—pneumonia sudden, with a rigor, and pain in the side—catarrhal pneumonia with an ordinary bronchitis, and a feeling of soreness rather than pain under the sternum ; pneumonia, as a rule, is unilateral, self-limited, terminating by crisis, or ceasing within three weeks —catarrhal pneumonia is bilateral, not limited nor terminating by crisis, and indefinite in duration ; if double, which is rare, pneumonia is limited to a portion of either lung, while catarrhal pneumonia is diffused over both. The differentiation of bronchitis and croupous pneumonia rests upon the same points. In respect to physical signs, the differences are marked : In pneumonia, the vocal fremitus is increased, and there is increased resistance on palpation—in bronchitis, the vocal fremitus is unaffected, and there is no change in the resistance ; in pneumonia, there is dullness on percussion—in bronchitis, the percussion-note is unaltered ; in pneumonia, on auscultation, there is audible the crepitant *râle*, which disappears and is replaced by bronchophony—in bronchitis, there is no crepitant but a sub-crepitant *râle*, followed, not by bronchophony, but by sub-mucous and mucous *râles*. The *râles* in pneumonia or the bronchophony are audible at the seat of inflammation only—in bronchitis, they are diffused over the chest. An uncomplicated pneumonia differs from a pleuro-pneumonia in the following particulars : In pleuro-pneumonia there is more acute pain, a friction murmur as well as a crepitant *râle*, displacement of the heart and of other organs by the fluid, more absolute dullness on percussion, and less of the tympanitic quality to the percussion-note. Cases of pneumonia with cerebral symptoms may be mistaken for meningitis, but this can only happen should the chest not be examined. In pneumonia of the aged, and, in some cases, in subjects of delirium tremens, there may be no cough or other rational symptom to direct attention to the chest.

Treatment.—The constitutional tendencies, the actual state, and the surrounding circumstances should receive careful attention in deciding on a plan of treatment. A vigorous, healthy subject, free from constitutional vice, will require and bear a more vigorous handling than a broken-down alcoholic. If seen at the beginning, during the stage of congestion, there can be no doubt as to the good effects of venesection or the local abstraction of blood. The author, for the most part, has not been in favor of blood-letting, but his experience has taught him that systemic or local bleeding may be of the highest service in cases of pneumonia. He has seen no cases of late years that could not bear from five to ten full-sized leeches, and many that were improved by venesection of two to eight ounces. Whether or not the abstraction of blood being decided on, the next question is the use of calomel. On this point, also, the author's opinions have been modified by more extended observation. No cases of pneumonia are seen in these latitudes that are not accompanied by considerable hepatic disorder. There is a more intimate relation between hepatic

disorder and pneumonia, I believe, than has been made out as yet. A high degree of overirritation and a temporary increase of productivity is a nearly constant or quite constant condition of the liver in pneumonia. The external evidences are the intense yellowness, or the jaundiced hue of the skin of the conjunctivæ and elsewhere. If, instead of overaction, it is a disturbance in which excretion from the pancreas, liver, and intestinal glands is the cause, the indications afforded thereby are equally in the direction for the action of calomel. Modern researches have shown that calomel has a sedative action on the liver, lessens bile production, and probably the whole functional activity of the liver. It may be that, as the blood in the hepatic veins is so much higher in temperature than in the portal, the reduction in the febrile heat is due to the lessened functional activity of the liver. It is clear that calomel has an antipyretic action. Hence, at the outset, it should be given in a dose of from three to five grains. The repetition of the dose depends on the result of the first ; but as a rule from two to four doses are required—on the first, second, and third days of the actual inflammation.

