

vessel itself, until they are thoroughly distended with red blood-corpuscles, and hence appear to the eye as a red spot having a wedge shape. In another form of infarction, a diseased vessel giving way, the blood enters a bronchus, and is drawn up into the lobules, distending them. This differs from the other form in appearance; it is less dark in color, is irregular in outline, and is shaded off into the surrounding normal tint.

Pulmonary apoplexy is a hæmorrhage which breaks up and infiltrates the lung, and is usually due to traumatism, to gunshot injuries and contusion, to the rupture of aneurisms, to gangrene, etc. Bronchial hæmorrhage arises from primary and secondary causes. The primary causes are of an irritative kind, and induce congestion: prolonged exertion of the voice, mechanical straining, inhalation of irritating gases and fumes, etc. An abnormal weakness of the vessel-wall inherited; that state of the circulation which exists in the subjects of hæmophilia, the so-called "bleeders"; the condition of the vessels in young subjects of the strumous type, are factors in the production of bronchial hæmorrhage. The most important of the causes is tuberculosis. As has been stated elsewhere, the initial change in the development of pulmonary tubercle is a proliferation of the connective-tissue corpuscles of the adventitia; and, although the multiplication is chiefly outwardly, the media and intima are weakened. Hæmorrhage may therefore be an early symptom of tubercular deposit. In the extension of the tubercular deposit a vessel may be invaded at any time. A large hæmorrhage may result from the opening of a vessel by erosion in the process of softening and formation of cavities, or by the development of an aneurism on a vessel in the wall of a cavity. The vessels still pervious are subjected to a much greater pressure by reason of the closure of so many, and hence this increase in the vascular pressure enters into the question of hæmorrhage. The suppression of an habitual discharge has long been supposed to cause pulmonary hæmorrhage, but this is no longer admitted. The menstrual flow may take place vicariously by the bronchial mucous membrane, as it does by various channels. A substitution is very different from a vicarious hæmorrhage.

Pathological Anatomy.—Hæmorrhage may be caused by a diapedesis of red-blood globules, and hence no solution of continuity can be detected under such circumstances. Even when there has been a considerable hæmorrhage, the source of it may elude the most painstaking investigations. If the examination is made immediately after a hæmorrhage, there will be found both fluid and coagulated blood, drawn up into the bronchioles and alveoli, and through the larger tubes. In consequence of violent struggles for breath, in the case of large hæmorrhage, the inspiratory efforts draw up a good deal of blood into the lungs, distending them, so that they overlap the heart and do not collapse. They present a mottled appearance, because of the filling of

many alveoli with blood. The mucous membrane of the bronchi may be congested or reddened by patches of extravasation, or of a dull-red by imbibition of blood, or uniformly pale from anæmia, according to the causes producing it and the source of the hæmorrhage. The infarction presents a most characteristic appearance: it is wedge-shaped, with the base outward, and is, when small, just under the pleura; when large, nearer the root of the lung. Infarctions vary in size, from a pigeon's to a hen's egg, or may even occupy a half or nearly the whole of a lobe. They are found more frequently in the inferior part of the lower lobe. If under and next the pleura, they appear as dark-blue masses, projecting somewhat above the general surface of the lung, which just about the infarction is pale and exsanguine, while the pleura is roughened by exudation, confined to the infarction. Sometimes effusion occurs in the pleural cavity, which contains flocculi of membranous exudation, and is red by admixture with blood. When a section is made through an infarction, it appears as a dark, reddish-blue, well-defined mass, from which some dark, reddish-brown liquid and granular matter may be pressed. Fibrinous exudation, distending some of the alveoli, gives to the otherwise smooth surface a granular aspect. At first firm and elastic, the infarction soon becomes friable. The surrounding pulmonary tissue is more or less hyperæmic and œdematous. An infarction may undergo several kinds of change: the blood may disintegrate, the fibrin become granular and fatty, and the corpuscles break up into fat-granules; absorption may take place in part, extrusion in part, and recovery ensue, the elasticity of the lung remaining impaired to some extent. Recovery may ensue in part only: the lobules collapsing and inflammation occurring in the connective tissue, a brownish-red indurated mass remains; or, after an imperfect absorption of the blood and inflammatory exudation, the remaining reddish, pulpy mass solidifies by infiltration with calcareous salts, or, merely inclosed by a limiting membrane, a cyst remains—a process only resembling hæmatoma of the dura mater. Or, again, inflammation may result in suppuration, an abscess forming; or, finally, the whole may become gangrenous. Pulmonary apoplexy not unfrequently forms a blood-mass of considerable size, the blood breaking up the pulmonary elements and diffusing into the surrounding parts, in part coagulating. If next the pleura, this membrane may be perforated, and the blood, entering the cavity, produce a hæmothorax.

