenous cavity found *post-mortem* was not large enough to contain more than about an ounce of fluid.

When in any amount, the sputum is more or less fluid and separates on standing into three layers. The *upper* frothy, yellowish-green, opaque, and containing grayish or yellowish mucopurulent masses; the *middle* layer is opalescent, turbid, and resembles saliva in appearance. It contains albumen and much mucus. The *lowest* layer consists of a greenish or brownish sediment containing, besides much pus, greenish or brown foetid lumps. It is composed of pus and altered blood cells with blood-colouring matter, and fragments of lung tissue more or less disintegrated. The whole sputum teems with the organisms of putrefaction, and contains the various products of albuminous decomposition, *e.g.*, leucin, tyrosin, fatty acids, valerianic, butyric, lactic, caproic, etc.

The sputum is at first alkaline, but soon becomes acid owing to the development of fatty acids. A kind of digestion appears to take place in it, for the fragments of lung tissue rapidly disintegrate, and may in the end disappear. This affects even the elastic fibres, so that Trousseau regarded the absence of elastic fibres from the sputum as of diagnostic importance.

In these properties of the sputum there is nothing characteristic of gangrene, for they are all equally met with in the foetid secretion from chronic cavities or in putrid bronchitis. They are the simple result of the putrefactive decomposition of albuminous fluids, and readily take place in such fluids outside the body.

Cough is generally troublesome, but it varies with the amount of secretion, and, when the discharge is expectorated in gushes, is often paroxysmal.

Hæmoptysis is not infrequent, but it is rarely profuse, and in most cases not enough to do more than colour the sputum. In the subsequent stages, when the gangrene has ended in a chronic cavity, hæmoptysis is one of the chief dangers, and is not rarely fatal, but it is a risk not peculiar to gangrene and the result of the chronic excavation to which the gangrene has led.

Vomiting and diarrhoea are not uncommon. Sometimes both the vomit and motions have the same offensive foetor as the breath and sputum, possibly in consequence of the swallowing of the sputum.

In grave cases towards the end the patients pass into a typhoid state. Hiccough and tympanites then often occur, and are both very ominous symptoms.

The **physical signs** vary greatly both in extent and kind. They are only characteristic when together with the foetor and foetid sputum the signs of excavation develop in the seat of previous consolidation. As in other cavities, the physical signs vary greatly according to the amount of secretion which the cavity contains.

¹ *Lancet*, 1887.

The clinical state of patients suffering from gangrene differs greatly in different cases, and it is convenient to adopt some such clinical classification as Lebert suggests.

1. A pneumonic form, in which the case presents itself at first under the guise of a pneumonia. This is the usual type in the septic or embolic group, and where gangrene develops after pneumonia.
2. A typhoid form. In this form the symptoms may for some time be very misleading, and until the fœtor appears there may be little to suggest more than septicæmia. The most puzzling cases of all are those in which the exciting cause of the gangrene lies in foul discharges from the mouth, pharynx or air-passages.
3. A form in which the gangrene develops in the course of chronic excavation of the lung from any cause. In such cases the symptoms are often sub-acute or even latent. The fever may be slight, but the prostration is extreme, and a fatal result may occur sometimes suddenly and unexpectedly from collapse.
4. A group of cases in which the origin and course are insidious and obscure throughout, and, except for the fœtor, not characteristic.

Complications.—Many of the acute cases die before complications have had time to develop. The most likely complications are inflammation and perforation of the pleura, or septic broncho-pneumonia.

Broncho-pneumonia.—The fœtid secretions, as they pass out through the tubes, are liable to be sucked into some of them, and thus excite in healthy parts of the lung patches of broncho-pneumonia, which, as would be anticipated, are likely themselves also to gangrene, and thus in a case of gangrene there may be found many small gangrene spots irregularly scattered and in all stages of progress. It is remarkable that this secondary infection of the lung from its own secretions is not really more common than it is.

Pleurisy and Pneumothorax.—The pleura is always inflamed and often associated with effusion, which, as a rule, is purulent. In this way a fœtid empyema may be produced, and that without any direct communication between the pleura and the lung.

When the gangrenous patch is near the surface of the lung, the pleura itself is likely to become gangrenous and break.

Sometimes a great piece sloughs away and a large hole is formed; but at others perforation takes place in many places, as in a specimen in the St. Bartholomew's Hospital Museum, where the pleura over a gangrenous lower lobe is riddled with holes thirty or more in number.

The result of the perforation is pneumothorax, with more or less fœtid pus. In such cases death may follow the perforation at once as the result of shock, but more often death follows gradually from the fœtid empyema excited.

If the pleura be adherent, the thoracic walls may become involved and an abscess be formed with fœtid contents, and when this reaches the subcutaneous tissue widespread emphysema may develop. If the abscess burst externally or be opened, the gangrenous contents of the cavity may discharge themselves externally. If an abscess form either in the lung or in the pleura, and life be prolonged, it may burst, of course, anywhere, *e.g.*, into the abdomen or one of the abdominal viscera, into the mediastinum or œsophagus, or externally.

Another group of complications is connected with the vessels, and may be attributed to embolism. The vessels round the gangrenous parts become filled with clot. If this were not so hæmorrhage would be frequent. Some hæmorrhage is almost constant, but the amount is hardly more than sufficient to colour

the secretion. Even when more copious it can rarely be called profuse, and is seldom dangerous. As already stated, hæmorrhage in large amount is more likely to occur in the chronic cavity left behind by the gangrene than in the recent cavity while it is forming.

Although in most cases the clotting in the vessels is conservative and beneficial, it still brings with it certain risks; for example, the clot may disintegrate and give rise to embolisms, which, being septic, excite suppuration in the parts where they stick. In this way, no doubt, the occurrence of cerebral abscess in the course of gangrene is to be explained.

Diagnosis.—The diagnosis of gangrene of the lung is by no means easy. It is only when asthenic fever is present, associated with fœtid sputum containing fragments of lung tissue and with the physical signs of extending excavation, that the diagnosis is moderately certain. If fœtor were absent, the diagnosis could not be made from abscess or diffuse suppuration. If fœtor were present without expectoration, the odour might come from lesions in the mouth, nose or pharynx, but the fœtor in these cases rarely has the peculiar, penetrating, sickly quality of gangrene. If the expectoration were fœtid, this might also be due to fœtid bronchitis, or to the putrid decomposition of secretions in bronchiectatic or other chronic cavities, or to the discharge of a fœtid empyema. Fœtid expectoration containing lung tissue would raise a presumption in favour of gangrene, but even this would not be conclusive, for the fragments of lung tissue might be due to a sloughing or phthisical process, and the fœtor be derived from a chronic cavity. It is only by considering the difficulties and weighing the facts for and against in each case that a diagnosis can be accurately made. Even then it often amounts to no more than a presumption, and only becomes certain when the *post-mortem* examination has been made and the gangrene demonstrated.

Prognosis.—The mortality of gangrene is very high. The diffuse and multiple form is probably always fatal, but in the simple localised form recovery may occur.

There are few statistics to appeal to, but out of Lebert's 36 cases 6 recovered; this, however, I believe to be much above the ordinary average.

When death occurs it is usually after but a few days' illness—three or four days to a fortnight. The cause of death is usually either asthenia, with the general signs of septicæmia; or some complication of which pneumothorax is the most serious.

In any given case the prognosis depends upon the strength, constitution and general condition of the patient; the cause of the gangrene; and the complications which arise. When recovery takes place, a chronic cavity is left, which can only become obliterated in those rare cases in which it is very small and favourably placed, so that under the most fortunate circumstances a chronic cavity remains with the risks attendant on it.

Treatment.—Gangrene most frequently presents itself in the form of asthenic pneumonia, and calls for similar treatment. The temperature is rarely high enough to require antipyretic remedies, but the prostration is usually extreme, and must be combated by tonics, stimulants and good feeding.

Food must be given frequently, and in as large quantities as the stomach will bear. Stimulants are always necessary, and in good quantity during the early and acute stage, but when the affection has passed into the sub-acute or chronic condition, the amount may be reduced. Quinine in full doses is one of the stock remedies, as in other septic states; and another is iron, best given in the form of the perchloride, if the stomach will bear it.

Strychnia is useful either alone or in combination with other drugs, and it has the additional advantage that it can be administered *sub cutem* on an emergency.

Digitalis may be prescribed in small doses in combination with quinine or iron to strengthen the action of the heart, but the effect requires to be watched, and if the rate of the pulse fall much, as it may and that without any warning, its administration must be suspended.

One of the best drugs is caffeine, given in the form of the citrate. In doses of 5 grains two or three times a day it is a useful adjuvant to other remedies, and if dissolved in salicylate of soda it can be given *sub cutem*.

Specific treatment is unsatisfactory, for there is no drug known which exerts any reliable action upon the germs, or on the toxic products, to the absorption of which no doubt the asthenia is mainly due. Antiseptic remedies have been greatly used, and sometimes with apparent benefit, but more frequently with no obvious effect upon the process. When given by the mouth, they may do some indirect good by checking the putrefactive changes in the stomach and intestines set up by the foetid sputum which has been swallowed, and to which the vomiting and diarrhoea may be referred.

Creasote, either in the form of pill or emulsion, is the favourite drug, but carbolic acid, turpentine, eucalyptus, thymol, chlorine water, the sulphocarbolates, and many others have been recommended. The most efficacious remedy, at any rate in reducing the foetor, but also the most expensive, is musk, in doses of 3 to 10 grains two or three times daily. This drug not only seems to check the foetor, but also acts as a good general stimulant.

The antiseptics are most commonly administered in the form of inhalations, either dry or moist, but inasmuch as the patients are generally far too ill to be able to use an inhaler, or to bear a respirator over the mouth, the inhalation has to be provided for by medicating the whole air of the room in which the patient lies. This may be done by evaporating creasote, carbolic acid, thymol, eucalyptus, or other antiseptic over a lamp, or diffusing them by means of a steam kettle or spray.

In these ways, however, little is done except to destroy the foetor in the room, desirable enough as this is for the sake of the attendants as well as of the patient. For this object also the sputum should be received into a covered vessel containing some strong solution (20 per cent.) of carbolic acid, and the patient's mouth, teeth, and throat should be frequently cleansed to remove any particles of sputum which might hang about them.

It is only in the sub-acute or chronic cases, where the patients are not so seriously ill, that an inhaler or respirator can be used. A respirator is most convenient, for it can be worn continuously; and as good and pleasant an inhalation, as can be devised, consists of equal parts of creasote, tincture of iodine, ether, and alcohol, of which 10 or 20 drops can be sprinkled on the sponge or cotton wool two or three times in the day. The spray has no therapeutic advantage over the inhaler, and is more fatiguing and cumbersome to apply.

Attempts have been made to apply antiseptics directly to the affected parts by injections through the chest walls. The substances used for this purpose have been generally creasote, carbolic and salicylic acid, tincture of iodine, and thymol.

In the diffuse acute cases experience does not show any advantage from this method of administration, though repeated punctures have been made, without, at any rate, doing any harm.¹ In the sub-acute and chronic stages,

¹ Mosler, *2te Congr. f. Inn. Med.*, Wiesbaden, 1883.

however, cases are recorded in which benefit and sometimes cure have followed. Two instances of cure and one of improvement after injections of menthol and eucalyptus are recorded by Hewelke.¹

Where the gangrene is localised, or has ended in a cavity, the chief difficulty lies in the diagnosis of its seat, but where this can be done with tolerable accuracy, treatment by direct injection can be fairly tried and without risk.

