The area of cardiac dullness is increased when the effusion is sufficient in amount. The enlargement of the area of relative dullness is more important in a diagnostic point of view, because there may be no change in the absolute dullness, even when there is considerable effusion. The diminished sonority is first perceived at the sternal end of the third and fourth ribs—at the base of the heart. The dull

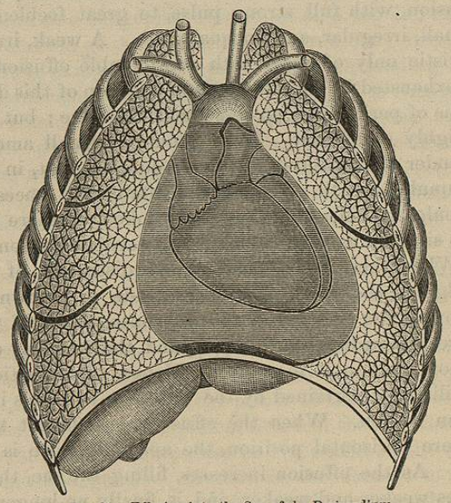

Fig. 21.—Effusion into the Sac of the Pericardium.

space has a triangular form, with its apex uppermost and base downward—the right line of the triangle extending from the apex at the second rib and sternum, along the right border of the sternum, and even beyond, to the right sixth and seventh ribs and sternum; the base-line of the triangle passing through the seventh intercostal to the axillary border, and there intersecting the left line. When the effusion is extreme, the epigastrium is pushed outward by the descent of the diaphragm and the left lobe of the liver. The size of the triangular space is enlarged by sitting up and by bending forward. When the apex-beat can still be felt, and the area of dullness extends beyond it, this fact indicates that the sac of the pericardium is greatly distended, and consequently forced beyond the apex, and is therefore an important sign of effusion. A change in the position of the dullness may be slightly effected by changing the decubitus of the patient, the fluid obeying the laws of gravity. The pressure of the lung in the neighborhood of the pericardium is a necessary result of the accumulation of fluid; but this condensation is distinguished from effusion

by the vocal fremitus, which is weakened or absent in the latter, but increased or normal in the former. In estimating the results of percussion, two sources of error may interfere: the dullness may be more extensive than the amount of the effusion warrants; it may be less. The first is due to adhesions which have the effect to retract the lung from the pericardium, and to push the heart forward, thus enlarging unduly the area of absolute dullness; in the other, the lung is attached anteriorly, and the heart lies deeply, and is still further depressed by the weight of the effusion. The pericardial *friction murmur* is the most significant of the physical signs of pericarditis, and is produced by the rubbing together of the two surfaces roughened by exudations, or by one roughened surface. This *bruit* makes the impression on the ear of scraping, grating, creaking, churning, and various modifications of these noises. They are, ordinarily, resolvable into three: the creaking of new leather, grating, or scraping. The sound may be partial or general; it corresponds to the seat of the exudation, and is not confined to the situation of the orifices of the heart, but is heard with the maximum intensity at the third intercostal space on both sides of the sternum. The area over which it is audible depends on the extent of the exudation. The *bruit* accompanies the heart-sounds, but is not confined to them, and extends into the interval, and may indeed occupy the whole revolution of the cardiac movement. Hence the term "*bruit de galop.*" Usually or frequently, the *bruit* is presystolic, systolic, and diastolic—the presystolic corresponding to the auricular systole, and the others to the systole and diastole of the ventricles. When there is no effusion (dry pericarditis), there will be usually no rational symptoms of the malady—nothing but fever, and the physical signs of pericardial inflammation.