Symptoms.—It is but rarely that a hæmorrhage occurs in full health without the least intimation of its approach. In this way may the onset of pulmonary disease be announced. Usually there is a sense of heat and oppression of the chest, which those recognize who have experienced former attacks, or there may be general vascular fullness, headache, vertigo, palpitation of the heart, a quick, strong pulse, etc. The signs of pulmonary disease precede the hæmorrhage, in

a majority of cases, rather than succeed to it. At the moment the attack is experienced, there are a sudden cough, a warm feeling under the sternum, and a mouthful of fluid, tasting both saltish and sweetish, comes up. Cough now succeeds cough, and with each effort a teaspoonful or more of blood, somewhat frothy, or, if in large quantity, bright—red blood and somewhat darker clots, are discharged. Even with a small amount of blood, the moral effect of the blood-spitting is so great that much depression, paleness of the face, and a weak pulse result. If the loss be great, there will come on the subjective sensations of fainting, and actual syncope will happen. If the hæmorrhage is great, the blood will come up with a sudden gush, spurting from the nose as well as the mouth. If a fatal hæmorrhage, the blood will pour out of the mouth and nose, there will be gurgling in the fauces, frantic efforts at respiration, a deadly pallor will overspread the face, and, with a general convulsion in which the breathing ceases, all is over, but the heart will beat for a minute longer. The expectoration of blood does not cease with the arrest of the hæmorrhage; for some days subsequently dark-brownish coagula will be brought up, with some rather viscid mucus. The source of the hæmorrhage may not unfrequently be determined by the moist *râles* heard in the bronchi. The signs and symptoms of infarction have already been mentioned under the head of embolic pneumonia, so that it is necessary only to mention that, when an infarction of sufficient size is formed, the symptoms are sudden dyspnoea and the physical signs of consolidation.

Course, Duration, and Termination.—There are great variations in the amount and duration of pulmonary hæmorrhage. The whole course may be concluded in a few hours. The expectoration may go on during several days, from a tea- to a tablespoonful being spat up each time, and the hæmorrhage in the aggregate amounting to several pounds, causing great depression and a tedious convalescence. In other cases, there may be a number of large hæmorrhages, occurring after an interval of several days, the arrest being due to syncope, and the hæmorrhage recurring when sufficient blood has been made to produce it. Such cases may continue for several weeks, the system being much reduced and the convalescence very protracted. In cases of hæmorrhage with infarction there will follow a period of inflammatory reaction, the expectoration will continue bloody for a week or ten days, and, if the area of tissue involved is small, recovery will ensue, and convalescence will be established in about ten days. The reader is referred to embolic pneumonia for further details in respect to this group of cases. An ordinary croupous pneumonia may be accompanied by considerable hæmorrhage, which occurs with the initial hyperæmia, when the pneumonic process may be confounded with the results of hæmorrhage. The debility caused by pulmonary hæmorrhage is quite disproportioned to the actual loss. A few tea-

spoonfuls may induce fainting and an unexpected degree of anæmia. Any considerable loss will be followed by pallor, weakness, breathlessness on slight exertion, palpitation, etc., and the restoration of the blood will require several weeks or months. The moral effect of the hæmorrhage and the association of ideas connected with the bleeding are in part responsible for the depression, but more is due to the fact that, in most cases, the system is already enfeebled by a dyscrasia. To this important element is also due the prolonged condition of anæmia—the slow reproduction of the red blood-corpuscles.