For the bronchorrhœa, which is excited by the foetid secretions, and which goes so far to form the bulk of the expectoration, there is no better remedy than the antiseptic inhalations already spoken of.

Complications such as pleurisy, pneumothorax, broncho-pneumonia, and hæmoptysis must be dealt with in the usual way.

When gangrene ends in chronic cavity, the treatment becomes that of chronic excavation, and will be more fully dealt with elsewhere; suffice it to say here that some cases have been successfully treated by incision and drainage. The prospects are less favourable, where the contents are foetid, as they usually are after gangrene, than where they are simply purulent.

49. HÆMOPTYSIS.

Blood-Spitting.—When blood is spat, it may be derived from the mouth, pharynx, nose, œsophagus and stomach, or from the respiratory organs, but it is only to those cases where the blood comes from the respiratory organs that the term hæmoptysis is applied.

The blood spat presents different appearances under different circumstances. What is spat up may consist of pure blood, or of blood mixed with other discharges. If the hæmorrhage be copious, the other discharges, even when present, may not be obvious, but if the hæmorrhage be slight, the presence of the other discharges may clearly indicate the source from which the blood comes.

The following varieties of blood-stained sputum are met with:—

1. A watery fluid, uniformly stained with blood, and presenting a pink or bright red colour. Such sputum consists almost entirely of saliva, and the blood is derived from the gums or mouth, as examination easily proves, for the gums may be found spongy and the bleeding points be seen in the gums or elsewhere in the mouth.

Care must be taken not to confound the colour due to blood with that due to adventitious substances, such as port wine, tobacco juice, liquorice or medicine.

2. Streaks of bright blood, upon the outside of pellets or strings of mucus or mucopurulent secretion.

In such cases the blood comes from the large air-tubes or from the pharynx, usually from the former.

In chronic bronchitis the vessels in the trachea or main bronchi are dilated and often varicose, and during violent paroxysms of coughing may rupture. The little hæmorrhages that result will then appear as small streaks upon the outside of the secretion which the tubes contain.

In the same way in chronic pharyngitis, where the mucous membrane is spongy and congested, the violent hawking and coughing may cause some of the small vessels to give way and streak the secretion with blood.

Streaky hæmoptysis when following upon violent coughing has but little clinical significance.

¹ *D. med. Woch.*, 1891, xvii. 1130.

3. Viscid pellets, containing minute air-bubbles, and presenting a uniform pink, red or rusty colour, or sometimes of a darker purple, prune juice hue. Such sputum is characteristic of acute pneumonia, though occasionally met with under other conditions, *e.g.*, morbus cordis and phthisis.

4. Small solid lumps, of a purple or black colour, which contain no air-bubbles and sink in water. These are nothing else but small blood coagula derived from the alveoli, and due to the rupture of the distended capillaries in the walls of the air-cells, the dark colour being due to the fact that they have lain some time *in situ* before being expectorated. They are almost pathognomonic of mitral disease.

5. Pure blood, in some quantity, it may be many ounces, generally bright red in colour.

(a) Brought up repeatedly at short intervals in small quantities at a time.

If it contain many air-bubbles, it is derived from the respiratory organs, and constitutes the ordinary form of profuse hæmoptysis; if it contain no air-bubbles, it probably does not come from the respiratory organs, but from the mouth or pharynx; and on the whole is most likely to be due to epistaxis.

(b) Brought up in gushes and mixed with little or no air.

If bright red, it may come from the respiratory organs or the stomach; from the latter, in all probability, if the blood be in large quantity, for profuse hæmorrhage from the lungs would be almost immediately fatal.

If dark in colour, it is not likely to have come from the respiratory organs, but from some part where it can have remained long enough to have changed colour, and, therefore, probably from the stomach.

6. *Blood clots.*—These are generally bulky and gelatinous, and derived from the stomach. They may come from the lungs, and are then moulded to the shape of the bronchial tubes.

Blood casts of the bronchi are very rare. Some specimens were exhibited by me in 1880 before the Pathological Society.¹ The patient, a man of 43, was under treatment for phthisis of the left apex. While under observation he had a severe attack of hæmoptysis, after which a good deal of crepitation developed at the left base. This gradually disappeared, and was doubtless due to the passage of blood into the tubes of the lower lobe. Two months later he was again attacked with hæmoptysis, and brought me some lumps which he had coughed up, with great difficulty but with much relief to his dyspnoea. At the same time some dulness was present at the left base, which ultimately again cleared up as before.

Of the four casts shown three were nearly the same in size, about two inches long and a quarter of an inch in diameter at the thickest part of the stem. Two of them were much branched. The fourth cast was very much larger, being of the size and shape of the little finger, one and a half inches in length and half an inch in diameter. This was not branched.

They were all tough and partly decolorized, but still dull red in colour to the naked eye, and microscopical examination showed them to consist of nothing but consolidated blood congestion.

Blood casts of the bronchial tubes are casts due to the coagulation of blood in the air-tubes, and are very rare. At the time the specimen was shown, no other specimen had been exhibited to the Society. Indeed, their occurrence has been categorically denied by many competent observers, and in many books no reference is made to them. Dr. Cheyne² quotes a case in which a man was attacked with severe dyspnoea after hæmoptysis and expectorated a blood clot with great relief. The next day he expectorated a similar clot, and died soon afterwards from uncontrollable hæmoptysis. Dr. Cheyne states that such clots were the precursors of fatal hæmoptysis, but in the case I have quoted, the patient was living more than twelve months after the time the clot was expectorated, and the hæmoptysis, though considerable, was not really very severe.

Diagnosis of Hæmoptysis.—In most cases the diagnosis has to be made between hæmoptysis and hæmatemesis, but, as a rule, there can be little difficulty if the patient be under observation at the time.

¹ *Path. Soc. Trans.*, vol. xxxi. p. 53.

² *London Med. Rec.*, 1875.

If the hæmorrhage be small and come from the lungs, the blood is bright in colour, alkaline, frothy, mixed with sputum, brought up with a cough, does not clot, while the symptoms and physical signs show the lungs to be the seat of disease.

If the hæmorrhage be small, and come from the stomach, the blood will be dark in colour, often acid, contain no air-bubbles, be mixed with the contents of the stomach and brought up by vomiting, while the symptoms and signs indicate the stomach as the seat of disease.

If the hæmorrhage be copious, it will be in either case bright in colour, alkaline, not frothy, not obviously mixed with the sputum or the contents of the stomach, and will clot. The diagnosis must then be made by the general signs and symptoms. But in profuse hæmoptysis the respiratory symptoms are so well marked that a mistake in diagnosis would be difficult.

The diagnosis is, however, not so easy when the amount of hæmorrhage is not large, or where we have to depend only upon the history of the attack from the patient or the patient's friends.

The best question, then, to ask is this—Whether the patient continued to bring up blood for two or three days after the first attack, and whether the bleeding only ceased gradually; for, as a rule, in hæmatemesis the blood is brought up once or twice in a given day and then abruptly ceases, while in hæmoptysis the sputum continues to be stained with blood for several days after, and the blood-spitting only ceases gradually.

Hæmoptysis is the technical term given to that form of blood-spitting in which the blood comes from the respiratory organs.

The causes fall naturally into two groups. In the first the hæmorrhage takes place through the lungs, but is due to disease outside them, as, for instance, the rupture of a thoracic aneurysm. In the second the hæmorrhage is due to causes which lie in the lung itself, for example, phthisis, and they might be named extrinsic and intrinsic respectively.

A. EXTRINSIC—the cause of the hæmoptysis lying outside the respiratory organs.

Thoracic aneurysm.—When the final rupture occurs, the blood is brought up in gushes in large quantity and quickly suffocates the patient, so that in a few minutes from the commencement of the attack the patient is dead.

When a thoracic aneurysm presents beneath the skin, however thin the coverings may be, it is rare for the rupture to take place externally. It bursts, almost without exception, internally, and generally, as would be expected, into the lung or air-tubes. Nor does the final hæmorrhage occur without some warning of its approach. It is preceded by slight leaking, which shows itself either in the form of streaky hæmoptysis or occasionally in the expectoration of an abundant thin, watery, blood-stained fluid like saliva, and containing but few air-bubbles. The latter form is met with only, I believe, where the trachea is involved and perforation is threatened through it. I have seen both kinds of expectoration continue regularly or intermittently for as much as three weeks before the final rupture took place.

New growth.—Slight hæmoptysis in malignant intrathoracic growths is not uncommon, but profuse hæmoptysis is rare. In the latter case the tumour must have involved the lungs or bronchi as well as some large or fairly large vessel. Almost any vessel within the thorax may be opened out in this way, even the aorta itself. So also with a malignant growth in the neck, the end may be brought about by the rupture of the carotid into the trachea.

In the same way hæmoptysis may be caused by *suppurating glands*, *suppurating hydatids* or *abscesses* of other origin, as well as by foreign bodies within the air-tubes, and lastly by *injuries* of all kinds, *e.g.*, crushes, bruises, fractured ribs, stabs, and bullet wounds.

A curious case is recorded by Devergne, in which very profuse hæmoptysis occurred from an angioma of the epiglottis. These cases require no special consideration in this place.

B. INTRINSIC.—In the second group the causes of the hæmoptysis are intrinsic, that is, they lie within the respiratory organs themselves, either in the air passages or in the lung tissue, and they may be called bronchial and pulmonary respectively. Of course the terms bronchial and pulmonary could be applied with equal fitness to either extrinsic or intrinsic hæmoptysis, but it is in the latter group that the distinction is of the greatest importance.

Bronchial Hæmoptysis.—In the commonest form of bronchial hæmoptysis, viz., that connected with *chronic bronchitis*, the blood appears as streaks on the outside of the sputum, and is due to the mechanical rupture of distended vessels by violent coughing. In the same way it may be produced by the paroxysms of whooping-cough or by other violent expiratory efforts.

Ulceration of any kind in the tubes, *e.g.*, tubercular, syphilitic or malignant, often leads to hæmoptysis, though each may exist and run its course to the end without any hæmorrhage at all. The amount of blood lost varies greatly from a few streaks to a considerable quantity.

In *plastic bronchitis* hæmorrhage may occur and even be profuse. It precedes, as a rule, the expulsion of the cast, and is no doubt in great measure due to the paroxysms of coughing. There is no conclusive evidence to show where the blood comes from in these cases, but on the whole it is probably from the bronchi rather than from the lung. At the same time it must not be forgotten that plastic bronchitis is sometimes found associated with phthisis.

In one interesting group of cases the fault lies in the blood or blood-vessels. Thus in *hæmophilia*, *scurvy*, and *purpura*, hæmorrhage may occur from the bronchi as from any other free surface, but the bronchi are perhaps the last parts of the body to be affected in this way.

The same is true of the hæmorrhagic form of *acute fevers*.

I remember in one extreme case of this kind where bleeding had occurred into almost every tissue and from every free surface. At the autopsy the air-tubes were found covered with recently effused blood, even down to their very small divisions.

The following may be an instance of purpuric or hæmophilic hæmoptysis, but it is not quite clear.