The friction murmur, as well as the friction fremitus, occur early, and are recognized, if at all, within the first two days, and they persist for several days or weeks, according to the progress and amount of the effusion. They may decline in two or three days and disappear, as the effusion fills the sac and separates the two surfaces, so that friction is no longer possible. If the effusion is absorbed, then the *bruit* will become audible again. When the silence of the *bruit* is due to adhesions, there will be no return of it when it ceases. With the increase of the effusion the heart-sounds become weaker, and finally are no longer heard in some cases; but usually they continue to be audible, although very feebly. The character of the pulse, during pericarditis, has no special quality; it may be but slightly elevated above the normal; it may be very much accelerated; its rhythm may be much altered. At the onset of the inflammation, the pulse may be strong, the tension high; but this is not maintained, the pulse becoming weak, and the arterial tension low from depression of the vital powers and the occurrence of myocarditis. A large effusion exerts a

mechanical pressure upon the great vessels within the pericardial sac —the aorta and pulmonary artery—and interferes with their proper filling. Also, as the veins can not empty their blood into the auricles fully, they are kept over-distended, and an abnormal fullness of the venous system in general is the result. Stasis of the venous system causes passive congestion of the lungs, bronchial catarrh, difficult breathing, cyanosis, and œdema. The venous congestion occurs in the brain, and is manifested objectively by headache, vertigo, epistaxis, etc.; in the liver, causing enlargement of the organ and hyperæmia of the portal system; and in the kidneys, inducing albuminuria. Irritation of the phrenic excites a most distressing hiccough. Difficulty of breathing, cyanosis, feebleness of the heart's action, are also produced by myocarditis, which is really an acute fatty degeneration. The heart's movements are not only feeble, but scarcely distinguishable; the pulse irregular, intermittent, feeble; the sounds of the heart are hardly recognizable, and the first sound is often absent; the temperature falls, the legs become œdematous, and death soon closes the scene. When severe dyspnœa and cyanosis come on in the course of pericarditis, they are more frequently due to the damage done to the heart's muscle than to the mechanical effects of the effusion. Again, the same symptoms, in a less extreme degree, however, may be due to nervous disturbance—to irritation of the pneumogastric and phrenic. Dysphagia may be caused by pressure of the effusion on the œsophagus, and aphonia by pressure on the recurrent laryngeal nerve.

Course, Duration, and Termination.—The course of pericarditis is not always upon a uniform plan, and there are peculiarities due to the causes and complications. Those cases arising in the course of puerperal septicæmia, scorbutus, or pyæmia, are shorter in duration, and greatly more fatal than those which are due to the rheumatic diathesis. The duration is influenced by many circumstances. In simple, uncomplicated cases, terminating in health, the effusion may be absorbed and recovery take place in from ten days to two weeks. When a case tends to recover, the severe symptoms subside, the fever and the difficulty of breathing cease, the appetite returns, and convalescence is established. When there is much effusion, and yet the tendency is toward health, the area of dullness lessens, the apical impulse returns, the friction murmur and fremitus reappear for a short period, the normal sounds are heard again, and, with these evidences of improvement afforded by the physical signs, are also the rational symptoms of cessation of dyspnœa, of fever, and return of appetite. In other cases the improvement is partial; the rational and physical signs of pericarditis persist, and the subsequent history is that of chronic cardiac troubles. In other cases a fatal termination takes place early —in the scorbutic form with hæmorrhage in a few hours after the well-defined symptoms come on; in cases with large effusion, dyspnœa,

delirium, etc., death will occur in a week or ten days; in cases with myocarditis and syncopal attacks, according to the age and other circumstances, a fatal termination may occur within the first two weeks. According to Thompson, the average duration of rheumatic pericarditis in St. George's Hospital is fifteen days.

Prognosis.—Simple cases of pericarditis, and rheumatic pericarditis, are not often fatal, and a favorable prognosis may be expressed in a very large proportion. As an intercurrent disease, coming on in the course of certain grave maladies, it is is extremely fatal. Among these may be mentioned scorbutus, pyæmia, puerperal diseases, Bright's disease, some of the eruptive fevers, pneumonia, etc.