Diagnosis.—In every case of doubt, the mouth, fauces, and nares should be carefully examined. Is it vicarious hæmorrhage? The patient is a female, the hæmorrhage occurs at the menstrual epoch, and takes the place of the menses, or nearly so, and no untoward results are experienced, nor does any evidence of pulmonary disease exist. In many of these supposed vicarious hæmorrhages it will be found that the subjects are of the phthisical type, and that, if the physical signs are wanting, there are suspicious rational symptoms. In these cases, it usually happens that the menstrual flow does not return, and that phthisis rapidly develops. Hæmoptysis is to be differentiated from hæmatemesis. In the latter, the blood is black, contains no air, has an acid reaction, is mixed with articles of food, and is vomited; in the former, the blood is bright red, contains air, has an alkaline reaction, and is coughed up, while there is no nausea. If the blood of pulmonary hæmorrhage is swallowed, it will present the characteristics of blood derived directly from the stomach, but the distinction is then made by observing that some of the blood is coughed up, and has the ordinary character of blood derived from the lungs. It should be noted that blood swallowed may pass away with the stools. Hæmoptysis is accompanied by *râles* in the chest, and preceded in the largest number of cases by symptoms referable to the chest; hæmatemesis by symptoms referable to the stomach.

Prognosis.—It is very rare indeed for the life to be put in jeopardy by a pulmonary hæmorrhage. If the patient is much reduced, a severe hæmorrhage may materially hasten a fatal result. Hæmorrhage proceeding from a cavity is more unfavorable than a bronchial hæmorrhage, for the vessel may bleed again and again, since any coagulum, which in other situations might close it, will here be readily detached. The prognosis must be guarded when the subject of the hæmorrhage is much reduced and the quantity lost is considerable. In a case of supposed vicarious hæmorrhage, the probability of a rapid development of the pulmonary lesion should not be forgotten.

Treatment.—The management of cases of hæmoptysis includes the treatment of the hæmorrhage and of the conditions on which the hæmorrhage depends. If the subject be a plethoric one, and there is much oppression from fullness of the vascular system, bloodletting

may be practiced, either by venesection or by application of a dozen leeches. These are, it must be admitted, rare cases.

The most effective of the remedies acting dynamically are ergot and barium chloride—the former given subcutaneously and the latter by the stomach. Fluid extract of ergot may be given internally, combined, if desirable, with digitalis and opium—with digitalis if the action of the heart is rapid and excited, and with opium if there is a troublesome cough. Ipecac is, next to those mentioned above, one of the most efficient hæmostatics. Its utility has been disputed on theoretical grounds, but not by those who are practically acquainted with its real advantages. Ipecac produces an exsanguine condition of the lung, and arrests hæmorrhage, also, by the enfeebling effect of nausea on the heart. It is even successful in stopping *post-partum* hæmorrhage. Besides its hæmostatic effect, one advantage of its use consists in mechanically clearing the alveoli of retained clots. Ipecac should not be prescribed in those cases of hæmorrhage from a cavity, the difficulty of keeping a clot in the position necessary to close the vessel being already great. The most suitable form for the use of ipecac is the fluid extract, which may be combined with ergot, digitalis, and opium if desirable. Tincture of veratrum viride may be used with great advantage to keep down the action of the heart. Ice has a similar effect to these dynamical hæmostatics; it slows the heart and contracts the arterioles. It should be applied to the chest, especially to the nape of the neck. The alternate application of heat and cold is usually more effective than the continuous cold. A sponge dipped in hot water can be applied first, then an ice-bag, and so on alternating—the heat remaining in contact but a few minutes, while the cold is kept applied the rest of the time. Absolute rest is an agent of the same kind. The patient should maintain a recumbent posture, and not exert a muscle if he can exercise such restraint. All emotional disturbances should be avoided as well. There are remedies called astringents which are supposed to possess hæmostatic powers, such as tannic and gallic acids, acetate of lead, alum, and the mineral acids, especially sulphuric. These are decidedly inferior to the remedies above named, yet they are freely used, especially the acetate of lead in combination with opium. That they are serviceable, an immense experience confirms, but they do not deserve the very great confidence reposed in them by many practitioners. In cases of debility, characterized by relaxation of tissue, or in examples of the hæmorrhagic diathesis, or in cases of purpura, oil of turpentine is highly useful. Inhalations, by the atomizer, or spray douche, of a solution of Monsel's salt (subsulphate of iron) or the chloride of iron, will sometimes arrest a violent hæmorrhage at once. This undoubted fact is all the more difficult of explanation, since but little, very little, of the iron salt can pass the chink of the glottis, and none of it can reach the point of disease in the lung. Tannin in solution may be em-