A woman aged 44 came under treatment for hæmoptysis. She had at the time purpura with small petechiæ, on the legs chiefly, but also on the eyelids. No physical signs could be found in the chest. The hæmoptysis continued some time and gradually ceased.

In another class of cases the disease of the vessels is of a degenerative character, either of the nature of *atheroma* and met with in elderly persons, or associated with *granular kidney* and occurring earlier in life.

The former was described by Sir Andrew Clark as "non-tubercular and non-cardiac hæmoptysis of elderly persons," or, on account of its frequent connection with gout, as "arthritic hæmoptysis." It cannot be very common, for Sir Andrew Clark stated that he had seen only 20 instances of it in persons over 50 years of age in the course of fifteen years; the hæmorrhage had usually subsided, but had been occasionally fatal.

One well-marked instance has come under my own notice in a man over 70, who died at the age of 74 of apoplexy, in whom the first sign of illness was slight hæmoptysis, presumably of bronchial origin.

Of bronchial hæmorrhage in granular kidney there are not many cases recorded,¹ but considering the frequency of hæmorrhage from the nose, into the eye or brain, and the general change in the vessels throughout the body, hæmorrhage from the bronchi ought hardly to be as rare as it really appears to be.

¹ Case recorded by Dr. Francis Hawkins, *Lancet*, May 21, 1892.

Probably it has been overlooked or misinterpreted, as was the case with the bleeding from the bladder, to which I drew attention some years ago.

Laryngeal Hæmoptysis.—Bleeding may take place from the larynx in the course of any destructive disease, *e.g.*, syphilis, tubercle or cancer, but its amount is small and its clinical significance slight.

In the absence of such gross lesions, laryngeal hæmoptysis is very rare, if it occur at all. The cases recorded are very inconclusive. *Post-mortem* proof is lacking, for the cases are not fatal, and laryngoscopic evidence is defective, for when blood is seen in the larynx it may come from other sources, *e.g.*, the pharynx or trachea, or be due to ulceration of the larynx itself, not within sight of the laryngoscope. Some of the recorded cases are associated with general conditions more likely to cause hæmorrhage from other parts, *e.g.*, hæmophilia, cirrhosis of liver, heart distension, etc.

I have seen one case in which primary laryngeal hæmorrhage had been diagnosed, but the man soon after developed tubercular disease of the lung and larynx, and probably had it at the time.

I think the existence of this form of hæmoptysis open to very serious doubt.

Parasitic hæmoptysis.—In 1880 Barls¹ described a peculiar form of recurrent hæmoptysis, lasting often for many years, as common among the inhabitants of Tokio. He referred to 19 cases, 12 of which he had met with in a single year. It affected males only between the ages of 15 and 25, and was not associated with phthisis. The patients spat up pellets of a dirty, viscid, rusty mucus in which certain bodies were found. These Barls regarded as psorosperms, and he named the affection gregarinosis pulmonum.

In 1882 Patrick Manson² showed the so-called gregarine to be the ova of a distoma, the *Distoma Ringeri*. The ova were carried by drinking water, and so gained access to the body. The intermediate host has not yet been discovered, nor the seat of the parent in the body.

Manson found the disease very common in Formosa, where 15 to 30 per cent. of the population suffered from it. The disease does not appear materially to affect the general health, and is often disregarded by those who are affected by it.

Pulmonary Hæmoptysis.—*Injury.*—Hæmoptysis may be the result of a direct lesion of the lung, as by a fall, blow, or crush producing a bruise or contusion, or by a fractured rib causing a laceration. In either case hæmoptysis may be the only direct evidence that the lung itself has been injured. It is rarely profuse, and consists of pure blood mixed with air-bubbles, at first bright in colour, but getting dark later if the blood be not expectorated until some little time after the accident.

In the case of more serious injuries, as from a stab or bullet wound, the amount of blood will depend upon the size of the vessel wounded. If a large vessel be wounded the hæmorrhage may be so large that death may speedily ensue from suffocation, but where the large vessels escape the amount expectorated is rarely great.

Chronic congestion of morbus cordis and infarct.—The varicose and distended capillaries in the walls of the vesicles which are characteristic of the "heart-lung" often give way, and blood then escapes into the air vesicles, driving the air out before it. When the blood is not more than enough to fill a small group of vesicles no symptoms are produced, but the blood lies there, clots, and becomes of a dark, almost black, colour. After a time the clot is loosened and expectorated as the black or dark purple airless lumps which have been already described. When the amount of blood is larger, a greater part of the lung tissue is infarcted, even to the extent sometimes of nearly a whole lobe. This gives rise to one of the forms of infarct, and it may be large enough to be recognised by physical signs.

¹ *Centralbl. f. Med. Wiss.*, 1880, No. 39.

² *Med. T. and G.*, 1882, ii. 42.

With the infarction that follows pulmonary embolism the symptoms are, as a rule, more sudden and severe. The signs of pulmonary disturbance, dyspnoea pain and cough, date from the occurrence of the embolism, and may precede by some hours the appearance of hæmoptysis.

Where the embolism involves a large branch of the pulmonary artery, or where the mechanical congestion is sudden and extreme, as, for instance, in sudden failure of the left ventricle, the hæmoptysis may occur at once, and the amount of blood expectorated may be considerable. In speaking of infarction I have referred to a case of this kind where the symptoms were so urgent that life was only saved by timely venesection.

Acute inflammatory congestion.—The best example of this is seen in acute pneumonia. The amount of blood is usually small and intimately mixed with the products of inflammation; in other words, the sputum consists of inflammatory exudation stained with blood. Expectoration does not usually occur until a little while after the exudation, so that the colour is not bright but dull, rusty or even darker. The amount of blood is, as a rule, quite small, and only sufficient to stain the sputum; when more copious it may give the sputum the prune-juice colour which is of such bad omen.

Occasionally the hæmoptysis is profuse and bright.

I have recorded an instance of this in a case of very severe pneumonia, where there was so much bright blood brought up that the patient was thought to have phthisis as well, and to have ruptured a blood-vessel in an old cavity. The autopsy showed that no other lesion than acute pneumonia was present.

Another but less severe case of this kind is referred to in the chapter on pneumonia.

In *gangrene* of the lung the sputum is generally stained with more or less of altered blood, but except when the gangrene has become limited, and the slough detached, pure blood is but rarely expectorated. It may then be profuse, but I do not know of any instance in which the hæmorrhage of itself has been fatal.

With *abscesses* of the lung, whether formed in the lung or bursting through it, some blood may be mixed with the pus, but rarely in any quantity unless a large vessel has been opened. When an abscess has ended in a chronic cavity, the same lesions of vessels may be produced with which the ordinary forms of hæmoptysis are associated, viz., aneurysm and erosion of the pulmonary vessels, but these will be conveniently dealt with in connection with phthisis.

Vicarious menstruation.—I suppose it is right to refer to vicarious menstruation as a cause of hæmoptysis, but I have never seen a reputed instance of it which stood critical examination. I do not know of any satisfactory case recorded, and the majority of those best qualified to judge either doubt or deny its existence. I think, therefore, that I am justified in dismissing the subject thus curtly.

HÆMOPTYSIS IN PHTHISIS.

Hæmoptysis in phthisis varies greatly in amount from mere streaks in the sputum up to a pint or more in the twenty-four hours, and it may be described accordingly as *streaky*, *small*, *moderate* or *profuse*. In most cases it is moderate, and not more than two or three ounces in the day. It is usually recurrent, that is to say, a patient who has had one attack of hæmoptysis is likely, sooner or later, to have another. In some cases hæmoptysis is so frequent an occurrence that a special hæmorrhagic tendency seems to exist, and this tendency is sometimes observed to run in families and to constitute the family type of the disease.

Hæmoptysis is, however, not constant in phthisis, and appears to be entirely absent in about 20 per cent. of all cases¹; in many of the rest there may be but one or two attacks of hæmoptysis, and those, perhaps, only of slight degree, throughout the whole course of the disease. The importance, therefore, of hæmoptysis lies not so much in the amount of blood lost as in the indication it gives of the existence or progress of the disease.

Hæmoptysis is rarely fatal, and never except when it is profuse. It is not the cause of death in more than 1 or 2 per cent. of all cases.¹ When fatal it leads to death in one of two ways, either at once by suffocation, or gradually after frequent recurrence by exhaustion from loss of blood.

On the whole, hæmoptysis is not so common in acute cases as in the sub-acute or chronic forms of the disease.

Except where the hæmoptysis consists of a mere streak or two in the sputum, its cessation is not abrupt but gradual, so that the sputum continues to contain blood, though diminishing in quantity and changing colour, for two or three days, or even longer, after the bleeding into the lung has stopped. This fact is important in diagnosis, and serves in most cases to distinguish hæmoptysis from hæmatemesis.

Streaky hæmoptysis—the blood appearing in small streaks on the outside of the sputum. This may come from the lungs and precede a large hæmorrhage, but in the vast majority of cases it comes from the bronchial tubes. As a matter of fact, streaky hæmoptysis is far more frequent in bronchitis than in phthisis. When it occurs in phthisis it is due generally to the same cause, viz., the rupture of distended capillaries in the bronchial tubes as the result of violent coughing; but when the tubes are the seat of tubercular ulceration, bleeding may sometimes take place from the ulcerated surface, usually in small amount, producing the form of hæmoptysis we are describing, but occasionally in larger amount.

Small hæmoptysis.—The blood may be in very small amount and intimately mixed with the sputum, giving it a dull red or rusty colour, as in pneumonia, or a pale pink salmon tint. This is often not recognised by the patient as blood-spitting.

Sometimes small dark lumps are brought up, as in mitral disease, and produced in the same way by slight leaking from some of the minute vessels in the walls of the vesicles, the blood having been kept long enough in the lung to undergo the colour changes. Such lumps are usually expectorated at the end of a larger hæmoptysis, and rarely constitute the whole attack.

Moderate hæmoptysis.—The ordinary hæmoptysis in phthisis is small, and does not amount to more than one to two or three ounces in the twenty-four hours. The blood is bright red in colour, frothy, i.e., containing air-bubbles, most of them of small size; it is alkaline, mixed with the sputum, which presents the ordinary phthisical characters, and does not clot. It is brought up in small mouthfuls at a time with repeated coughing, but, as a rule, without much effort. The patient often volunteers the statement that after a short cough something was brought up into the mouth which felt warm, and which was recognised by the taste, or observed on expectoration, to be blood.

Profuse hæmoptysis.—When the blood expectorated in the twenty-four hours reaches half a pint or more, it may be called profuse. The patient coughs almost constantly, and with every cough brings up a little blood, so that in the

¹ West, *Med. Chir. Tr.*, lxviii.

course of the twenty-four hours the quantity mixed with the sputum may amount to a pint, but this quantity is rarely exceeded. Such hæmoptysis may last for many days. Thus I have known an instance in which the patient continued to spit up more than half a pint daily for forty-five days, at the end of which time he died of exhaustion.

Clinically profuse hæmoptysis may be divided into two groups, according as it is fatal or not.

Fatal hæmoptysis is rare in phthisis, and does not account for more than 1 or 2 per cent. of the deaths, that is to say, out of every 100 persons who die of phthisis, 98 die of some other cause than hæmoptysis.

In the fatal cases death is brought about in one of two ways, either by suffocation or by exhaustion.