Diagnosis.—The differentiation of pericarditis from endocarditis, hydropericardium, and left pleurisy, presents some points of difficulty. The separation of the endo- and exo-cardial murmurs is often an affair of extreme nicety. Dropsy of the pericardium is to be distinguished from the inflammatory affection by the absence of fever, local pain, and friction murmur. The character of the fluid in any case is to be determined only by the concomitant circumstances. If the patient is scorbutic, it is probably hæmorrhagic; if a subject of chronic alcoholismus, it may be hæmorrhagic; if the accompanying malady is pyæmia, or a septicæmic process, it is probably purulent; if rheumatism, it is sero-fibrinous; if albuminuria, serous. The differentiation of exo- from endo-cardial murmurs is based on the character, quality, seat, and persistence of the sounds. The friction murmur is a sound of rasping, of crackling; the endocardial murmur is softer, smoother. The friction murmur may be local or general, and has no constant relation to the orifices of the heart; the endocardial murmur is heard with maximum intensity within certain valve areas. The friction murmur is not regularly isochronous with the valve-sounds, or with the cardiac rhythm; the endocardial murmurs are usually systolic or disastolic, or coincide with the rhythmic movements of the heart. The friction murmur continues where it began; the endocardial murmurs are propagated in the direction of the blood-current—basal or apical. The friction murmur varies from one hour to another in intensity and extent; the endocardial murmurs remain constant. The friction murmur increases with pressure of the stethoscope on the chest-wall; the endocardial murmurs are not affected by pressure. The friction murmur increases in loudness with the upright position and bending forward; the endocardial murmurs are most distinct in the recumbent posture. The friction murmur disappears when the effusion reaches a certain amount, and reappears for a short time when absorption has taken place; the endocardial murmurs are permanent. The friction-sound of pleuritis is synchronous with the respiration; the pericardial is synchronous with the cardiac movements, or nearly so; suspension of respiration arrests the former, but does not affect the latter. When

that portion of the pleura in contact with the pericardium is the seat of inflammation, a friction murmur, synchronous with the cardiac movements; in that case the distinction is impossible. In pleuritic effusion, as a rule, the dullness changes with the position of the patient, and in the upright position is over the inferior part of the thorax. In pleuritis with effusion, all voice and breath sounds disappear; in pericarditis, they are unaffected, except in so far as the lung is displaced by the enlarging pericardium. In hypertrophy of the heart, the action is heaving, and the apical impulse is strong; in pericarditis, with or without effusion, the impulse becomes weaker, and, as the effusion increases, the apical impulse will cease, or at least greatly diminish in force. In hypertrophy the absolute, in effusion the relative, dullness is increased; and, as has been pointed out, dullness exists beyond the apex of the heart when the effusion is large.

Treatment.—If the initial symptoms are recognized, a full dose of quinine sulphate (ʒj) should be administered with a half grain of morphine, and the cinchonism should be maintained, by repeated smaller doses, for twenty-four hours or longer. When the evidence of effusion exists, there is no longer any indication for the use of quinine, since the inflammatory process has passed beyond control. The next object of treatment, and that which usually engages our attention at once, is the management of the exudation. There can be no question, at present, respecting the influence of ammonia salts in lessening the coagulability of the fibrinogenous substance. The carbonate should be given in solution of the acetate—five grains every two hours—when the exudation is forming, and to procure its disintegration and absorption, thus preventing adhesions. If the state of the patient will warrant, pilocarpine can be given with much advantage in most of the cases. Untoward results need not be feared if the proper dose of the remedy is not exceeded, and the mode of administration may be stomachal or hypodermatical—for there is little difference in the amount of the action in either way. As it exerts remarkable power in the removal of effusions and exudations, every case, resisting the more simple expedients should have the advantage gained by the use of pilocarpine. If the dose does not exceed $\frac{1}{5}$ grain of the hydrochlorate—and in susceptible subjects one-half ($\frac{1}{10}$) of this amount may suffice—no dangerous symptoms can arise from it in the case of adults. As, however, the production of physiological effects is essential to therapeutical action, the dose insufficient to cause symptoms can not do good.