ployed in the same way, but the iron spray is distinctly better. In administering iron spray great care must be exercised to protect the teeth and the clothing, which may be permanently stained. A mouthful of common salt is a domestic remedy, which may be used until more efficient means are available. Counter-irritants are serviceable. A mustard-plaster or a flying-blister is sufficiently active, or a turpentine liniment, the latter being useful also because of its vapor. Good results may be obtained by inhalation of the vapor of turpentine disengaged for this purpose in those cases appropriate for its internal administration. If the hæmorrhage has shown a disposition to recur, the recumbent position, quietude of mind, and the remedies employed to check it, if not objectionable, should be continued until all possibility of danger has passed.

HYPERÆMIA AND ŒDEMA OF THE LUNGS.

Definition.—*Hyperæmia* signifies an abnormal increase in the blood-supply, which may be active or passive. *Œdema* is usually a consequence of hyperæmia, but it may be due to causes producing general œdema. The term signifies the presence of serous fluid in the alveoli, the intervening connective tissue, the perivascular lymph-spaces, etc.

Causes.—There may be an increase in the amount of blood going to the lungs, the result of increased pressure in the arterial system, from greater force of the heart's contractions, or from narrowing of the arterial field elsewhere, throwing an additional quantity on the lung. Undue exercise of the vocal apparatus in speaking or singing, the inhalation of cold, or very warm air, or the sudden transition from one extreme of temperature to the other, and the inhalation of irritating gases or vapors, are causes determining congestion of the lungs under favoring circumstances. The form and character of the chest and the existence of a constitutional vice or dyscrasia are necessary to bring about the results from the operation of such causes, especially the type of chest and the bodily conformation of phthisical subjects. The ingestion of cold drinks, the body in a warm and perspiring state, will sometimes induce extreme congestion of the lungs. The sudden impact of cold air or cold water on the surface will more surely produce the same result, since a larger surface of the capillaries is made to contract, forcing the blood within. One part of the lung, the seat of a disease obstructing the circulation in it, will necessarily throw on another part an excess in its supply; pneumonia, atelectasis, and obstruction in some branches of the pulmonary artery, are examples. Passive congestion is produced by causes interfering with the return of blood from the lung; mitral stenosis and insufficiency, aortic stenosis and insufficiency, and obstructive lesions maintaining venous stasis, are examples. A weak heart may produce the same result by insufficiency

in propulsive power, and hypostatic congestion results from such a state of adynamia that the blood simply obeys the force of gravity. Œdema is a result of congestion, whether active or passive, or a local effect of the causes producing a general dropsy.

Pathological Anatomy.—When the lung is congested it is heavier, contains less air and more blood, and crepitates less than is normal. The color is darker and redder; on section it is found to contain more fluid in the interstices, more blood flows out from the divided vessels, and the bronchi are injected and filled with a sanguinolent, frothy serum. In chronic cases the congestion is considerable, the color of the affected portions is dark red, almost blackish red; the interstitial connective tissue is distended with serum, the capillaries are so swollen as to compress the alveoli, almost or quite obliterating the cavity, and numerous extravasations are found through the parenchyma. So firm and dark becomes the tissue of the lung as to resemble the appearance of the spleen, whence the term *splenization* to characterize this condition. In the dependent portions of the lungs of the very adynamic or of aged persons confined to a recumbent position, a serous fluid, having considerable viscosity, exudes, giving to the lung on section a somewhat granular aspect, whence the term *hypostatic pneumonia*. In œdema there is a serous infiltration into the interstitial connective tissue and in the alveoli, which may be sufficient to distend the lung and afford pitting on pressure. On section of the lung under these circumstances, a quantity of serum flows out; the serum is reddish when there is much congestion associated with the œdema. When œdema of the lung coexists with general dropsy, the fluid that exudes is colorless, and the tissue of the lung is pale. The dependent and inferior portions of the lungs first become œdematous; thence it spreads to the superior and anterior portions as the fluid increases in amount. As a result of congestion of the passive kind, due to disease of the mitral valve, the lungs generally become denser, more resistant, and are much increased in size. The color, externally, varies from a reddish-yellow to a brown, and on section its texture is found to be firm, to crepitate but little, to exude blood very freely, and not only blood, but, on pressure, to exude a yellowish or brownish fluid. While the general color of the divided surface is yellowish-red or brownish-yellow, there are spots interspersed having a brownish almost blackish color—whence the designation *brown induration*. Some of these brown spots are very dense, and sink in water.