In the former, blood is brought up in great gushes and chokes the patient, so that he dies suffocated in a few minutes. To this form the descriptive name of *Suffocative Hæmoptysis* has been given. In the latter the hæmorrhage is not so profuse, but is frequently repeated day after day, until at last the patient dies of exhaustion consequent on the continued loss of blood.

The pathology of fatal hæmoptysis in phthisis.—The following account is based upon the experience of fifteen years at the Chest Hospital,¹ Victoria Park. The cases number 26—20 males and 6 females.

Age.—Of the 26 cases, 20 were males and 6 females.

	Age	15-20	20-25	25-30	30-35	35-40	40-50	50-60		
Males	.	2	2	4	2	2	3	4	...	19
Females	.	3	1	1	0	0	1	0	...	6
		—	—	—	—	—	—	—		
Totals	.	5	3	5	2	2	4	4	...	25

In one of the 26 the age is not given, and although the numbers are too small to justify any absolute conclusion, this table seems to show that there is no special liability at any particular age.

The earliest case was in a lad of 16, and the latest in a man of 53.

Sex.—It is noteworthy how much more frequent these cases seem to be in men than in women, in the proportion of 20 to 6 or $3\frac{1}{3}$ to 1.

This may be associated with the fact that the male has a greater power of resistance to phthisis than the female, or, in other words, that the disease more often becomes chronic in the male.

Cause of hæmorrhage.—The source of the hæmorrhage was found in 17 cases out of 25, and proved to be aneurysm in 11 and ulcerated vessel in 6.

In the cases in which I made the autopsy myself I only twice failed to find the source, and one of these cases of failure is not included in the present number. Kidd examined 80 cases and found the ruptured aneurysm in 70.

Too much stress must not be laid upon the term used, for ruptured aneurysms sometimes look like ulcerated vessels, especially when, by the force of the blood-stream, the whole or nearly the whole of the sac has been torn away, and I further observe in the *post-mortem* notes of recent years that aneurysm is a term more commonly found than ulcerated vessel, while in some of the earlier notes the diagnosis of ulcerated vessel has the explanation added, "No saccular dilatation found," but, for the reasons given, the explanation is hardly adequate.

In my own cases I have only once or twice seen ulcerated vessels among many cases of aneurysm, and Rasmussen says that he has himself never met with an ulcerated vessel as distinguished from aneurysm. The difference is therefore probably in most cases rather one of terms than of reality.

¹ *Med. Chir. Tr.*, lxviii. Two important papers deserve reference to—Wald Rasmussen, *Ed. Med. J.*, vol. xiv., and Douglas Powill, *Tr. Path. Soc.*, vol. xii.

The condition of the lungs.—Phthisis was in all cases present, and was clearly the primary cause. Cavities were always found, and cavities not of recent date, but with thick fibroid walls, and ribbed or crossed by coarse trabeculae, such as are found usually in the most chronic forms of phthisis.

Both sides were similarly affected, though not to an equal degree, in 16 cases, while in 9 one side only was excavated, the opposite lung being in the condition of complementary emphysema or compensatory hypertrophy.

In other words, profuse hæmoptysis generally occurs in chronic phthisis, or from chronic cavities, not very often in the sub-acute, and rarely in the acute form of the disease.

The side.—The hæmorrhage came from the left side for certain in 11 cases, and with probability in 5 more; and from the right side for certain in 6, and with probability in 3 more. So that the left side is more often the source of the hæmorrhage than the right in the proportion of 16 to 9, or nearly 2 to 1.

It does not, however, necessarily follow that the source of the hæmorrhage is found upon the side most affected, or, if so, in the most diseased part of it.

The seat.—Any chronic cavity, whatever its size, may be the source of the hæmorrhage, from a small single cavity—the only spot of disease, it may be, in the lung—not larger than a filbert, to an enormous cavity, produced by the complete excavation of the whole lung.

The cavity from which the hæmorrhage came was in the upper lobe 9 or probably 10 times, in the middle lobe twice, and in the lower 7 times, and of these last it was found 6 times in the apex of the lower lobe. In two cases the whole lung was excavated.

In the majority of cases the hæmorrhage came from the upper lobe. The next commonest source was the apex of the lower lobe, or in the middle lobe. The lower part of the lower lobe is an unusual position. The favourite seat, therefore, is the middle of the lung laterally, and near the periphery, whether in the lower part of the upper lobe or the upper part of the lower. This is also the spot at which perforation most frequently occurs in pneumothorax, and it is interesting to associate these two facts together.

Aneurysm of the pulmonary artery.—A distinction is often made between aneurysms, *i.e.*, definite sacs or pouches, and ectasias or dilatations. I do not think it desirable or necessary to make this distinction, for the two classes are differentiated by no fixed characteristics, and there is every transitional stage between the one and the other, so that it is simpler to regard them as different degrees of the same affection, and to speak of them all as aneurysms.

These aneurysms spring always from a branch of the pulmonary artery, sometimes from one of the main divisions, but more often from a medium-sized branch, though frequently at only a very short distance from the origin of the smaller branch from the main trunk, so that a bristle may pass quite easily at once into the main vessel. These facts are of importance, I think, as bearing upon the origin of these aneurysms. They develop, as a rule, in the longitudinal axis of the vessel, away from the main trunk and into the lung-cavity.

They are usually found upon a trabecula which forms a more or less prominent ridge in the walls of the cavity; sometimes, though rarely, upon a trabecula which crosses the cavity. Occasionally there is no indication of the trabecula, but the aneurysm projects at once from what appears to be the smooth wall of the cavity.

The trabeculae are the remains of the indurated vessels and bronchi of the lung, and are largest towards the root of the lung. Hence the aneurysms also are found in the part of the cavity nearest to the root. Cavities, therefore, in which aneurysms are being searched for may be opened through the pleura, *i.e.*, from the periphery, without risk of destroying the aneurysm.

Their *shape* is, as a rule, more or less globular, arising from one side of the vessel. Those of large size have frequently secondary pouches or sacculations upon them, and sometimes look almost like a mulberry. At other times they form more irregularly oval swellings, also with secondary sacculations. This is

the condition to which the name of ectasias is given. True fusiform aneurysms are, I think, from the nature of things almost impossible.

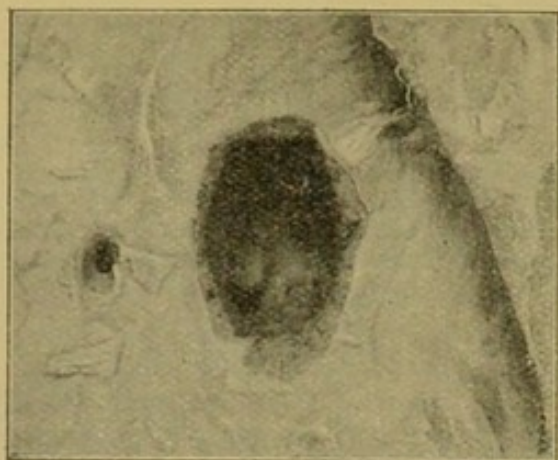


Fig. 101.

Aneurysm of the pulmonary artery in a chronic cavity of the lung, the walls of which are very thick. The cavity has also perforated into the pleura, producing pneumothorax. The patient died of hæmoptysis, the bleeding taking place into the pleura as well as from the mouth.

The aneurysms are usually small, sometimes not larger than a pea, only very rarely larger than a Morella cherry, *i.e.*, $\frac{1}{2}$ inch to $\frac{3}{4}$ inch in diameter. Aneurysms as big as a walnut are very unusual. I have recorded two of this size; one of them, oval in shape, measured one inch and three quarters long and one inch wide. I have never met with a description of a larger aneurysm than this. There is no relation between the size of the cavity in the lung and the occurrence or size of an aneurysm. The cavity may be so small as to be completely filled by the aneurysm, and that too when other much larger cavities are present (*cf.* fig.). In two of the cases I have recorded the cavity was formed by excavation of the whole lung, while the aneurysm was of the usual size.

Aneurysms are in most cases single. This is remarkable, but instances of multiple aneurysm are recorded by many

observers. One of the cases in this series had several,¹ and Kidd records a case in which there were 22.

With regard to *clotting*, Rasmussen stated that pulmonary aneurysms never contain laminated clot. Further observation proves this statement to be incorrect. It is, however, true that they frequently do not. The larger aneurysms generally do according to my experience, and many of the smaller ones may. Dr. Percy Kidd's case is a notable instance of this, for nearly every one of the numerous aneurysms there found was occupied by laminated clot, which in some nearly filled the cavity completely.

Many of the solid lumps found projecting from the walls of chronic cavities are, I believe, aneurysms, which have become obliterated or cured by clotting, in the same way that aneurysms cure elsewhere; but clotting does not necessarily prevent rupture in pulmonary, any more than in other, aneurysms.

Clotting often occurs not only in the sac, but also after rupture outside it in the cavity, and this clot, too, is sometimes laminated. The size of the aneurysm thus becomes sometimes deceptive, and it appears much larger than it really is.

This clotting may explain the cases of remittent hæmoptysis, but though essentially a conservative process, whether within or without the sac, it does not necessarily prevent a fatal result.

The seat of *rupture* is generally at the periphery, *i.e.*, at a point distant from the vessel from which it springs, but occasionally it is found at the base of the sac, as it were between the sac and the vessel.

The aperture is sometimes small, and may then be easily closed by clotting; at other times it is irregular and large, and in some cases the whole sac or the greater part of it is torn bodily off, and the rent into the vessel is represented by

¹ The cases are set out in the original paper.

the origin of the sac. Some of these cases would look very like simple erosion of the vessel. In some of my own cases, I was only able, after careful examination, to satisfy myself that I had an aneurysm and not an erosion to deal with.

