be freely washed out with antiseptic precautions. Although the admission of air in cases of empyema is not sought to be prevented, nevertheless the air should be deprived of its germs of putrefaction.

As death has occurred several times very unexpectedly after the operation of thoracentesis, certain precautions are necessary. When the effusion is large, the whole amount should not be withdrawn at once, for the sudden removal of the pressure might induce a quick outpouring of fluid, or the great vessels, relieved of pressure, would overdistend the right cavities, or the heart, moving from its position, might cause compression of some of the vessels. Sudden death might very unexpectedly be caused by any of these accidents, notwithstanding the operation of thoracentesis is simple, not painful, and is otherwise free from danger. After the removal of the liquid exudation by absorption or by thoracentesis, a quantity of solid and semi-solid remains behind and is very slowly transformed. A succession of flying-blisters, painting with the tincture of iodine, and friction of the affected side with ointment of the red iodide of mercury, are the most effective external or topical applications. The best results are obtained, not from the use of supposed stimulants of the absorbents, but from means to promote the nutrition. The iodide of iron (sirup), cod-liver oil, extract of malt, and a generous diet, the digestion stimulated by bitters and mineral acids, are the best means for increasing absorption. The amount of fluid taken should be reduced to the minimum ; for, although the restrictions imposed in a "dry diet" may be too rigid for ordinary patients, yet they can submit to a considerable reduction of the fluid. Absorption is promoted by lessening the water of the blood, which can be accomplished by saline laxatives and jaborandi. The laxatives should not be given so as to interfere with digestion, and a daily dose of jaborandi can be so administered as not to interfere with the appetite or exercise. To procure complete distention of the lung, and to promote the oxygenation of the blood, compressed air should be inhaled daily, or a sojourn in an elevated, dry mountain-region should be enjoined. Although we may not agree with Dr. Leaming, of New York, and Dr. Andrew Clark, of London, in the importance of pleuritic exudations as a factor in phthisis, we must admit that they exercise some influence in initiating the process of fibroid degeneration and of tuberculosis.

HYDROTHORAX—DROPSY OF THE CHEST.

Definition.—By the term *hydrothorax* is intended an accumulation of watery fluid in the chest. It differs from pleuritis in the character of the fluid and in the state of the pleura. In pleuritis the effusion is an inflammatory exudation, and the pleura is the seat of an inflammation ; in hydrothorax the fluid transudes—a merely physical process—and the pleura is unaffected except by maceration.

Causes.—The various conditions giving rise to general dropsy will cause hydrothorax—cardiac and renal diseases. Local obstruction to the course of the circulation produces pure hydrothorax, i. e., hydrothorax not a part of a general dropsy. The most important of these local causes are emphysema and sclerosis of the lung, tumors so situated as to compress the vena cava, vena azygos, the right auricle, etc. A general dyscrasia may induce hydrothorax, as Bright's disease, chronic malarial poisoning, etc. The most influential factor is the condition entitled by the older authors *latent pleurisy*. In this malady there is a state of the pleural membrane closely allied to pleuritis—to that form known as *dry pleurisy ;* but instead of a plastic exudation there is an abundant outpouring of serum.

Pathological Anatomy.—When the hydrothorax is due to any of the causes producing general dropsy, the effusion is bilateral, but usually more abundant on one side. There will be found associated with the hydrothorax the anatomical changes in the lungs, heart, and kidneys, proper to the particular form of dropsy. The fluid has a pale sea-green color, is transparent, and frequently coagulates on exposure to air, the coagulation consisting in the formation of an excessively fine reticulation of the minutest fibers. In the case of the so-called latent pleurisy the membrane is thickened, congested, and coated usually with a pellicular exudation, portions of which are, to a greater or less extent, floating in the fluid. The amount of serum present is from half a pint to two or three gallons. The effect of the fluid on the position of the heart and other organs is precisely the same as in pleuritis. The retraction of the lung and its subsequent compression also take place, as in pleurisy, except that it occurs more regularly.