In cases accompanied by depression, or with light sthenic reaction, a few cups or leeches may be applied, and small and frequently repeated doses of the tincture of aconite-root (two to five drops every two hours) may be given. A large mustard poultice should be put on the chest, and removed when the skin is reddened, to obtain its stimulant effect on the vaso-motor nerves within, and the feet should be immersed in a hot mustard foot-bath. If the viscid secretion is pouring out in the air-sacs and bronchioles, and coagulating, it is necessary to use some agent which possesses the power to lessen the viscidity and coagulation. Hughes Bennett employed the potassa salts (liquor potassii citratis) or an extemporaneous solution of the bicarbonate, and his results were admirable. Ammonia, originally suggested by Richardson, has been latterly used more freely than potassa, and, as the author believes, with better results. Probably the most advantageous method of administering it is the solution of the carbonate in liquor ammonii acetatis (℥ ss.—gr. v to x) every three or four hours. By the German school the muriate is preferred in corresponding doses, but it does not appear to the author to be so useful. The ammonia solution should be continued up to the crisis. As soon as consolidation of the lung is accomplished, all arterial sedatives of every kind should be discontinued.

Assuming that pneumonia is a specific disease, like typhoid, Juergensen maintains the necessity for the use of antipyretics, among which he places the cold bath first ; and the success of his treatment certainly seems to justify his theory. He demonstrates that there is no danger in putting a pneumonic patient in a bath, and that the reduction of temperature by it exercises a favorable influence over the

progress of the disease. Next to the bath, quinine is most useful as an agent for reducing fever, but it must be given in scruple doses every four hours until the temperature falls to a proper point, when it may be suspended until the temperature rises again in twenty-four to thirty-six hours. The new antipyretic—antipyrin—has proved to be so certain and powerful a means of reducing febrile heat, that it may be resorted to in cases where the temperature approaches the condition of hyperpyrexia, or instead of quinine as suggested above for this purpose.

The author feels it necessary to emphasize the evil effects of cardiac sedatives during the stage of exudation and of coagulation of the exudate. The administration of veratrum viride, digitalis, aconite, and tartar emetic, can only add to the burden of the heart, already laboring in consequence of the stasis on the venous side, and lack of blood on the arterial side. Paralysis of the heart is one of the most imminent dangers, because of this state. It is true that a continued high temperature contributes to bring about paralysis of the heart, but we possess the means of correcting this by the administration of quinine, and by cold baths or the cold wet pack. While arterial and cardiac sedatives are to be avoided at the stage of red hepatization, it is necessary also to avoid the immoderate use of alcoholic stimulants. These are needed, and in full doses in inebriates at the period of crisis, and when the stage of purulent transformation is reached if there are a rapid and weak pulse, a relaxed and clammy skin, and delirium. Protracted wakefulness and delirium need careful management. Opium or morphine must be avoided, owing to the state of the pulmonary circulation, and the collateral hyperæmia and œdema. Then it is that chloral hydrate serves a most useful purpose; it procures sleep, quiets delirium, and has a good effect on the exudation. Care must be exercised, for large or frequently repeated doses may cause paralysis of the heart; fifteen grains at night, with ten more in two or four hours, if the first dose is insufficient, is all that is required usually. Aliment must be carefully administered from the beginning, without waiting for depression to come on. Beef-juice, milk, egg-flip, wine-whey, chicken or mutton broth, etc., should be systematically administered every three hours. In weak subjects, a little wine may be given from the beginning. As already stated, the pneumonia of the inebriate requires alcoholic stimulants from the first symptom—for the delirium accompanying it is due largely to the sudden withdrawal of the supply, or the inability to retain it. Much has been said about the blistering-point in pneumonia. Counter-irritation is useful during the stage of congestion, as already indicated, but a fugitive counter-irritant, as a mustard-plaster, is all that can be properly used. When the crisis occurs, a blister is very useful. During the stage of red hepatization, turpentine-stupes, cotton wadding, or a flannel jacket, is useful unless the temperature is very high, when they do mischief. Fly-

ing-blisters are serviceable in promoting absorption, when resolution is imperfect and exudations still linger at the site of inflammation. To facilitate absorption in chronic, succeeding to acute pneumonia, the iodide of ammonium is highly beneficial. It may be administered with the iodide of iron, and in conjunction with the hypophosphites. If there are "prune-juice" expectoration, weak pulse, relaxed and sweating skin, turpentine in small doses, or eucalyptol, is extremely useful. During gray hepatization, they may be given for the double purpose of acting on the organ by which they are eliminated, and as cardiac stimulants.

EMBOLIC PNEUMONIA—PNEUMONIA FROM EMBOLISM.