As in aneurysms elsewhere, the final rupture rarely comes without warning. In most cases there is, or has been within recent periods, some premonitory hæmorrhage. This clinical fact, associated with the known pathology, shows the

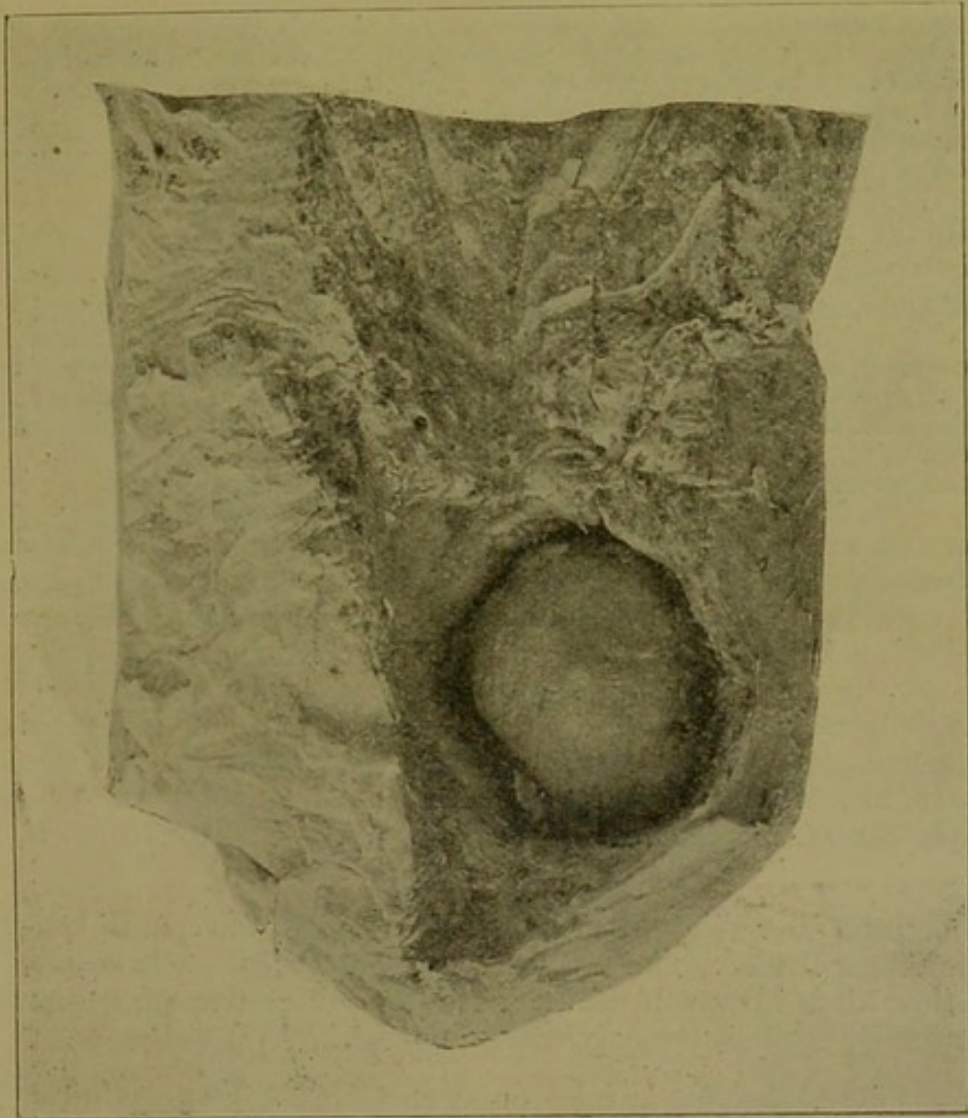


Fig. 102.

Very large aneurysm of the pulmonary artery in a chronic cavity at the base of the lung. From the Museum at St. Bartholomew's Hospital.

importance of early and strict treatment of even slight hæmorrhage in chronic phthisis.

Rasmussen draws a distinction as to the access and the amount of hæmorrhage between the two classes, which he makes, of aneurysm and ectasia, but I do not think so sharp a distinction is warranted either by clinical experience or by pathological observation.

The *pathogenesis* of pulmonary aneurysm is, I think, simple. There is no case recorded, so far as I am aware, of an aneurysm of the pulmonary artery developing in an otherwise healthy lung. It is conceivable that atheroma or syphilitic disease, or some other primary affection of the pulmonary artery, might

lead to aneurysms, but such conditions are, to say the least, very rare, and I know of no conclusive case recorded. Hence we are justified in connecting the development of the aneurysm with a pre-existing cavity in the lung.

The *development* of the aneurysm may be attributed :

I. To changes set up in the walls of the vessel, of an inflammatory or degenerative nature, by direct extension from the walls of the cavity.

In nearly all acute inflammatory, or rapid cases of phthisis, clotting occurs in the vessels of the neighbourhood, and they are early obliterated. When the process becomes circumscribed, and a chronic cavity is formed, it frequently happens that a vessel of larger size remains unobliterated, and that its coats become subsequently involved in the same fibroid change which has occurred in the walls of the cavity. If the vessel remain patent the blood pressure within slowly distends the fibroid part to form a pouch.

II. To the want of support upon the side towards the cavity, so that this side of the vessel is not only the weakest in itself, but also the direction of least external resistance.

It is, moreover, not unusual to find the vessel from which the aneurysm springs constricted, or even perhaps completely obliterated peripherally, a condition which tends still further to promote the formation of aneurysm, by making the same spot also the point of maximum pressure from within.

These facts explain the association of aneurysm with chronic cavities only.

Ulceration of Vessels.—Where a cavity is of more recent formation and has not passed into the chronic stage, or where in a chronic cavity ulceration has set in, the changes in the walls of the vessel are of a more acute character. They too may become involved in the ulceration, and after considerable thinning may rupture. But in most cases this is prevented by clotting within, and the vessel is quickly obliterated. This occlusion of vessels is so common in phthisis that it has been regarded as one of the most characteristic features of the disease. If it were not so, hæmorrhage ought to be even more frequent than it is, and fatal hæmoptysis one of the commonest causes of death. The contrary is, however, the case.

Although the pulmonary artery is the vessel usually affected in both aneurysm and ulceration, still the pulmonary vein may be attacked. I have met with one case of this kind in which death was due to ulceration of a branch of the pulmonary vein, but it is the only case I know of.

The facts established about fatal pulmonary hæmoptysis are briefly these :—

1. Chronic phthisis is the predisposing condition of the lung.
2. Rupture of an aneurysm or ulcerated vessel is the immediate cause.
3. It may occur at any age. There is no period of life which is specially liable, nor any which is exempt.
4. Men suffer more frequently than women, in the proportion of about 3 to 1. Possibly the greater frequency of chronic phthisis in men may account in part for this.
5. There is, I believe, no case of fatal hæmoptysis recorded in which the *post-mortem* examination disclosed the lesions of acute phthisis only.
6. Diligent search will rarely fail to discover the source of the hæmorrhage, and, considering the difficulties, occasional failure is hardly matter for surprise.
7. In the great majority of cases the hæmorrhage is due to the rupture of the sac of an aneurysm of the pulmonary artery. In the small minority it is traced to ulceration of a branch of the pulmonary artery, or possibly, in some very rare cases, of the pulmonary vein.

8. Of *pulmonary aneurysms* the facts known are these:—

a. They are of small size, rarely larger than a Morella cherry, often much smaller.

b. They always occur in chronic cavities, which they may sometimes completely, but more often only partially, fill.

c. They spring from a prominent trabecula either situated upon the walls of the cavity or crossing it, or else directly from the walls of the cavity itself.

d. They are, as a rule, globular in shape, but not unfrequently irregularly pouched and attached to the vessel by a broad base.

A distinction has been made between aneurysms and ectasias or partial dilatations, but the difference is only one of degree.

e. They are often surrounded by laminated clot, so that their size becomes deceptive.

f. Frequently also they contain laminated clot, though this has been denied.

g. The vessel from which they arise is generally of moderate size, two to three lines in diameter. They are, however, often situated close to the origin of this vessel from one of the main branches of the artery.

h. The rupture varies much in size; sometimes it is a linear slit, more often an irregular rent, and sometimes nearly the whole sac of the aneurysm is torn off; so that the difficulties of diagnosis from an ulcerated vessel become very great.

i. They are frequently single, but many cases are recorded in which more than one existed, and in a few cases they have been numerous.

j. The cause of aneurysm is to be referred in the first place to chronic changes set up in the walls of the vessel by extension from the walls of the cavity, and secondarily to want of support on the side towards the cavity, as well as to partial obliteration of the distal portion of the vessel.

k. The cavities in which aneurysms are found are always chronic, with fibroid and usually trabeculated walls. They may be of any size and occupy any position in the lung, but they are more frequently small, and their favourite position is in the mid-lateral region peripherally. They are sometimes completely, but more commonly only partially, filled with the aneurysmal sac, and frequently contain clot, which may be decolorised and laminated. They may sometimes be so small, and the pulmonary disease so limited, that diagnosis of the lesion during life may be almost impossible from physical signs alone.

9. *Ulceration or erosion of vessels* is a much less common cause of fatal hæmoptysis than aneurysm.

It occurs, however, under similar conditions. Usually it is the pulmonary artery which is affected, but on one occasion I found the rupture in a branch of the pulmonary vein.

It is conceivable that ulceration might lead to fatal hæmorrhage in acute phthisis, but I do not know of any *post-mortem* of this kind recorded; for the vessels, though of course early and considerably involved in the disease, become quickly plugged and impervious.

The question now arises, "Are the causes of profuse pulmonary hæmoptysis the same in the non-fatal as in the fatal cases?"

In order to establish the identity of pathology in the fatal and non-fatal forms, it will be necessary to show—

1. That, except in respect of the result, there is no clinical difference between the two sets of cases; and

2. That aneurysm and ulceration of the pulmonary vessels, whether after rupture or not, may heal.

Of the fatal cases there are two groups. In the first, death is sudden, and to this the name of *Suffocative Hæmoptysis* has been well given, for the patients die after a few minutes, suffocated by the blood poured into their air-tubes. In the second, the hæmorrhage occurs again and again, and death is the result of exhaustion from loss of blood; but even in this group death may be sudden at the last, though it is then due not so often to suffocation as to cardiac syncope. To this second group the name of *Remittent Hæmoptysis* has been given—an appropriate name if used, in the sense in which it is applied to fevers, to mean a hæmorrhage which recurs before the previous one has completely ceased. But all remittent hæmoptysis is not fatal, and from this group of non-fatal remittent hæmoptysis we pass to another class, which may on the same analogy receive the name of *Intermittent Hæmoptysis*, but which differs from the previous group only in the longer intervals between the attacks and in the complete recovery meanwhile, so that we may trace clinically every gradation, from the non-fatal intermittent and non-fatal remittent to the fatal remittent, and finally to the suffocative hæmoptysis.

I omit entirely for the present the consideration of hæmoptysis which does not deserve the name of profuse. For here we have to deal with a more obscure and difficult pathology, though there can be little doubt that the process and the lesions are the same.

1. The following cases establish the first of the propositions, viz., that the cases of profuse hæmoptysis, whether fatal or not, all belong to the same clinical family.

(1) Hæmoptysis fatal from suffocation.

CASE I. — *Remittent hæmoptysis—Short duration—Death from suffocation—Ruptured aneurysm.*—John R., aged 38, ill two years, admitted with very extensive excavation of the left lung. After he had been in the hospital for five weeks, he was seized with profuse hæmoptysis, and spat daily for six days about a pint of blood. On the seventh day he died suddenly in a more profuse attack than usual.

The *post-mortem* showed that the left lung was completely excavated, a few coarse ridges only remaining at the root over the course of the great vessels and bronchi. Upon one of them was a ruptured aneurysm as large as a cherry.¹

CASE II. — *Remittent hæmoptysis—Long duration—Death from suffocative hæmoptysis—Large ruptured aneurysm.*—George G., aged 21, a labourer, was admitted for hæmoptysis.² He had had a cough for about fifteen months, and had occasionally spat up a little blood, but never much. A few days before admission hæmoptysis began again, and rapidly became severe. The patient was in the hospital forty-five days, and spat up on the average half a pint of blood daily, at first regularly every day, but towards the last, two or three days at a time passed without hæmoptysis. In the last attack he brought up 37 ounces, and died of suffocation.

The *post-mortem* examination disclosed but little change in the left lung, but the right was adherent, except the lower part, where there was a localised empyema, containing about one pint of pus. The upper wall of this cavity was formed by the collapsed lower lobe of the lung, in the mid-lateral region of which was an irregular cavity about two inches in diameter, filled for the most part with laminated clot. Occupying the upper part of the cavity was an aneurysm, oval in shape, and measuring $1\frac{1}{4}$ inch by $\frac{3}{4}$ inch. The rupture was a small linear slit, one eighth of an inch long.

(2) Hæmoptysis fatal from exhaustion.