Symptoms.—In latent pleurisy, so called, there is some pain felt in various parts of the chest, but it is not acute and well defined as in pleurisy. It is usually situated in the side, and is a rather dull, tensive, heavy pain, or a feeling of soreness. It is increased by a full inspiration, or by coughing, but is not so severe as to interfere with daily duties ; and it is often transient, and makes so little impression on the mind as to be forgotten until attention is directed to it. There is some feverishness toward evening, but not much attention is paid to it, and hence it is usually overlooked. The cough may be rather troublesome, especially on lying down, but the expectoration is nothing more than frothy mucus. Often these symptoms pass unnoticed, and the first thing which attracts attention is an increasing difficulty of breathing. In the cases of hydrothorax pure, without pleural inflammation, there is no fever, nor pain in the side, and the first symptom referable to the thorax is difficulty of breathing greater than in pleurisy, because the effusion is on both sides. In latent pleurisy, the left side of the thorax is involved in two thirds of the cases ; consequently the heart is pushed over to the right, and the semilunar space is oblit-

erated. In hydrothorax there is no displacement of the organs, because of the effusion on two sides and in the abdominal cavity. The physical signs are much the same in hydrothorax as in pleurisy; but in the former there can not be that complete filling of the cavities, and hence there must be a considerable space of both lungs where the voice and breath sounds remain unaffected. Furthermore, in hydrothorax, there being no limitation of the effusion by neo-membrane and by adhesions, the fluid gravitates with the changes of position, and the area of dullness shifts accordingly. The course, duration, and termination of hydrothorax are those of the disease on which it depends. The formation of a large effusion in the chest adds to the severity of the case, and is not unfrequently a cause of death. This is especially true of dropsy, whether cardiac or renal. The hydrothorax is a source of extreme distress when it may not prove fatal, for the patient is unable to lie down, or to make any muscular effort without experiencing a suffocative attack. The author has witnessed a case of sudden death from hydrothorax in an aneurism of the arch of the aorta which was solidifying. The behavior of latent pleurisy is that of the sero-fibrinous form of acute pleurisy, when sufficient fluid has accumulated to produce symptoms by compression.

Treatment.—If there is large effusion, delay is unsafe and thoracentesis should be promptly performed. As serum will flow through a fine capillary needle, but little pain and no danger attend the operation of aspiration. If the effusion is not sufficient to produce distress by pressure, the treatment is directed to the condition on which the dropsy depends. The treatment for latent pleurisy is the same as for acute pleurisy with effusion. As the inflammatory symptoms are usually overlooked, the physician is not consulted until the difficulty of breathing comes on, and then the sole question is, aspiration or not. The rules for guidance are the same as those already laid down.

PNEUMOTHORAX—HYDROPNEUMOTHORAX.

Definition.—The presence of air in the cavity of the thorax is called *pneumothorax;* of air and fluid, *hydropneumothorax.*

Causes.—Air or gas of any kind is rarely present in the cavity without liquid, and if air alone should enter an exudation would soon be excited. It is now settled that a serous membrane can not secrete air, and that, therefore, if air be found in the cavity of the pleura, it came there from without, or is a gas the product of decomposition or fermentation. Almost always it enters from without by perforation of the pleura, by the lung, or by the wall of the thorax. The most frequent mode of entrance of air is the giving way of a superficial cavity of the lung, tubercular or caseous. Very rarely the air passes through a communication made by a gangrene patch, or a hæmorrhagic infarction, and still more rarely by the giving way of emphysematous alveoli. Abscesses of the liver ulcerating through the diaphragm may form a secondary purulent collection in the pleural cavity, which may communicate through the lung with a bronchus, constituting pyopneumothorax. One of the modes of termination of a purulent pleuritis is by a fistulous passage to a bronchus, through which air is admitted to the pleura. Suppuration may occur in neighboring organs in a way to involve the pleura and thus secure some outlet. For example: suppuration of bronchial glands, bursting into the pleura and ulcerating into a bronchus; abscesses of the liver or of the kidney, perforating the diaphragm and the lung, etc. Traumatism is an important factor, pyopneumothorax being caused by penetrating wounds, incised or gunshot, the air entering from without.