Definition.—By *embolic pneumonia* is meant an infarction of the lung, due to embolic blocking of a vessel.

Causes.—From the right cavities of the heart, or from some part of the venous system, an embolus is dislodged, and, entering the current of the blood, is deposited in a branch of the pulmonary artery. The circumstances under which clots form in the right cavities of the heart have been set forth elsewhere.

Pathological Anatomy.*—The emboli which give rise to embolic pneumonia are of two kinds, simple or non-infective and infective. The former act in a merely mechanical manner by closing the vessels and preventing the passage of blood to the parts supplied by them; the latter not only obstruct vessels like the former, but the infective material contained in them sets up a local infectious process. The size of the embolus, and consequently the capacity of the vessel obstructed, varies considerably, the resulting infarction being from a pea to a hen's-egg in size. If a simple embolus, the damage is confined to the area occupied by the infarction; but, if an infective embolus, a suppurative inflammation arises and an abscess is the result. To the formation of an infarction it is necessary that the embolus lodge in a terminal artery of Cohnheim—an artery without anastomoses—for, if the obstructed artery is connected by branches with others, the circulation in the obstructed area may be restored through collateral channels. If the obstructed artery be a terminal one, as are those of the outer part of the lung in a restricted sense, the pressure in the veins causes a gradual filling of the obstructed vessels through the capillaries. Now, as the walls of these obstructed vessels are not properly nourished by the blood thus in a state of stasis, the blood diffuses through into the surrounding textures, which constitutes the infarction. Such an infarction is not often possible at the root of the lung, for here the anastomoses are too numerous, although they do sometimes occur;

* In the account of this process, Cohnheim's classical work, "Untersuchungen ueber die embolischen Processe," Berlin, 1872, Hirschwald, is followed.

but it is at the periphery that they usually form. As the vessels proceeding from the root of the lung toward the periphery divide dichotomously, it is obvious that, when an embolus obstructs one, the resulting infarction must be wedge-shaped—the base of the wedge being toward the periphery of the lung, or outwardly. If a section be made through an infarction, its outline will be seen rather sharply defined, its color of a deep blood-red, and it will exude blood on slight pressure. If it has been formed for some time, its structure is denser from an infiltration of the alveoli, whence it presents a granular appearance; it is dark-brownish in color, is drier, and exudes but little blood, and is very friable, easily breaking up into a pulverulent mass. The bronchi contain a frothy, bloody fluid. The tissue of the lung about the infarction becomes hyperæmic and œdematous. The pleura overlying it is deeply congested, or it may be inflamed and coated with a firmly adherent albuminous exudation, while the cavity contains more or less bloody serum. The infarction undergoes various changes; the blood is gradually transformed, becomes fatty, and is absorbed, although patches of altered hæmatin remain; the proper tissue of the lung undergoes atrophy, the connective tissue multiplies, and in this way a cure is effected, the lung being rendered useless to the extent of the infarction. In other cases an embolic abscess is produced, the embolus being infective; but it does not have a wedge-shape; it is globular, and presents the appearance of an ordinary purulent collection. In rare cases an infarction becomes gangrenous. Infarctions are found more frequently in the right lung.

Symptoms.—As the embolus proceeds most frequently from the right side of the heart, the clinical history is that of some cardiac disease; but it may be produced in some distant part of the venous system under circumstances which favor thrombosis. The prominence and urgency of the symptoms will depend on the size of the infarction. If it be small in extent, there may be no disturbance; even if quite large, the symptoms may be masked by the coexistent disease. If a large branch of the pulmonary artery be suddenly closed, there will be acute dyspnœa of extreme severity, the patient will gasp for breath, become deeply cyanosed in a few minutes, and, may be, die at once. Sudden difficulty of breathing is the most significant symptom at the time of lodgment of the embolus, especially if there is nothing in the condition of the heart to account for the dyspnœa. Fever comes on some days after the obstruction, but the rise of temperature is not very great. There may be chills, but they are not constant, except in the case of pyæmia. Bloody expectoration appears in a few days after the initial dyspnœa, and is usually inconsiderable in quantity. Besides blood, there is a viscid mucus which is the body of the sputa, and, as it adheres rather tenaciously, a good deal of coughing is necessary to bring it up. Pain begins with the implication of the pleura, and has