CASE III. — *Remittent hæmoptysis—Death from exhaustion—Aneurysm—Limited lung lesion.*—A man, aged 45, had been in good health and in active work until fourteen days before admission, when, after running some distance, he was seized with hæmoptysis, which since that time had returned on the slightest exertion. In the hospital he had several attacks of profuse and obstinate hæmoptysis, and finally died of exhaustion.

¹ *Pathological Society's Transactions*, vol. iii. p. 25.

² *Ibid.*, vol. xxxv. p. 94.

Both the lungs were emphysematous, and in other respects healthy, except that in the left, in the upper part of the lower lobe, two small old cavities with fibroid walls were found, and in one of these a ruptured aneurysm the size of a cherry.

This case is important, as showing how very limited the disease may be, and how difficult, and perhaps impossible, it may sometimes be to diagnose it.

The next case illustrates these facts again.

CASE IV.—*Remittent hæmoptysis—Death from suffocative hæmoptysis—Limited lung lesion.*—A woman, aged 46, was brought in dead, having been found lying in a pool of blood.¹ She had been, it transpired, an out-patient for a few days for slight hæmoptysis, but until this attack she had been, though never strong, in her usual health. She was the mother of twelve children.

Both lungs were healthy except in two places. At the apex of the right lung was a small wedge-shaped patch of fibroid induration, containing several small cavities with dense fibroid pigmented walls. In the base of the lung was a second patch of similar induration with similar cavities, and in the largest of these, the size of a walnut, was the aneurysm which had ruptured.

I desire to draw special attention to these latter cases, as showing how limited the lung disease may be.

The last case especially suffered from only slight hæmoptysis, such as would ordinarily give no anxiety, until the sudden fatal hæmorrhage occurred. This is very suggestive as to the pathology even of slight hæmoptysis.

(3) The cases which belong to the third group, that of remittent hæmoptysis with recovery, are common, and I need only select one or two as illustrations.

CASE V.—*Remittent hæmoptysis—Long duration—Recovery.*—Maurice N., aged 32, with no family history of phthisis, had slight pleurisy at 20, and since then had suffered occasionally from cough. He spat blood for the first time four years before admission, in slight amount only. In the second attack, one and a half years later, he brought up a pint of blood on one day, and small quantities for about a week. The third attack came on three months before admission. It was very profuse, and he was laid up for a month. On 21st December he had another attack, and expectorated a pint of blood. On the 24th and 25th he drank a good deal, and had spat blood every day since in varying amount. After his admission the bleeding rapidly subsided, and after a fortnight he was discharged. A week later he was readmitted with hæmoptysis. On the 14th of January he spat about 6 ounces; on the 15th about 10 ounces; on the 16th, 6 ounces; on the 17th, 13 ounces. A little only on each succeeding day until the 21st, when he again brought up 10 ounces. The bleeding then gradually subsided, and he was free until 4th February, when a few ounces more were brought up, and a little spat for a few days longer. On 14th February the patient had another slight attack, lasting also a few days, and on 20th March he was discharged.

The physical signs were very indefinite, but there was some crepitation in the region of the right nipple.

(4) The fourth and last group consists of cases of intermittent hæmoptysis.

It is quite unnecessary to bring forward cases of intermittent hæmoptysis which did not die of hæmorrhage. All the cases last referred to would serve as illustrations prior to the last fatal attack.

The series of cases of profuse hæmoptysis is as follows:—

1. Cases of simple suffocative hæmoptysis.
2. Cases of remittent hæmoptysis which were fatal—
 - (a) From suffocation.
 - (β) From exhaustion.
3. Cases of remittent hæmoptysis which recovered.
4. Cases of intermittent hæmoptysis which, after several attacks, ended at last fatally from hæmoptysis.
5. Similar cases to the last, which recovered.

In all the fatal cases above referred to the same pathological lesions were discovered *post-mortem*.

¹ *Pathological Society's Transactions*, 1878, p. 41.

2. I turn now to the second proposition.

If the pathology of the fatal and non-fatal forms of hæmoptysis be the same, we require evidence that aneurysm and ulcerated vessels, to the rupture of which the hæmorrhage is in both cases attributed, may heal.

I will take the question of aneurysms first. Several of the cases described show the presence in the aneurysm of laminated clot, and disprove therefore the assertion of Rasmussen that laminated clot is never found in pulmonary aneurysms.

In two cases the aneurysm was embedded in laminated clot.

Partial adhesions also often form between the sac of the aneurysm and the walls of the cavity. When the cavity is small and the aneurysm completely fills it, as it often does, complete adhesion may take place, and in this way the sac may obtain adventitious strength. In one case of this kind rupture took place in the only unprotected part, viz., at the mouth of a bronchus.

It may be objected, however, that if pulmonary aneurysms did heal in this way, they ought to be frequently found *post-mortem*. It is quite true that not many cases of this kind are described, but the explanation is, I think, simple; for, in the first place, they are hardly ever looked for, except when hæmoptysis has been a leading recent symptom, and the difficulties of finding them are much increased when there is no blood-clot to guide the search.

Dr. Percy Kidd found aneurysms in 15 per cent. of his *post-mortems*, usually only one, sometimes several, and in one case he found twenty-two in one lung, most of them filled with consolidated clots, but one had ruptured.

Many of the "fibroid masses," so frequently described in phthisis as existing on or in the walls of cavities, and of which no satisfactory pathological explanation is often given, prove to be, on careful examination, obliterated aneurysms.

The cure of ulcerated vessels admits of clearer proof. Though rarely a cause of fatal hæmoptysis, ulceration is a very common cause of profuse hæmoptysis.

From the very earliest commencement of excavation in the lung there is an active destruction of vessels. That hæmoptysis is not constant in every case alike, and that profuse hæmoptysis is not more common, depend upon the obliteration of vessels, which is almost part of the disease.

As with vessels in other parts of the body, the more acute the disease in their neighbourhood, the more certain, if they become involved in the process, is their rapid obliteration. It is only in connection with the more chronic processes that they are likely to remain pervious, and so lead to hæmorrhage. The most cursory examination of phthisical cavities establishes the applicability of these facts to the pathology of the lungs.

Though possible, it is extremely improbable that profuse hæmorrhage should occur in acute phthisis. Many, if not all, of the cases of phthisis ab hæmoptœ, where profuse hæmoptysis is the first symptom of a disease which afterwards runs an acute course, are not cases of new disease, but of old disease starting afresh. Cases III. and IV. show how limited the lesions may be in the lung, which give rise even to fatal hæmoptysis, and prove that the absence of physical signs is no evidence of the absence of chronic lesions, which may well be too small to be recognised by physical examination.

Instances of complete recovery after severe bleeding into the lungs are so common from various causes as to prove beyond question that it is not the blood itself which produces the disease, but something which the blood brings with it. This something is, of course, the infective tubercle bacillus.

From these considerations we should *a priori* expect that profuse hæmorrhage, in tubercular phthisis at any rate, could only occur in chronic disease; for aneurysms take some time to grow, and ulceration, if acute, leads at once to thrombosis; and further, that aneurysm will be a far commoner lesion than erosion. Each of these expectations is abundantly confirmed by *post-mortem* examination.

The evidence brought forward is sufficient, I think, to establish the required propositions, viz., that the causes of profuse hæmoptysis are the same, whether the case be fatal or not, viz., aneurysm or erosion of a vessel; and further, that in both cases alike cure is possible, and of not uncommon occurrence.

PROGNOSIS OF HÆMOPTYSIS.

Profuse hæmoptysis may be fatal at once by suffocation, or after a time, by exhaustion. Where the amount is small, it is not the loss of blood but the cause of the hæmoptysis that affects the prognosis. Thus even a streak or two may be alarming where the patient has a thoracic aneurysm, and may be the warning of rupture.

Again, hæmoptysis in an apparently healthy person is a grave symptom as indicating the existence of tubercular disease of the lung. In established phthisis, hæmoptysis often, so far from doing harm, seems to do good, and is quite as often followed by improvement as by exacerbation of symptoms.

The effects of injury and wounds of the lung prove that the mere presence of blood in the lung is followed by no evil results, the blood being rapidly and completely removed by absorption and expectoration, and no lesions follow. Dead blood, however, is a favourable soil for the development of infective organisms, so that in some cases infection may occur either from germs already present within the body or introduced afresh from without. Thus it may happen that inflammation of any degree of intensity may follow hæmorrhage into the lung from the mildest form up to the most septic, ending in abscess or gangrene. So again when phthisis follows or starts afresh after hæmoptysis the explanation is that the blood has come from some pre-existing tubercular cavity, and has carried with it over the lung the germs of tuberculosis. The risks of inflammation or phthisis depend upon the nature of the lesion to which the hæmorrhage is due, and the prognosis therefore varies with the cause of the hæmoptysis, but, speaking generally, blood of itself is simply absorbed and leads to no lesion.

TREATMENT OF HÆMOPTYSIS.

The importance of hæmoptysis and the need for active treatment varies with the amount of bleeding and the cause which has produced it. When the hæmoptysis is symptomatic as in pneumonia and infarct, accidental as in whooping-cough and bronchitis, or premonitory as in thoracic aneurysm, the nature of the case will determine how far treatment is necessary and what form it shall take. But in many instances the cause is not known at the time, and cannot be determined, for minute examination is impossible while the bleeding continues, and in such cases the treatment must be conducted on general principles.

In phthisis the general tendency of hæmoptysis is to spontaneous cure, *i.e.*, it tends to cease of itself if the patient be not placed under unfavourable conditions. It is this fact that makes it so difficult to estimate at their proper value the effect of drugs and the other methods of treatment employed.

Hence it follows also that with the slighter degrees of hæmoptysis, general treatment is all that is necessary, and that it is only with profuse hæmoptysis that the special forms of treatment will be required.

1. *Treatment of slight and moderate hæmoptysis.*—For the treatment of slight and moderate hæmoptysis, general management upon common-sense principles is all that is necessary, and the administration of special drugs is of subordinate importance.

General management.—Rest of the body in general and of the respiratory organs in particular is the chief guiding principle.

The patient must be kept in bed and all movement forbidden. He should not be allowed to get out of bed for any purpose whatever. If this is unavoidable, or such strict rules are not thought necessary because of the slightness of the hæmorrhage, the patient should be told to move slowly and gently. Too rigid adherence to the rule of absolute rest is not always advisable; for instance, the difficulty of passing water and motions while lying in bed, experienced by those unaccustomed to it, may lead to an amount of straining and excitement which may cause more effort than if the patient had been allowed to get up out of bed quietly for those purposes; but as movement accelerates both the respiration and circulation, the more strictly the rules of rest are enforced the better.

The patient should lie in the semi-recumbent position, with the shoulders raised and the head comfortably supported, the object being to keep the bleeding parts as high as possible, so as to relieve them of the pressure of gravity. This position is the one the patient often spontaneously assumes, as being that in which he is able to cough and expectorate with least exertion.

All talking must be forbidden on account of the excitement and fatigue it causes, as well as because of the respiratory effort it entails. Whatever the patient may have to say should be said quietly in a low voice or in a whisper.

Cough is a necessary part of hæmoptysis, and need not be checked unless it be excessive. This must be estimated by its relation to the expectoration. In most cases the cough is short and easy, and the sputum is brought up without much effort. Sometimes it is paroxysmal and out of all proportion to the expectoration. Then it must be checked, and for this purpose nothing is better than a little opium, in a mixture with glycerine and chloroform. Where the paroxysms are very severe, a few drops placed upon the tongue often control them.