Pathological Anatomy.—The accumulation of air in a given case is much influenced by the formation of the orifice of communication. If the entrance is easy and the exit difficult, a very large amount of air may accumulate, and very often a sort of valvular arrangement, a fibrinous flap or plug, may exist at the orifice which has this effect. The lung quickly retracts until there is an equilibrium of the pressure; compression is then exerted on it if the orifice is such that the air which entered without obstruction can not escape. The quantity of air which can be contained in the cavity depends on several conditions; on the compressibility of the lung, which may be slight, owing to solidification by caseous or tubercular deposits; the degree in which the other organs can be shoved aside; the amount of liquid present, etc. It is a mixture of gases, not air, usually found in the cavity—of nitrogen and carbonic acid, and but little oxygen, with some sulphuretted hydrogen if there be unhealthy pus present. If atmospheric air enters, the pleura inflames, and sero-purulent, then purulent exudation is poured out. As air contains the bacteria of decomposition, they are the active agents in exciting purulent inflammation; but, as, in pneumohydrothorax, ichorous, ulcerating, or decomposing materials pass in under the usual circumstances, these play a considerable part in exciting inflammation, though probably less than the air and its contained germs. The exudation which results from the action of these noxious matters is purulent, often ichorous and bloody. The gas is contained in the space above the liquid, and the lung, having had the air squeezed out of it, lies flattened against the spine, unless old and firm adhesions resist the compressing forces. If there be much fluid, that side of the thorax will be enlarged, the intercostal spaces prominent, the diaphragm depressed, the heart pushed aside, etc. In some rare instances adhesions form in a circle between the two pleural surfaces, making a central cavity in which gas and fluid will accumulate to a large extent, a fistulous communication having been established with a bronchus.

Symptoms.—Pneumothorax is to be studied in connection with the

diseases from which it arises. It may develop insidiously, so that it is discovered only on making physical examination of the chest. But, when a perforation occurs suddenly, pronounced, even formidable, symptoms are at once produced. Perforation may be announced by a condition almost of collapse, a temperature of 97° Fahr., and a small, weak, but very rapid pulse. If the temperature does not descend so low, the pulse is weak and rapid, and the respirations are hurried—the former reaching so high as 140, the latter up to 40, even 60. At the same time dyspnœa sets in with orthopnœa, and a severe pain, due either to sudden stretching of the pleura or tearing apart of adhesions. In other cases, for example phthisical subjects, none of these severe symptoms are produced, probably because narrowing of the respiratory field has been going on so long as to prepare them for this additional discomfort. The decubitus varies, the largest number seeking a position on the diseased side to permit the freest possible play of the healthy lung; but a considerable proportion lie upon either side, although, when air first entered the cavity, orthopnœa was experienced by

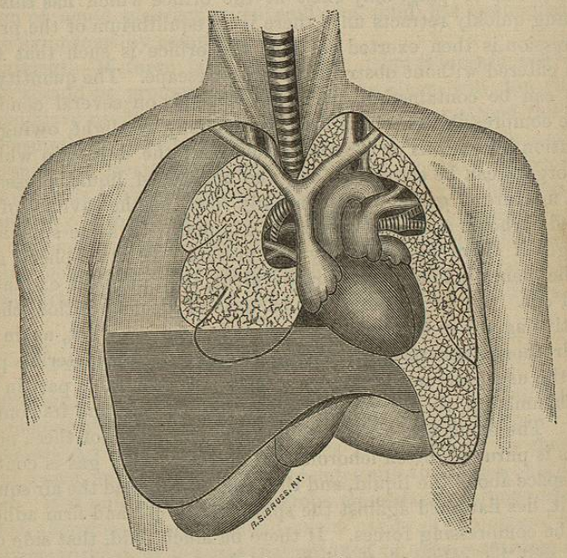

FIG. 28.—Hydropneumothorax.