When the initial symptoms make their appearance, if the patient is robust, six to ten leeches should be applied to the epigastric region; they should be allowed to fill and fall off, but the bleeding should not be encouraged. Dry cups may be applied to the same point, if the condition be that of debility. With or without previous abstraction

of blood, if the patient is not depressed and the action of the heart feeble, an ice-bag should be applied to the præcordia during the initial period, but this expedient ceases to be useful when there is much exudation, and may be very injurious if the heart is weakened by myocarditis. When the time comes for the removal of ice, good results may be expected from the application of flying-blisters. As a condition of quietude of the diseased organ is a measure of the highest utility, remedies which slow the heart are necessary. Aconite-root tincture and veratrum-viride tincture may be given to quiet the heart before considerable damage has been done. When, however, the heart begins to flag, remedies of a depressing kind are not suitable, and then digitalis becomes extremely serviceable, not only to lessen the work of the heart, but to promote absorption. The infusion is the best form, and it should be given in a tablespoonful-dose every four hours. The absorption of a pericardial effusion may be hastened by the use of pilocarpine—so administered as to act freely on the skin. But pilocarpine is too depressing a remedy when the action of the heart is feeble and the pulse is small and irregular. Stimulant doses of quinine and alcoholic stimulants are very important when the powers are failing and syncopal attacks are occurring. Mechanical means are proper when the effusion into the pericardial sac is great and does not yield to the remedies proposed. Paracentesis of the pericardium has now been performed many times with success, so that it can no longer be regarded as a doubtful experiment. The hypodermic syringe may be used to ascertain the character of the effusion. The needle, as in the operation for capillary puncture, is inserted close to the border of the sternum, in the fifth intercostal space. The operation of paracentesis is required when the effusion is great, or when it is purulent. If the effusion returns repeatedly, it is safe practice to inject the tincture of iodine (ʒ ij—℥ iv) to prevent the reaccumulation. If the contents of the sac are purulent, the iodine should be used more freely (ʒ ij of the tincture, ℥ ss potassium iodide, and ℥ iv water). To avoid wounding the heart, the patient should be recumbent when the puncture is made. The disadvantages of the operation are, that it is rarely curative; that it has caused a pneumo-pericardium; that the fluid is quickly replaced, because of the lessened extravascular pressure; that hæmorrhages take place by rupture of the thin-walled vessels of the neo-membrane. Better results are claimed from the operation of paracentesis when a part of the fluid is drawn at a time, rather than all at once.

ADHESIONS OF THE PERICARDIUM.

Adhesions of the two pericardial surfaces are results of pericarditis. They occur in a variety of forms: as narrow bands, as membraniform partitions, dividing the cavity into several smaller cavi-

ties, and sometimes these secondary sacs contain exudation, in the form of a caseous mass, or dark-brown deposits, a product of altered blood. The adhesion may be total, so that after some years no line of union can be made out between the two surfaces. The mass of exudation uniting the surfaces may be converted into an apparently bony case enveloping the heart by calcareous deposition. Bands of adhesion may exist externally to the pericardium, and unite this membrane to the neighboring pulmonary pleura, to the pleura costalis in front, to the mediastinum, etc. As has been pointed out in the preceding chapter, an inflammation of the pericardium leads to acute myocarditis—an acute fatty degeneration of the muscular tissue. Hypertrophy and dilatation are among the results of adhesions. Opinions are divided as to the precise part played by the adhesions, but there can be no doubt that atrophy with hyperplasia of the connective tissue are results of the myocarditis, which, in turn, induces dilatation of the cavities. When the cavity of the pericardium is obliterated, and adhesions have been contracted to neighboring parts also, the heart works to great disadvantage; but the most serious result is the interference with the nutrition of the organ. On the other hand, there may be entire adhesion of the two pericardial surfaces, and the heart be not at all incommoded.