The circulation must also be kept quiet. This is another reason why movement must be avoided. But as both respiration and circulation are easily affected by nervous conditions, all mental effort and excitement must be avoided or controlled. Hæmoptysis always causes great alarm, especially if it be a first attack, or if, when not the first, it be unusually severe. The patient almost always thinks that he is going to die. An assurance that this is most unlikely is a better sedative than any drug; but if a drug be necessary, the best for this purpose is opium or morphia, administered as required either by the mouth or by a subcutaneous injection.

The room in which the patient is lying should be kept cool, *i.e.*, neither too hot nor too cold; cold especially is to be avoided as so likely to increase cough.

The bowels should be assisted with a slight laxative, for the confinement to bed is likely to cause constipation and consequent straining. For this reason it is usual to administer a mild purge at once, and to keep up the action of the bowels by small doses of some saline purge like Epsom salts.

The diet must be carefully regulated. The food should be light and easily digestible. It should be given cold, not actually iced but cool, for very cold food is, I think, worse than very hot. Stimulants must be entirely avoided, unless specially indicated by exhaustion, and then discontinued as soon as may be. The patient is therefore put at once on liquid food; but even this must not be given in unlimited quantity, for an over-distended stomach will embarrass the respiration, so that the amount of liquid must be kept somewhat under the average. It is a mistake to let the patients slake their thirst with milk alone, for this will not be digested in too large a quantity, but will lie fermenting in the stomach, and produce wind. For the thirst it is best to let the patient suck

small lumps of ice, or to drink water acidulated with phosphoric acid or lemon juice.

As soon as may be the diet should be concentrated, and small quantities of solid food given, such as pounded or finely mixed chicken or mutton.

Medicinal Treatment.—So far as drugs go, all that is necessary in these cases of slight hæmoptysis is a little opium and a saline purge, with perhaps some ergot.

It is a common practice to administer small doses of mineral acid, of which the dilute or the aromatic sulphuric acid are the favourite, or to give gallic acid, tannic acid, alum, or lead acetate. Opinions as to the actual efficacy of these drugs vary greatly, but all writers agree that the addition of opium greatly increases their efficacy, from which I think we may conclude that it is to the opium that their real activity is due.

2. *Treatment of profuse hæmoptysis.*—In the severe cases of hæmoptysis the general principles of treatment are the same, but they must be most rigidly enforced, and supplemented by other methods, the aim of which is specially directed towards controlling the bleeding.

Hæmostatics.—Of these tannic acid, gallic acid, the mineral acids, and acetate of lead are the favourite remedies.

Gallic and tannic acid.—Tannic acid is probably not absorbed as such but is first converted into gallic acid in the stomach, and it is the latter drug that is most commonly prescribed. Gallic acid is most conveniently prescribed as the glycerinum, of which half a drachm to a drachm may be given in water every hour or so, according to the urgency of the case.

The mineral acids are also frequently given, the favourite being sulphuric acid. Their effect upon hæmoptysis is absolutely nil, but they are grateful to the palate, and in combination with gallic acid and opium, enter into most of the stock prescriptions for hæmoptysis.

Acidi Gallici, gr. x; [or Glycer. Acid Gall., 3 ss to 3 i]; Acidi Sulphur. dil., ℥ xv; Aquam. ad 3 i.

This dose to be taken every hour or every two hours.

To this may be added Tt. Opii, ℥ ii or ℥ iii; or Tt. Camphor Co., ℥ xxx.

When the preparation contains opium the frequency of its administration will be determined by the amount it contains.

Alum has been also employed, but it is useless, and so are *rhatany* and *catechu*. *Common salt* is a popular remedy, given in doses of a teaspoonful every half hour or so. Its real effect is nil, and it, of course, considerably increases the thirst.

The *perchloride* and other astringent preparations of *iron* are usually regarded as mischievous in hæmoptysis. If administered in full doses they are very likely to upset the stomach and to confine the bowels, results which it is desirable to avoid.

Acetate of lead is a stock remedy. It is stated to act as a sedative to the heart,¹ and at the same time to increase the clotting properties of the blood. It is given in full doses, not less than 2 grains every hour, and almost invariably in combination with opium. Whatever effect lead and opium may have upon intestinal hæmorrhage, the effect upon hæmoptysis is probably due rather to the opium than to the lead.

The drugs are given either in solution or as a pill. The solution is the best form, for pills take some time to dissolve, and are less certain in action.

Pill.—Plumbi acetat., gr. ii; Opii, gr. ½ or ¼; Extr. Hyoscyami, gr. ii.

Mixture.—Plumbi acetat., gr. ii; Acidi acetic dil., ℥ xxx; aquæ, 3 i; to which may be added Tt. Opii, ℥ ii or iii, according to requirements.

Digitalis.—The use of this drug is opposed to theory, and is not supported by experience, so that it is no longer included in the list of remedies for hæmoptysis.

Ergot and ergotine.—These are the stock remedies of the present day. The greatest difference exists as to their physiological action, so that their usefulness must be determined by

¹ Stillé and Maisch, *Dispensatory*, p. 1189.

clinical observation and not by theory. The widespread belief in their efficacy ought not to be disregarded, and, indeed, experience seems to prove their utility. Ergot and opium are the two remedies upon which greatest reliance is placed, but neither must be expected to do impossibilities in bleeding from the lung any more than in bleeding from any other part.

Ergot may be given in the form of the liquid extract by the mouth, but if more immediate action is required subcutaneous injections of ergotin are preferable.

Of the liquid extract a drachm may be given at once, and repeated in half an hour or an hour, after which 20 or 30 drops may be given as thought necessary.

Ergotine is given in doses of 3 to 10 minims of the official hypodermic solution, and repeated as required. The solution does not keep well and should be freshly prepared, or the tabloids or lamels used, and dissolved when wanted.

Astringent inhalations and sprays.—The only remedy used as a vapour for inhalation is *turpentine*.

A teaspoonful of the oil is sprinkled upon lint or a handkerchief, which is placed over the mouth. It is difficult to see what action is expected from it, but it is frequently employed. I have used it myself and seen it used frequently by others, but I have never obtained any convincing proof of its efficacy.

In the form of spray various astringent substances have been advocated.

Tannic acid (gr. 5 to 20 in the ounce); *alum* (gr. 30 to 60 in the ounce or a saturated solution); *perchloride of iron* in varying strengths.

The spray may be produced either by hand, *i.e.*, by compressed air or by steam. It is difficult to see what use a spray can be. The greater part of the spray is deposited in the mouth and throat, and unless the patient has learnt how to inhale properly none of it reaches even the larynx. If any did make its way to the distant parts of the lung, it is clear that it would pass more easily into any other part of the lungs than into the bleeding parts, where the tubes are more or less filled with blood; and even if it did gain access to these parts it could not be in sufficient amount or strength to have any effect. When to these objections are added the inconvenience and discomfort of the inhalation and the irritation and coughing it often excites, it is obvious that such inhalations are likely to do harm and cannot do good. In my opinion they are all useless.

Sedatives.—*Opium and Morphia.*—How far these drugs have any effect upon the large vessels whence the bleeding comes in hæmoptysis may be fairly questioned, but they certainly allay nervous excitement, and thus quiet both the respiration and the circulation. In this respect they are of the greatest use, and in most cases of severe hæmoptysis their administration constitutes an important part in the routine treatment of the disease.

In urgent cases morphia is best administered *sub cutem*, but in less severe cases small doses of laudanum may be given at short intervals by the mouth.

Remedies directed to diminish blood pressure.—*Cardiac depressants.*—Of these the chief are *tartar emetic*, *aconite*, and *ipecacuanha*, but they must be given in full doses sufficient to produce the effect intended.

The treatment of hæmoptysis by emetics was much advocated by Trousseau, and both tartar emetic and ipecacuanha were given by him until vomiting was produced. Most subsequent advocates of this treatment advise that their administration should be stopped short of emesis.

The objection to tartar emetic is that it is so likely to cause stomach and intestinal irritation. From this defect ipecacuanha is free. On the other hand tartar emetic produces more diaphoresis.

Tartar emetic.—This may be given either in powder or in solution in water.

The nauseating dose is $\frac{1}{4}$ to $\frac{1}{2}$ a grain every hour until the nausea is evoked, when the dose should be reduced. It is better given in solution rather than in powder; 4 grains may be dissolved in an ounce of water, and half a teaspoonful to a teaspoonful given as required.

Ipecacuanha has the advantage over tartar emetic in that it causes no intestinal irritation. It is said, moreover, to produce, besides the fall in blood pressure, marked pulmonary anæmia, which tartar emetic does not. It must be given in full doses to establish toleration, and with this object it is best combined with opium. A decoction may be made by boiling 120 grains in 6 ounces of water, and giving half an ounce (10 grains) every quarter of an hour till nausea is

excited, when the dose should be reduced or given less frequently. If the powder be used, one or two grains may be given every quarter to half an hour, but the decoction is more convenient. No doubt both these remedies cause much discomfort, and on that account, perhaps, they are less popular than they were.

Aconite has been also advocated on the ground that while reducing cardiac action it also lowers pressure in the pulmonary artery. However, it is a drug that is hardly ever employed for hæmoptysis nowadays.

Venesection.—The object of venesection is to produce at once the fall in blood pressure to which profuse hæmorrhage leads, and upon which cessation in great part depends. For this purpose the bleeding must be very free, and venesection can, therefore, only be indicated when the danger to life is very great, that is to say where the hæmoptysis threatens to suffocate the patient.

It requires some courage to bleed a case of hæmoptysis freely, and I have never had occasion to do it, but Huggard¹ records some favourable experiences of it.

Derivative methods, i.e., methods the object of which is to relieve the vessels of the lung by detaining or turning aside the blood into other channels. This might be achieved by dilating some of the great vascular systems of the body, such as those of the skin or abdomen, and thus providing in them a temporary reservoir where the blood could be stored up while the rest of the vessels were depleted.

a. Derivation to the skin.—In order that dilatation of the cutaneous vessels should have any effect upon the circulation in the lungs, it must be either general, or, if local, involve a large area.

Local dilatation may be produced by means which are more or less mechanical, *e.g.*, Junod's boot, or by local irritants.

Junod's boot is an apparatus rarely seen nowadays, and still more rarely used. It is a large metal case, made to hold the whole leg, and looking like a boot. Round the thigh it is closed by a tight-fitting indiarubber membrane, by which it is made air-tight, and then the air is pumped out of the interior. In this way the vessels and lymphatics of the leg are made to swell. Indeed, Junod's boot is nothing else than a gigantic dry cup.

The same result may be arrived at by a ligature placed round the thigh, or even on both legs and arms. They must not be put on too tightly, for all that is required is to compress the veins, and if too tight they cause so much pain that they have to be removed. I have used such ligatures occasionally, I think with benefit.

Cupping.—Dry cups between the shoulders and over the lower part of chest is said to do good at times, but the objection to their use is that their application disturbs the patient. They are little used now, nor are wet cups.

Counter irritation.—The old-fashioned application of hot cloths and hot water to the legs and feet, and mustard poultices to the thighs or calves, is not so irrational as might appear, for it produces great local vascular dilatation, and if this be extensive enough it will have a marked effect upon the circulation elsewhere.