most of the cases. The dyspnœa is due to several causes—to sudden compression of the lungs and the heart, and to a compensatory congestion, often with œdema of the other lung, whence the expiratory force is lessened and the voice weak and trembling. Cyanosis appears if

there is much difficulty of breathing, the surface becomes cold and covered with a cold sweat, the tongue is blue and cold, and death soon closes the scene; or, if life continues, general œdema supervenes from the venous stasis, while the arterial tension is low from ischæmia of the arteries. The lessening of the expiratory force makes the cough weak and ineffectual, and the expectoration diminishes. The low state of the arterial tension affects the urinary secretion, which is dense and red, with traces of albumen. The vocal fremitus may be present, diminished, or absent, in pneumothorax—present when there are strong bands of adhesion which communicate the vibrations to the chest-walls; diminished when the lung is not entirely collapsed; absent when the cavity is distended with gas. On palpation, also, increased resistance will be noted while there is fluid, and increased tension with diminished resistance where there is gas. The percussion-note is characterized by its marked tympanitic quality, resonance, and elasticity. The resonance is not limited to the part containing air, but extends downward to the lower margin of the ribs, extinguishing the hepatic dullness in its usual limits, and the semi-lunar space on the left side, and also extends across to the middle of the sternum. A peculiar metallic echo may be developed on strong percussion. Percussion over the fluid produces the usual dull sound which sharply contrasts with the metallic clang of the percussion over air, and the dullness here varies with the position of the patient and follows the gravitation of the liquid. The character of the percussion-note is affected by several circumstances: when thick, false membrane lines the thoracic wall it acts as a damper, and there is much less of the tympanitic and metallic quality; when an external opening exists, there will be produced the cracked-pot sound. On auscultation, there is no respiratory sound, except a modified, amphoric, blowing sound. All of the sounds audible in the chest—cough, *râles*, heart-beat, etc.—take on a distinct metallic quality. The dropping of fluid, or coughing, or movements of the body, produce under these circumstances *metallic tinkling*. But the most characteristic of the physical signs is *succussion*—a splashing of the liquid against the walls of the chest, produced by a sudden shake of the body. It is best heard by applying the ear to the chest, and then suddenly shaking the body by the hand placed on the patient's shoulder. The patient often recognizes this sound, and soon learns the best movement to produce it. It is like the splashing of liquid in a half-empty barrel.

Course, Duration, and Termination.—The course of pneumothorax is much influenced by the associated lesions and the extent of the pulmonary insufficiency. If, already, the respiratory field is much narrowed, death may ensue in a few hours or days. Death is more frequently produced by the secondary pleuritis and its products, causing slow failure of respiration after some weeks. A cure is not to be expected in cases, the most numerous, due to perforation of a superficially

placed cavity. Pneumothorax resulting from an incised wound in a healthy subject may get well after some weeks. A perforation occurring in the first stage of phthisis is not so important as one occurring later, and a cure is possible in the former before the constitutional forces are much depressed by the progress of the phthisis. A pneumothorax, produced by the discharge of a purulent pleuritis by a bronchus may get well after some months. It may be stated in general that the prognosis of pneumothorax is unfavorable, since very few cases get well even in the modified way of a permanent fistula.

Diagnosis. — Pneumohydrothorax may be confounded with the large caverns of phthisis, with dilated bronchi, with emphysema, with pleuritis having limited effusion. Vomicæ are confined to the upper part of the lung, have formed slowly without any sudden symptoms ; they present amphoric sounds and metallic tinkling, rarely succussion ; vocal fremitus is not lessened ; the chest-walls are retracted instead of distended, and the heart is not displaced. In pneumohydrothorax, loud, deep, tympanitic percussion-note is obtained all over the affected side ; the symptoms have occurred suddenly, and consist of severe pain, dyspnœa, and orthopnœa ; well-marked succussion ; vocal fremitus lessened or absent ; the intercostal spaces bulging instead of retracted ; heart and other organs displaced. Emphysema is bilateral ; the respiratory murmur not absent ; bronchial *râles* audible all over the chest ; vocal fremitus present. Pneumohydrothorax is unilateral ; the respiratory murmur entirely absent, and all voice and breath sounds and *râles* from the affected side wanting when the lung is collapsed ; vocal fremitus absent. In pleuritis, with effusion, the percussion-note has a tympanitic quality in the infra-clavicular region ; the dullness on percussion changes with the positions of the patient, and corresponds to the height of the liquid; an amphoric murmur is exceptionally audible over the root of the lung and at the summit ; with the increase of the distention of the chest, there is absolute dullness over the whole side ; no metallic tinkling, no succussion. In pneumohydrothorax, the percussion-note has a loud, ringing, tympanitic quality all over the chest, instead of a modified normal at the infra-clavicular region, and this tympanitic note is not supplanted by absolute dullness ; there are metallic tinkling and succussion in perfection.