Symptoms.—A sudden and complete congestion of both lungs may be a cause of sudden death. Between this extreme and a simple unilateral congestion of slight extent, there are numerous gradations in the severity of the seizures. In the mildest cases there occur a sense of internal heat, oppression of the chest, some slight difficulty of breathing, a flushed face, a strong, full pulse, beating of the carotids, and in-

jection and brilliancy of the eyes. When the congestion is sufficient to cause universal œdema of the alveoli, the symptoms are formidable. There are great difficulty and extreme rapidity of breathing, a strong sense of oppression, intense anxiety, rapid and violent action of the heart, beating carotids and pulsation in the temples, headache and fullness of the head, a flushed face, a hasty and troubled cough, and expectoration of a frothy liquid which may be tinted with blood.

On percussion the resonance of the lungs is but little altered—slightly diminished, with a tympanitic quality. The vesicular murmur is supplanted by sub-crepitant and mucous *râles*, which are very abundant and very loud. If the alveoli are filled with fluid, the sonority will be still more diminished, and the respiration will have a blowing character approaching bronchophony. If the alveoli are filled to that degree that the oxygen can not reach the blood, accumulation of carbonic acid must take place, and hence there will be blue lips, a livid face, headache, etc. When this condition is reached, there will be still greater anxiety and oppression, the breathing will be shallow and exceedingly hurried, the pulse will decline in volume, and at length will be merely thready and intermittent, the surface of the body will be cold and covered with a clammy sweat, the fingers will be blue and cold, and with the accumulation of carbonic acid there will be increasing somnolence, replacing the extreme restlessness, deepening into coma. With the increasing stupor there will be less and less effort at cough and expulsion of the fluid accumulating in the bronchi, and an increasing difficulty of breathing from this cause. In the cases of passive congestion of the lungs, due to cardiac disease, there are difficulty of breathing, cough and oppression, constantly present, and paroxysms of extreme dyspnea, in which the patient labors for breath, the face is cyanosed, the extremities cold and blue, the skin cold and covered with a clammy sweat, the pulse, small, weak, and irregular, the jugulars swollen, the mind clouded, etc. The severity of these attacks will be greatly increased if œdema come on suddenly; but if the œdema is gradual in forming, the difficulties of breathing will be slowly augmented, and carbonic-acid poisoning will also be slowly developed. The physical signs in cases of hypostatic congestion will indicate the existence of bilateral lesions if the decubitus is dorsal; or unilateral, if the decubitus is to one side. The sonority is diminished, or dullness with a tympanitic quality exists. On auscultation, the vesicular murmur will be weak, or supplanted by moist *râles*. The difficulty of breathing which arises during chronic Bright's disease is due to œdema of the bronchial mucous membrane—an interstitial œdema and swelling of the terminal bronchi.

Course, Duration, and Termination.—An acute congestion of the lungs may pass through its whole course and prove fatal within a few hours. The usual duration is from three to five days, and the termi-

nation may be by resolution, occasionally by hæmorrhage, and rarely by inflammation or pneumonia. The passive form associated with cardiac disease develops slowly, and is subjected to great variations; to periods of improvement under appropriate treatment; then exacerbations. Acute œdema may come on, and prove quickly fatal in acute, or chronic kidney affections.

Diagnosis.—Active congestion is to be distinguished from the stage of engorgement in pneumonia. The points of difference are: in congestion there are no chill, no pain in the side, and not the range of temperature of pneumonia. The subsequent course separates the two diseases more widely. Œdema occurring during hyperæmia is announced by dyspnœa, by the auscultatory signs of the presence of fluid in the terminal bronchi, and by the expectoration of a frothy, serous, and reddish fluid. The hyperæmia of a passive kind produced by valvular lesions is accompanied by rational and physical signs, which make the diagnosis merely a question of the recognition of these signs.