Symptoms.—The disturbances produced by adhesions are manifested in rational and physical signs. The propelling power of the heart being diminished, stasis takes place in the right cavities, in the lungs, and venous system generally. There are therefore constantly present bronchial catarrh; difficulty of breathing; swollen liver and spleen; gastro-intestinal catarrh; urine scanty, high-colored, and albuminous; veins full, face cyanosed; general dropsy. The apical impulse is either wanting entirely, or is a mere tremor; the pulse is rather quick, but low in tension, and the volume varies in different beats. These rational symptoms are chiefly indicative of the degeneration and atrophy which have occurred in the heart-muscle. Other symptoms are caused by adhesions. One of the most important physical signs of pericardial adhesions is a depression with the systole of the heart at the place of the apex-beat. Instead of an elevation of the intercostal space when the apex of the heart is tilted against it at the time of the systole, there occurs a *depression, or drawing in of the chest-wall.* There may also be, at the left of the sternum, several small depressions or "pittings" in the intercostal spaces. These depressions are frequently due to pericardial adhesions of the two surfaces, and to the parietal pleura; but they may occur independently of this, as has been demonstrated by Friedreich, produced by causes which obstruct the downward movement of the heart toward the left, and the tilting of the apex upward, the lungs at the same time not coming forward sufficiently. A diastolic elevation of the chest-wall is the compensatory sign of the preceding elevation. When the force producing the other

ceases to act, there is a rebound of the chest-wall, which, if not visible to the eye, may be felt on palpation. These two signs are highly significant, but their absence does not negative the existence of pericardial adhesions. It has already been stated that the area of absolute dullness is increased in those cases of adhesions which fix the heart against the chest-wall, and do not permit the organ to fall back, while at the same time the lung is prevented coming forward. If the heart is so fixed in position by adhesions, and is at the same time hypertrophied, and if the pericardium be adherent to the chest-wall, and to the spine behind, there must, of necessity, be produced the *systolic depression.* When the *diastolic rebound* ("diastolic concussion") occurs, a synchronous or diastolic collapse takes place in the cervical veins. Much distended during the systole, they suddenly subside and even disappear during the diastolic rebound, for during this act the chest is expanded and the blood is drawn into the cavity. The importance of pericardial adhesions depends much less on the adhesions than on the changes in the heart-muscle. Adhesion bands connecting the two surfaces may exist without injurious effects. When hypertrophy takes place compensation ensues, and the heart is equal to its duties for many years. On the other hand, when the heart-muscle undergoes atrophic degeneration, its propelling power is insufficient, venous stasis and dropsy follow, and then a fatal termination is near. The *treatment* in these cases must be directed to the nutrition of the heart-muscle. Rest must be enjoined; the appetite and digestion must be improved by bitters, mineral acids, and the ferruginous tonics. The heart must be toned up by digitalis and iron, and by the judicious administration of quinia and morphia—the latter in minute quantity ($\frac{1}{16}$ of a grain). The author has seen the greatest advantage from the use of sulphate of iron (gr. j), sulphate of quinia (gr. ij), sulphate of morphia (gr. $\frac{1}{20}$), and digitalis (gr. j) in pill-form, three times a day.

HYDROPERICARDIUM—DROPSY OF THE PERICARDIUM.

Pathogeny.—By *hydropericardium* is meant an accumulation of water in the sac of the pericardium without the occurrence of inflammation. After death, especially from chronic wasting diseases, there will be often found in the sac an ounce or two of fluid, poured out at the time of the death-agony and immediately after. In dropsy, properly speaking, the quantity of fluid may reach to one or two pints. It is a clear, yellowish, or straw-colored serum, usually, but it may present a somewhat turbid appearance from the presence of cast-off epithelium, or a bloody appearance derived from hæmatin. This fluid has the composition of the blood-serum, and its alkaline reaction, but does not contain the same relative proportions of its constituents. The albumen is less than in the blood-serum, and also some of the salts; but it

contains the fibrinogenous substance which sometimes coagulates when exposed to air. Urea is found in this fluid in renal diseases, and it is stained with bile-pigment in cases of jaundice. The fluid, if large in amount, dilates the sac, and its walls become thinned by the pressure, and often present a sodden appearance when there has been a protracted contact of the fluid with the endothelium. The subserous fat is absorbed by the pressure, and the areolar tissue is infiltrated with fluid.

The causes of hydropericardium are twofold: mechanical and dyscrasic. Diseases or neoplasms,* that interfere with the return of blood through the veins, as tumors, obstructive pulmonary disease, emphysema, and dyscrasia, such as Bright's disease, cancer, and tuberculosis, are the principal etiological factors.