Treatment.—As respects the condition associated with pneumothorax and pneumohydrothorax, the treatment is indicated under the head of these maladies, and need not now be discussed. If there are much dyspnœa and danger of acute asphyxia, no time should be lost in making a free opening to permit the exit of air. The pyopneumothorax is to be treated by incision and the drainage-tube, and the use of antiseptic injections, of which iodine appears to the author to be the best. The severe pain requires the use of anodynes, unless the free exit of air procured by incision relieves the distress. The congestion and œdema of the sound lung may be relieved by ligatures to the thighs, by which a considerable quantity of venous blood can be retained in the lower limbs long enough to bridge over the period of danger. This expedient is preferable to bloodletting, which has been recommended for this purpose.

PNEUMONIA—PNEUMONITIS—INFLAMMATION OF THE LUNG.

Definition.—*Pneumonia*, an acute inflammation involving the alveoli of the lungs, is designated by the German writers "croupous pneumonia," and by the French writers "fibrinous pneumonia." "Catarrhal pneumonia" differs from the fibrinous or croupous form in the seat and character of the inflammation. It attacks the capillary tubes immediately next the alveoli, and is a catarrhal instead of a croupous inflammation. The so-called lobular pneumonia is nothing more than catarrhal pneumonia, the changes in the lobules being secondary to the catarrhal process in the ultimate bronchi. Lobar pneumonia is a fibrinous or croupous pneumonia occupying and confined to a lobe. Pneumonia is also known in common language as "lung-fever," "winter-fever," etc.

Causes.—There is a growing belief that pneumonia is a constitutional or an infectious disease, like typhoid or relapsing fever. It differs from other inflammations in that it is self-limited, and terminates by crisis. It is a very common disease ; it occurs in all degrees of latitude, under every variety of climate, and at all ages. It is common in infants at the breast, but declines somewhat after the second year until after the second dentition, and is frequently encountered and is very fatal in the old. The male sex is most frequently attacked, because men are more exposed than women to those external conditions which tend to produce it. In-door life, a vitiated atmosphere, excesses, especially alcoholic, and bad hygienic influences of every kind which induce debility, favor attacks of pneumonia. Certain seasons appear to invite the disease—those parts of the year characterized by humidity, high winds, and low temperature. In the British Islands winter is the season of greatest prevalence ; on the Continent, spring ; in this country, winter and spring, the former especially—hence the name winter-fever. Occasionally, pneumonia occurs in so many persons in a particular district that it may seem to be epidemic, but there are atmospheric influences at work to produce the disease, especially excessive moisture conjoined with low temperature. It is a common belief that pneumonia is caused by exposure to cold, especially to draughts when the body is warm and perspiring. That catarrhal pneumonia is induced in that way no one will dispute, but it is more than doubtful that croupous pneumonia is thus caused, unless there exist a predisposition to it, either of a vulnerable constitution or an inherited tendency to pulmonary disease. A phthisical tendency, the author believes, is the chief factor, or that peculiar-

ity in the structure of the pulmonary tissue associated with consumption. There are other diathetic states concerned in the production of pneumonia—as gout, rheumatism, diabetes, the eruptive fevers, especially chronic alcoholism. Probably the real etiological factor is a *microbe*. Klebs* first made the attempt to define the pneumonococcus, but the *micrococcus* pneumococcus of Friedländer has been more generally accepted as the true infective organism of croupous pneumonia, and this is active under the atmospherical conditions above mentioned, but the *dipplococcus* of Fränkel is held by some to be, also, an efficient agent in setting up of the pneumonic process.