Treatment.—Active congestion in a plethoric subject may demand bloodletting, if not by venesection, by the application of cups or leeches to the chest. A ligature to the thighs applied merely firmly enough to retain the blood in the superficial veins is a useful expedient when the abstraction of blood may seem to be necessary. Counter-irritation in the form of a large mustard-plaster should be applied to the chest, and the feet should be put in a hot foot-bath. As the removal of the fluid in the alveoli and terminal bronchi is of the utmost necessity, an active emetic should be prescribed; of these apomorphine subcutaneously is probably the best, and next, the sub-sulphate of mercury. Stimulant expectorants should be prescribed to procure the expulsion of the fluid by expectoration. Squill, senega, and serpentaria are appropriate remedies for this purpose. To diminish the viscosity of the fluid, and thus secure its easy expulsion, the iodides, especially the iodide of ammonium, are highly serviceable. The iodide and carbonate of ammonium in sirup of senega is an excellent combination to secure the rapid and easy extrusion of the fluid present. In the œdema of cardiac disease and renal dropsy, digitalis and squill are very important remedies. If the blood is much impoverished, iron is indicated, especially the iodide of iron, which is a rapidly acting and an efficient chalybeate. When there is hypostatic congestion, changes in the position of the patient are very necessary, and the propulsive power of the heart must be increased by stimulants, quinine, and small doses of opium. In the cases of brown induration, the iodide and carbonate of ammonium should be persistently used together, with means to increase the energy of the heart, such as turpentine, eucalyptol, and alcoholic stimulants.

ATELECTASIS.

Definition.—This term means a collapse of the lobules, so that the cavity disappears and the walls approximate. Congenital atelectasis is the state in which the lungs are before being dilated with air (fœtal lung).

Causes.—The congenital condition is simply a failure to distend the alveoli. The whole lung may be in such a state, or only a part of it, in a premature child, or one so weak at full term as to be unable to expand the lungs fully, and hence some of the lobules or alveoli remain in a state of atelectasis. The acquired atelectasis is the collapse of lobules that have been expanded. A terminal bronchus may be closed against the admission of air by a plug of mucus which, acting like a ball-valve, permits the exit, but not the entrance, of air, so that gradually all the residual air is expelled, and then the sides approximate, and the cavity is closed—in other words, it has collapsed. This result is the more apt to occur in the case of feeble, ill-nourished, and ill-developed children, who are attacked with such troubles as measles, whooping-cough, etc. Collapse of lobules—of a large part of a lung, indeed—may be induced by pressure on a bronchus, of an aneurism, of enlarged bronchial glands, tumors, etc. The air remaining in lobules, to which the access of air is cut off, is gradually absorbed by the blood. Direct pressure may also cause atelectasis—such direct pressure as is made by hydrothorax, empyema, hydropéricardium, aneurisms, tumors of the thorax, and effusions in the peritoneal cavity, sufficient to push up the diaphragm.

Pathological Anatomy.—Seen from without, those portions of the lung in the atelectatic condition have a bluish-red color, or grayish, and are depressed somewhat below the general surface of the organ. These parts have a greater density than the healthy tissue, and, as they do not contain air, do not crepitate on pressure, and they are tough and not easily broken up. When divided, but little blood flows out, nor do they contain any kind of fluid, and appear smooth instead of granular. When inflated with air, as freshly atelectatic lung can be, an immediate change in color ensues, the lobules become pink, and crepitate on pressure as normal lung. If, however, they continued collapsed, changes of a nutritional kind ensue, and, after a time, dilatation can not be effected. When congenital, this condition is found to exist in the posterior and inferior parts of the lungs, in the apices and anterior borders, and may be limited to individual lobules, or a considerable part of a lobe may be affected. When atelectasis is acquired, usually isolated lobules, or small groups of lobules, are thus affected, they are more or less thickly disseminated through both lungs, and the superficial portions are first attacked, the deeper parts subsequently. This acquired atelectasis differs from the other in that the