the usual characteristics of pleuritic pain: it is acute and lancinating, and is increased by the movements of respiration. There are present the usual physical signs of consolidated lung—dullness on percussion, bronchial voice, and bronchial breathing. There may be a friction-sound due to the pleuritis, and also the evidences of effusion into the pleural cavity. It is obvious that the diagnosis of embolic pneumonia is difficult and uncertain. The sudden occurrence of dyspnœa, followed by bloody expectoration continuing eight or ten days, and the evidences of consolidation, are the only symptoms to indicate the real nature of the malady. If the history furnished the source of the embolus, the diagnosis would be proportionally facilitated. The prognosis is generally unfavorable, notwithstanding small infarctions may get well. There is no plan of treatment which can affect a mechanical condition of this kind, unless ammonia may dissolve an embolus. This should be tried.

CATARRHAL PNEUMONIA.

Definition. — Various terms have been applied to this disease, as *capillary bronchitis*, *lobular pneumonia*, *broncho-pneumonia*, etc. As right views with regard to it are necessary to a proper conception of pulmonary consumption, it is discussed here somewhat in advance of its proper position. By the term *catarrhal pneumonia* is meant a catarrhal inflammation involving the bronchioles and alveoli. It may be *acute* or *chronic*.

Causes.—Catarrhal pneumonia may be an extension downward of a catarrhal process beginning in the bronchial tubes. It is probable that a catarrhal inflammation never begins, under any circumstances, in the alveoli. Typical examples of this disease occur during certain of the exanthemata, notably measles and whooping-cough. It is intimately associated with certain diatheses, as rickets and scrofula, and with structural alterations of the heart and lungs, as mitral lesions and emphysema. It is frequent in early life and in old age, and is less so at the period of greatest bodily vigor. Bad hygienic influences as to dress, habitations, humidity, and exposure, favor its development. Climate is an important factor, and the period of most extreme variations is the period of greatest prevalence of this disease.

Symptoms.—The acute form is the type; the chronic differs from it merely in duration and severity of the symptoms.

The initial symptoms are chilliness followed by fever, soreness of the chest, chiefly beneath the sternum, cough, and expectoration of a frothy mucus, and some difficulty of breathing. These symptoms in the acute form of the disease quickly develop into the more serious and characteristic proper to catarrh of the finer bronchial tubes. An abundant secretion, poured out all along the bronchial tree, must greatly affect the functions of the lungs. The breathing soon becomes rapid,

superficial, and labored, the accessory muscles of respiration are brought into play, and the alæ of the nose work quickly and continuously ; the face is at first flushed and rather animated, and the eyes have a glaring expression, but the lips soon become bluish and cyanosis spreads over the face. The cough in the first onset is rather loud and bronchial, but, as the finer tubes become involved, it has more of a stridulous, husky character, and is often suppressed and partial because the difficulty of breathing is too great to permit the necessary expansion of the chest. The cough is also painful, and in children is attended with moans and crying, and they make attempts to restrain it because of the soreness in the chest. The fever soon rises to the maximum of 104° to 105°, and is nearly continuous, there being a slight morning remission. As the difficulty of breathing develops, there is increasing restlessness, never a moment of quiet, the struggle for breath and the search for an easier position being incessant. At first there are brief snatches of uneasy sleep, but, as the dyspnœa increases, a state of somnolence comes on which gradually deepens into coma, so profound at length that cough is suppressed. This somnolence is due to the deficient aëration of the blood and the accumulation of carbonic acid. Finally, the blood becomes wholly venous. Then the flush disappears from the face and is replaced by a death-like pallor, the cyanosis deepens about the lips, blue spots appear on the cheeks, and the superficial veins grow into thick black cords. The struggle for breath continuing, while the carbonic-acid poisoning increases, the most frantic but largely automatic efforts are made to remove supposed obstructions, and the patient, a child, may tear its skin about the neck and face with its nails, in the vain effort to remove them. On inspection, the cervical and other muscles auxiliary are seen actively engaged, and a deep depression of the abdomen from retraction of the lower ribs is made with every strong inspiration. On palpation, the vocal fremitus will be unaffected during the first few days, but, when the lobules have collapsed in considerable numbers, the physical conditions are changed, and the vocal fremitus will then be increased. On auscultation, *râles* are abundant all over the chest ; they consist of sub-crepitant *râles*, which are somewhat coarser and louder than the crepitant, and are audible with both inspiration and expiration. With these also occur mucous and sub-mucous *râles*, produced in the larger tubes. The respiratory murmur becomes more and more feeble as the condition of atelectasis is produced ; and, when a number of lobules are thus affected, over them the respiratory murmur ceases to be audible, a blowing sound is substituted, and this passes into bronchial breathing and bronchophony as the pulmonary tissue becomes consolidated. On percussion there is no change until the atelectasis occurs ; the sonority is diminished as the lobules collapse, until dullness is reached ; but the dullness has much of the tympanitic quality, owing to the proximity of