Pathological Anatomy.—The state of the affected lung in pneumonia is usually divided into three stages, following the original description of Laennec, based on the naked-eye appearances: engorgement; red hepatization; gray hepatization. The better arrangement, based on the description of Jaccoud,† but modified, is as follows: *The stage of hyperæmia*, or engorgement; the *stage of exudation* (red hepatization); the *stage of resolution* (degeneration and extrusion of the exudation); the *stage of purulent transformation* (gray hepatization). In the stage of hyperæmia or engorgement, as now described, there are two distinct and separate acts—the increased blood-supply and the pouring out of an exudation. The lung has a reddish-brown appearance, is heavier, floats in water, but sinks lower than the normal lung-tissue, crepitates but little when pressed, and it is no longer elastic, but when an impression is made by the fingers it is retained. On section it presents a pretty uniform brownish-red tint, and it exudes a quantity of blood. On microscopic examination the blood-vessels are found to be distended with blood, and the capillary network surrounding the alveoli is so much enlarged that the alveoli are encroached on by it.‡ The adjacent portions of the bronchioles are similarly engorged, the mucous membrane dark reddish from fullness of the vessels. This hyperæmia marks the first stage in the inflammatory process. The next step consists in the *pouring out and coagulation of an exudation*. There is exuded into the alveoli an albuminous or fibrinous fluid of great viscidity, and with it leucocytes which have wandered from the vessels, and red blood-globules present by diapedesis, and blood by the rupture of distended capillaries. This viscid albuminous fluid is poured out also into the bronchioles and bronchi of the inflamed section, and with it leucocytes and some red corpuscles. When the surfaces approximate, this adhesive fluid holds them tightly together until the incoming air separates them. In the capillaries of the inflamed area the blood-current is finally stopped, and the corpuscles are then seen to be closely packed together and flattened at the points of contact. The albuminous or fluid exudation remains fluid

* "Archiv für experiment. Pathologie u. Pharmacol.," vol. iv, p. 1,1875.
† "Traité de Pathologie Interne," vol. ii, p. 45. ‡ Rindfleisch, *op. cit.*

for a short time, and then solidifies or coagulates, beginning in the alveoli and extending through the bronchioles outwardly. In coagulating it incloses the white and red corpuscles, and fills out the alveolus or bronchiole, probably expanding somewhat in the act of coagulation. When this process is completed, the inflamed part is solid, entirely without air, and falls immediately to the bottom when placed in a vessel of water; it is also friable, is easily broken up between the fingers, and on section with the knife divides cleanly with well-defined margins. The cut surface presents a reddish color, and is granulated; this granular appearance being due to the little masses of coagulated exudation filling the cavity of the alveoli. These little masses may with some care be lifted out of the mold in which they are formed and held on the point of a pin. The tissue of the inflamed part, in respect to color, density, and granular appearance, so strongly resembles the cut surface of a section of the liver as to be called by Laennec *red hepatization.*

There are two directions which the inflammatory process may now assume: toward resolution, or return to the normal state; toward purulent transformation. As the first is the more usual, we describe first the *process of resolution.* The albuminous material which had solidified undergoes liquefaction, and the pressure is thus removed from the surrounding vessels. The watery parts of the exudation diffuse into the vessels, and the solids, together with the cellular elements, undergo a fatty metamorphosis, and are transformed into an emulsioned mixture without any of the viscidity of the original exudation, and capable either of absorption or of extrusion, much of it, doubtless, being expectorated. As the exudation liquefies, air again enters the alveoli, diffusion of oxygen into and of carbonic acid out of the blood is resumed, and the current of the circulation is fully reëstablished. The effusion into the connective tissue between the alveoli and bronchioles is finally taken up, and the normal color and density are restored to the inflamed part, but its elasticity continues impaired for a long time.

When the *purulent transformation* takes place, a change is wrought in the density, color, and constitution of the inflamed area. It has been much discussed whether the epithelium of the alveoli undergoes any change, and contributes, by multiplication of its cells, to the exudation in croupous pneumonia, and whether any of the pus-corpuscles which become so abundant during the stage of gray hepatization or purulent transformation originate by proliferation of the epithelial cells. The former is denied by most authorities; the latter is highly probable; but the pus-cells are derived chiefly from the wandering white cells by multiplication and division. With the formation of pus-cells a process of fatty degeneration takes place in the albuminous exudation, but the rapid and exuberant formation of pus-cells is the principal event, the tissue being changed in color from the reddish-brown appearance of the red hepatization to the yellowish or grayish-