Symptoms.—A small quantity of fluid will not produce sufficient disturbance to cause recognizable symptoms; a large effusion will be recognized by the rational and physical signs, such as were described under pericarditis, with effusion. There is, of course, no friction murmur. The apical impulse becomes more and more feeble as the effusion increases, and it ultimately ceases to be felt. The heart-sounds grow more and more feeble, and may disappear entirely. The area of relative dullness greatly increases and extends finally beyond the region of apex-beat, and has the characteristic triangular form of dullness from effusion. The diagnosis of hydropericardium, from the effusion of pericarditis, rests entirely on the history—the latter being due to inflammation, the former not. The prognosis of this malady is serious, not wholly because of the fluid, but on account of the conditions associated with it. The treatment is directed to the removal of the fluid, and consists in the use of eliminants and mechanical means; purgatives, diaphoretics, and diuretics are employed to procure absorption. Saline purgatives, compound jalap powder, elaterium, are given to diminish blood-pressure and the quantity of fluid; squill, digitalis, and cream-of-tartar, to excite diuresis; warm baths and pilocarpine to stimulate the skin. These means may be entirely successful in some few cases in Bright's disease, for example, but will have but little effect in cases of emphysema, tuberculosis of the lungs, and when the effusion is due to the pressure of a tumor. Aspiration is proper when life is threatened by the extent of the effusion, but there is danger of exciting pericarditis and of the admission of air.†

HYDROPNEUMOPERICARDIUM.—This form of disease differs from the preceding in that air or gas, as well as fluid, is present in the cavity. The fluid, when gas is also present, is composed of some decomposing exudation, of pus, or of blood. The first named is derived

* "Transactions of the Pathological Society of London," vol. xxii, p. 123.

† Roberts, "Paracentesis of the Pericardium." Philadelphia, 1880. An excellent work.

from pericarditis, the result of traumatism, or excited by an ulceration penetrating the cavity from the neighboring parts. The symptoms are physical. The space of absolute dullness is occupied by a tympanitic sound, except at the base, where it is dull from the presence of fluid. Change of the patient's posture alters the position of the dullness. The heart-sounds and the apical impulse are sometimes feeble and may not be perceptible, but are usually loud, splashing, and prominent. A peculiar, clanging, metallic character is imparted to the heart-sounds. The friction murmur has a rough, rasping, metallic resonance. Very remarkable sounds are produced by the churning of the liquid and air together by the heart-movements, and are designated "the water-wheel sounds." The functional disturbances produced by hydropneumopericardium are those of pericarditis, and need not therefore be recapitulated. The prognosis is grave; yet, of fourteen cases collected by Friedreich, only ten proved fatal. It has usually been regarded as more fatal than these figures indicate. It is probable that some of them were examples of the admission of air merely, and were not produced by the gas of decomposition. The treatment is that of pericarditis. The presence of decomposing materials, or such an excess of gas or fluid as to exercise dangerous compression, justifies the employment of the aspirator, and washing out the sac with an iodine solution.

INFLAMMATION OF THE MUSCULAR TISSUE OF THE HEART —MYOCARDITIS.

Definition.—The cardiac muscle is subject to attacks of inflammation, as muscular tissue in other situations. The term *myocarditis* includes several morbid conditions of an analogous kind, but different in seat and also in progress.

Causes.—The male sex is more liable than the female. The acute form is more common before than after thirty years of age. Myocarditis may occur during intra-uterine life, and then preferably on the right side, setting up important changes. It is supposed that chilling the body, suddenly, when in a warm and perspiring state, will cause this disease; again, violent muscular exertion is said to have excited inflammation; but these are very doubtful causes. In fact, nothing is definitely known of the influences setting up such a morbid process in the heart-muscle. As regards the secondary diseases, our information is more definite. It has already been pointed out that myocarditis is a result of pericarditis, the inflammation extending by contiguity of tissue. It results from valvular lesions also, and may be secondary to the acute infectious diseases—as typhoid, pyæmia, scarlet fever, etc. Inflammation and abscess may be the result of embolic obstruction of the coronary artery.

Pathological Anatomy.—The muscular tissue itself, or its inter-