unobstructed alveoli. In making percussion in children, it is important to strike lightly, otherwise the primary bronchi and trachea will be thrown into vibration. The pulse-rate does not always correspond to the range of temperature ; it is usually higher. The pulse ranges from 140 to 200 or more in children, while in the aged it may be but little accelerated. Protracted high temperature may induce changes —parenchymatous degeneration of the cardiac muscle. If, therefore, during the course of this disease the pulse becomes feeble, irregular, and very rapid, the condition of the heart is one to arouse great solicitude. The appetite is poor, vomiting often occurs, and diarrhœa is by no means infrequent. The embarrassment to breathing caused by the act of eating and swallowing induces young children to avoid eating solid food, although they will often drink greedily. Cerebral symptoms are present to a greater or less extent in all cases : there may be headache, hallucinations, muscular twitchings, even convulsions, and the coma of carbonic-acid poisoning. So closely do the nervous symptoms belonging to catarrhal pneumonia simulate those of tubercular meningitis that it may be exceedingly difficult to diagnosticate between them. In the chronic or, rather, subacute form of catarrhal pneumonia the development is slow, the fever of moderate intensity, and the difficulty of breathing not pronounced. If there has been an attack of acute bronchitis, or of whooping-cough with more or less extensive bronchitis, when the catarrhal pneumonia develops, the cough subsides, but the depression of the vital forces, the cyanosis, and the extreme emaciation, indicate the growth of the more serious lesions. When these cases tend toward a fatal termination, the grave symptoms just mentioned increase, and carbonic-acid poisoning comes on, death occurring in more or less profound coma. Some cases pursue a different course ; after a protracted subacute period in which the pulmonary lesions begin, an acute attack arises, and then the subsequent behavior is that of an ordinary acute case, death occurring in coma. When they tend to recovery, there is a gradual improvement in all the symptoms : the cyanosis diminishes, the dyspnœa lessens, the appetite improves, and gradually the general health is in part restored, the lungs imperfectly repaired.

Pathological Anatomy.—The changes involve the bronchial tubes and the lungs. The mucous membrane is the seat of an hyperæmia from the larynx down, but it increases in severity downward, reaching the maximum at the most dependent part of the lungs. The vessels are so deeply injected that the mucous membrane is a dark red, and at various points there are extravasations. The finer tubes are filled with a quantity of yellowish, creamy, purulent fluid. On section of the lung, drops of this exudation, escaping from the tubes, look just like pus escaping from a small abscess, especially if the divided tube has undergone dilatation—a change which takes place in the more pro-