Diaphoretics.—Of drugs which act upon the skin *pilocarpin* seems the most promising, but has been little used in hæmoptysis.

Nitrate of Pilocarpine might be administered in doses of one-twelfth of a grain *sub cutem* every two hours or so, or by the mouth in somewhat larger doses. Its action must be watched lest it cause faintness or collapse. My own experience of the drug is small, for I have always felt the great objection to its use to be the risk of causing effusion into the air-tubes, as it sometimes does even in small doses.

b. Derivation to the abdomen.—The use of mild laxatives has been referred to with the object of diminishing the straining and effort to which constipation leads, but the object now in view is different, *viz.*, to dilate and keep dilated the vessels of the intestinal tract.

¹ *B. M. J.*, 1893, Jan. 23.

For this purpose the more irritating or *drastic purgatives* are necessary, and of these jalap is the best. This effect upon the intestines may be one of the indirect advantages of the use of tartar emetic, but on account of the difficulty in controlling its action in full doses, antimony is less reliable than jalap.

✱ The injection of *large enemata of hot water* into the bowel has been employed with advantage, and it acts probably by producing general vascular dilatation within the abdomen.

Dieting.—The general management of the diet has been already discussed. In this place dieting has to be further considered as a means of cure. It is only to remittent hæmoptysis, *i.e.*, to those cases in which profuse hæmoptysis frequently recurs, that this method of treatment is applicable. Such cases, depending as they so often do upon the rupture of a pulmonary aneurysm, may be treated upon the principles advocated by Tufnell for other aneurysms, *viz.*, reduction of food and absolute rest. The diet should be reduced to as small an amount as the patient can exist upon; say for the adult 10 to 12 ounces of solids and 10 to 15 ounces of fluid; for instance:—

Fluids, $\bar{3}$ xii.			Solids, $\bar{3}$ xii.		
Milk, . . .	$\bar{3}$ v.		Meat (pounded or minced), . . .	$\bar{3}$ iv.	
Water, . . .	$\bar{3}$ v.		Potatoes, . . .	$\bar{3}$ ii.	
Tea, . . .	$\bar{3}$ ii.		Bread, . . .	$\bar{3}$ iv.	
			Egg, . . .	$\bar{3}$ i.	
			Butter, . . .	$\bar{3}$ i.	

Where the hæmoptysis is free it is not easy to reduce the amount of fluid so low as 12 ounces on account of the thirst which the bleeding causes. The thirst may be assuaged by lemon or orange juice or a little acid, and the amount of fluid should be then reduced to as small a quantity as the patient can get on with.

This method of treatment requires to be continued for three to six weeks, according to the severity of the case, but it has appeared to me on several occasions to yield very marked results, and where hæmoptysis is continued, I should strongly recommend its adoption.

Theoretical considerations.—In discussing this question it will be best to start from that form of profuse hæmoptysis which is not only the commonest, but also that about which our knowledge is greatest, *viz.*, the profuse hæmoptysis of phthisis. In this case, as in all others where the hæmoptysis is profuse, we know that the cause lies in gross lesions of large or fairly large vessels, and the question to be answered is, By what means can bleeding from such vessels be best controlled?

General treatment.—The general treatment of profuse hæmoptysis must be the same as that of profuse hæmorrhage from other parts of the body. Rest, absolute, of the body as a whole, and of the part diseased so far as possible, is the main essential principle; and, with this object, the patient will be kept in the semirecumbent position, speaking will be prohibited, cough checked, and excitement avoided, or, if present, controlled by drugs. So far as these conditions can be fulfilled by drugs, they will, for the most part, be met by the use of opium, which is as useful in most cases of profuse hæmoptysis as in profuse hæmorrhage from other parts.

Hæmostatics.—The action of the so-called hæmostatic remedies is, in the face of the present pathological views of hæmoptysis, most difficult to understand. The hæmostatics fall into two groups—the topical astringents, and the vascular constrictants.

The topical astringents.—Chief among the former group are perchloride of iron, alum, gallic and tannic acid, and acetate of lead; but, powerfully as these remedies act when applied directly to the bleeding surface, it is difficult to see how they can produce the same local effect when administered by the mouth. If the remedies of the first group really do act at all, it must be like those of the second group, by vascular constriction.

The vascular constrictants.—Of this second group, the prominent remedies are digitalis and ergot. Both of these drugs produce contraction of the peripheral arteries; and, if hæmoptysis were due to capillary oozing, they might, by contracting the vessels, cut off the blood from the capillaries, and control the hæmorrhage; but knowing, as we now do, that profuse hæmoptysis

is not due to capillary oozing, but to gross lesions of fairly large vessels, the usefulness of these remedies becomes open to grave question.

The effect of constricting the peripheral arteries would be, in great measure, to increase the quantity and pressure of the blood in the arteries of larger size; but, as the lesion is nearly always in these vessels, this is the very place where the least blood and the lowest blood pressure is wanted, and we might expect, *a priori*, that these remedies would increase rather than check hæmorrhage. Digitalis, at any rate, is admitted, I think, now to be of little or no service, unless it be given in doses large enough to make the patient sick, and then its action becomes that of a nauseant and depressant rather than of a stimulant.

The great efficacy of ergot in uterine hæmorrhage probably accounts for its introduction as a remedy for bleeding elsewhere; but ergot acts not only upon the vessels, but also upon the muscular tissues of the uterus, in which the vessels run such a course that its contraction leads to their closure by mechanical compression. In the lungs, however, there is no contractile tissue so placed that it can act in this way; and if ergot act upon the smaller vessels only, the same objections arise to its use that have been already urged against the use of digitalis.

If, then, the pathology of hæmoptysis seems to discredit many of the drugs upon which reliance has been long placed, it will be well to consider whether it gives any other more promising indications for treatment.

When hæmorrhage from a surgical wound ceases, the result is due, first, to closure of the vessels by contraction of the muscular coat; and, secondly, to the clotting of the blood, aided, and in the case of large vessels rendered possible only, by the great fall in the blood pressure which profuse hæmorrhage induces. In most cases of profuse hæmoptysis the muscular coat at the seat of hæmorrhage is so diseased that it has lost all power of contraction, and the effect of vascular constrictants acting upon the peripheral branches would be to dam the blood up, and so increase the bleeding. The first indication cannot, therefore, be met.

Nor can the second, for there is no drug which can be relied on to increase the clotting power of the blood; and though most of the astringents have this effect when applied directly to the seat of hæmorrhage, we have no evidence that they do so when administered by the mouth. Hence we must trust to the intrinsic clotting power of the blood determined in the required place by the lesion of the vessel, and by contact with the surrounding tissues.

The only drug known to have any power to increase the coagulability of the blood is *chloride of calcium*, but its efficacy in hæmoptysis is far from being proved. It has been given in doses of 25-45 grains every four to six hours, or a dose of 30 grains at once, followed by doses of 5 grains every quarter of an hour for two or three hours.

The third indication we may, however, endeavour to fulfil in various ways by imitating those effects upon blood pressure and circulation which severe hæmorrhage naturally induces. What is required is, that the circulation in the affected portion of the lungs should be as slow as possible, in order to give the blood time to clot, and that the blood pressure should be as low as possible, to give the clot formed time to consolidate *in situ*. These effects we may endeavour to obtain by acting upon the vessels, or upon the heart, or upon both combined.

As the chief danger of hæmoptysis is due to the immediate local effects of the hæmorrhage, rather than to the loss of blood, and as profuse hæmorrhage leads to the conditions of circulation most favourable to its cessation, it would seem that, in free blood-letting from artery or vein, we possess a means of rapidly inducing the conditions we require. Copious bleeding was, in fact, a much-vaunted remedy of past times for all severe internal hæmorrhage; and, though probably rightly but little employed in the present day, still theoretically it would not be out of place where the dangers are as imminent as in profuse hæmoptysis, or in apoplexy; for by no other means can the same rapid and sure result be produced.

But without free blood-letting, which can only be available in such very special cases as those just indicated, an attempt may be made to reach the same end, not by removing the blood from the body, but by detaining it in some part of the body distant from the seat of hæmorrhage. This may be done mechanically, as by Junod's boot or an elastic bandage round the limbs.

In the vessels of the cutaneous and abdominal systems, we have reservoirs large enough to contain a great portion of the blood of the whole body, and capable, therefore, of producing a considerable effect upon the blood pressure and circulation, if we possess the means of calling them into action. This may be the explanation of the use of free purgation, for the mere loss of fluid is not sufficient to account for the benefit which frequently results from it. I do not know that diaphoretics have been much employed in hæmoptysis, but it is possible that they might be of advantage in some cases, though, with the exception of pilocarpin, their action is, as a rule, too slow.

In pilocarpin and nitrite of amyl we have drugs which produce a very considerable vascular dilatation throughout the whole body, and these remedies may be found of service, though I do not think they have been thoroughly tried. Nitrite of amyl is a cardiac stimulant as well, but its action is very transient, and on both these accounts it would appear to be inferior to pilocarpin. The objection which might be urged against both of these remedies, namely,

that they dilate, not the vessels of the abdomen or of the skin only, but of the whole body, and therefore of the lungs as well, is not, I think, insuperable; but the value of these drugs must be settled by actual observation.

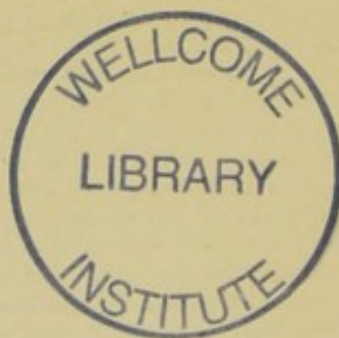
In counter irritation we have a means of producing vascular dilatation in the skin; but, to be effectual, it would seem necessary that it should be very extensive, unless its action be not mechanical but reflex.

By the remedies above referred to an attempt is made to fulfil the conditions indicated upon the circulation by means of the vessels. We may now turn to those which produce the same effect through the heart—to the group of cardiac depressants as well as to those of nauseating or depressant emetics.

Of the cardiac depressants, the only one that has been freely used is tartar emetic. This must be administered until marked depression is produced, and it is a remedy, therefore, which requires to be carefully watched. Of the nauseants, ipecacuanha has been highly vaunted. Trousseau used it largely, and praised it highly, quoting Baglivi, who, 150 years before, wrote of it as a specific, "*Radix ipecacuanhæ est specificum et quasi infallibile remedium in fluxibus dysentericis aliisque hæmorrhagiis.*"

One other method of treatment remains for consideration, to which sufficient importance is hardly attached, at any rate in writings upon hæmoptysis, *i.e.*, Dieting. It is an old rule of practice in hæmoptysis to place the patient upon a restricted diet. Knowing the close relation between the lungs and stomach which exists through the nerve-supply, it is not irrational to expect a detrimental effect upon the lungs by an overloaded or irritated stomach, and the harm which experience proves to arise from these causes is usually referred to reflex action. But careful dieting is not only useful because it avoids the risks which improper feeding may introduce, but because, when rationally employed, it becomes a real means of active treatment of the diseased condition.

END OF VOLUME I.



[NOTE.—For the Contents of Volume II. see the analytical table at the commencement of this Volume. The Index to the whole work will be found at the end of Volume II.]