tracted cases. This pus is probably made up of the young cells derived by multiplication of the epithelium, but especially of the lymphoid cells which migrate from the vessels, and are found in the sub-mucous connective tissue, in the alveoli, and in the bronchioles. There are two opinions now entertained in respect to the cellular elements which crowd the alveoli, and as to the part taken by the pavement epithelium. Among others, Rindfleisch maintains that these cells are produced by the multiplication of the epithelium, and derived in part from the proliferation of the lymphoid cells ; others, again, notably Buhl, deny the participation of the epithelium, and maintain that the products of the catarrhal inflammation are drawn into the alveoli by a species of suction. Besides the changes in the mucous membrane, the bronchial tubes and intervening connective tissue take part. The bronchioles become dilated if they have been long subjected to the inflammation, and the connective tissue undergoes hyperplasia, attaining to very considerable development. The formation of the very viscid exudation which takes place at the beginning of the process and the swelling of the mucous membrane are important elements in the collapse of the lobules (atelectasis) which is a conspicuous result in the sum of pathological changes. The collapse of the lobules takes place before the alveoli which form them are crowded with the products of the catarrhal inflammation. The mechanism of the collapse is about as follows : In the strong efforts in coughing or in expiration, or both, the air is forced out through the swollen tubes ; and, when the air has passed, the surfaces are brought into contact, and are made to adhere tenaciously. All of the residual air is gradually expelled in this way ; but, in the efforts at inspiration, the force is insufficient to separate the adherent surfaces, and, as the pressure is immediately increased in the adjacent lobules, the collapsed lobule is also compressed. The collapsed lobules are easily recognized by their appearance, which is of a dark-blue or purplish-blue color ; they are much firmer, do not crepitate, because they contain no air, and exude but little blood on section. The extent to which this process is carried varies in different cases. It begins in the most dependent part of the lungs, and advances forward and upward, involving much, sometimes the whole, of the lower lobe. In some chronic cases the process takes place chiefly in the upper lobes. Collapse of some lobules, the pressure continuing the same, necessarily involves the dilatation of others, and in this way emphysema results, the anterior portions of the lungs being affected chiefly. Attacks of catarrhal pneumonia in early life, imperfect repair only taking place, have much to do with the subsequent development of emphysema. After the lobules have collapsed, for a short period they continue permeable to air and may be inflated. The change in color and density which occurs when the collapse is effected is often mistaken for inflammation—whence the term "lobular pneumonia." If

the collapse continue, an inflammatory process is set up, similar to but not identical with that of croupous pneumonia, for it never becomes granular. The inflamed part becomes more solid, is of a dark-brown color, which terminates in grayish red ; it begins in the center of the lobules and spreads outwardly ; neighboring lobules affected in the same way coalesce, until ultimately a whole lobe may be involved. Then it presents to the eye, when the process is completed, a bluish-gray appearance ; on section it is found to be homogeneous, very firm, and tough. Before this final stage is completed it is very friable. The purulent matter in the bonchi and the catarrhal products in the alveoli undergo the cheesy transformation. The subsequent history is that of "caseous pneumonia." Those portions of the pleura in contact with the inflamed lobules become hyperæmic, inflame, an exudation is poured out, and adhesions form, or effusion takes place in the thoracic cavity. Not every case tends to death, or to the chronic changes above described. Partial recovery ensues in a considerable number, complete recovery in but few. When the collapsed lobules inflame, unless there be but few, restoration seems hardly possible even in the sense of a partly useless lung. If the lobules are capable of being distended again with air, and the catarrhal inflammation subsides in the bronchioles and alveoli, a cure is then possible. The purulent contents of the bronchi are brought up by coughing, and swallowed or expectorated ; the watery portion of the exudation in the alveoli is absorbed ; the cells disintegrate, become granular and fatty, and are ultimately absorbed—thus restoring the alveoli to the admission of air. The fluid and the cells of the intervening connective tissue pass through the same process, and thus the injured part is restored, except that its elasticity continues impaired for a long time.

Complications and Sequelæ.—The complications are really parts of the malady in its entirety. Bronchitis is always present, and laryngitis frequently. Pleuritis is a necessary result when the peripheral portion of the lung is involved. The sequelæ are very important. As was indicated under the head of pathological anatomy, there are two diseases which result from catarrhal pneumonia—emphysema and caseous pneumonia. The former is a result of the atelectasis or collapse of the lobules ; the latter is an outcome of the changes in the catarrhal products which crowd the alveoli, in the bronchi themselves, and in the intervening connective tissue. In the account to be presently given of these diseases, the course of development from one to the other will be set forth.

Course, Duration, and Termination.—The course of catarrhal pneumonia is from a catarrh of the larger tubes to a catarrh involving the ultimate bronchioles, and probably the alveoli. There are two principal phases in the subsequent course : the development of the catarrhal process ; the collapse of the lobules, and the transformations which