seat of an active hyperæmia, which is designated *collateral hyperæmia.* One result of the increased pressure in this hyperæmic area is the rupture of small or large vessels and extravasation of blood. If the vessel obstructed is small and not a terminal artery, the anastomoses may be sufficient to supply the anæmic district. If, however, the compensatory circulation is insufficient or absent, the ischæmic part dies—undergoes *necrobiosis*, *gangrene*, or *necrosis.* The consequences following arrest of the circulation by an embolus depend largely on the position, still more on the size, of the obstructed vessel. Dry gangrene is produced by embolic blocking of a vessel of an extremity. In internal organs, especially the brain, centers of softening and fatty transformation of the tissue elements, and hæmorrhagic extravasations in the area of collateral hyperæmia, are results of embolism. Besides the hæmorrhagic extravasations, infarctions occur in the parenchyma of those organs supplied with Cohnheim's terminal arteries.*

Symptoms.—The position of a thrombus or an embolus exercises a most important influence on the symptoms caused by them. When a thrombus occupies a vein of an extremity, œdema of all the parts below is a result, and, if the obstructed vein is adjacent to important nerves, excessive pain, or troubles of motility, will also be present by reason of the pressure of the distended vessel. Gangrene is not a result, since the nutrition of the parts is accomplished, although feebly and imperfectly, but moist gangrene may be produced if other injuries are superadded—as erysipelas, traumatism, compression, etc. A cure in such a case is in part effected by the collateral circulation, but in a truer sense by the canalization of the thrombus. Notwithstanding the similarity in the symptoms, caused by thrombosis and embolism respectively, there is a great difference in the time at which the phenomena manifest themselves: the symptoms of autochthonous thrombosis come on gradually; of embolism suddenly, with shock (Wagner). Two classes of symptoms arise—affections of nutrition, from the simplest disorder up to gangrene, and functional disturbances, proper to the organ affected. These symptoms are not ascertained with the same facility in all situations. In the extremities, every step in the local process is easily followed and interpreted, but in internal embolisms only those symptoms due to perversion or suspension of function are recognizable. Embolic obstruction of a member is announced by a sudden and often intense pain and a chill, with numbness, loss or diminution of tactile sense, coldness, pallor of the skin, and a feeling of deadness and weight, and paralysis of the muscles; the pulsations wanting below, while above the obstruction they are full and strong. If embolic blocking of a vein in the brain, there occur defects of speech, hemiplegia, etc.; if of a pulmonary artery, sudden difficulty of breathing and sense of oppression, with, it may be, intense oppression and anxiety and death. Sudden attacks of amaurosis in puerperal fever, acute rheumatism, and pyæmia, are usually due to embolism of the central artery of the retina. Those organs not well supplied with nerves, as the liver, kidneys, and mucous membranes, do not offer distinct reactions on embolic blocking of their vessels, and hence the symptoms are obscure.* If the immediate danger of an embolic obstruction is past, even if the symptoms are very formidable, provided terminal arteries are not obstructed, they may disappear in some hours or days by establishing a collateral circulation.

* Wagner, *op. cit.* "Untersuchungen über die embolischen Processe," von Dr. Julius Cohnheim, Hirschwald, pp. 112. Berlin, 1872.

Treatment.—As all the symptoms are due to the obstruction of vessels by a blood-clot, the point in the treatment of special importance is to effect a solution of this obstructing material. Theoretically, ammonia possesses a solvent power, and in its use the author has had most striking results in the case of thromboses and embolisms of the brain. To accomplish the purpose in view, ten grains of the carbonate of ammonia may be administered in a tablespoonful of solution of the acetate, three or four times each day. As, however, the action must be slow, the point of contact being small, the remedy must be very persistently employed. The iodide of ammonium may be administered in a solution with the carbonate also, and usually with good results. Other alkalies possess the same power, but to a less extent. The most generally useful is the phosphate of soda, in drachm-doses, three times a day, used for many weeks. As, however, prompt and speedy action is needed to avoid the serious structural alterations which occur so quickly, the ammonia preparations are preferable to any other having the same effects.

DISEASES OF THE HEART.

TOPOGRAPHY OF THE CARDIAC REGION.—METHODS OF PHYSICAL DIAGNOSIS.

The heart lies somewhat obliquely in the chest, behind the sternum, and extends downward to the left, so that the apex-beat is below and a little internal to the left nipple, and between the fifth and sixth ribs. The position of the heart varies somewhat with the position of the subject: it gravitates downward and forward when the posture is erect; backward and upward when the posture is recum-

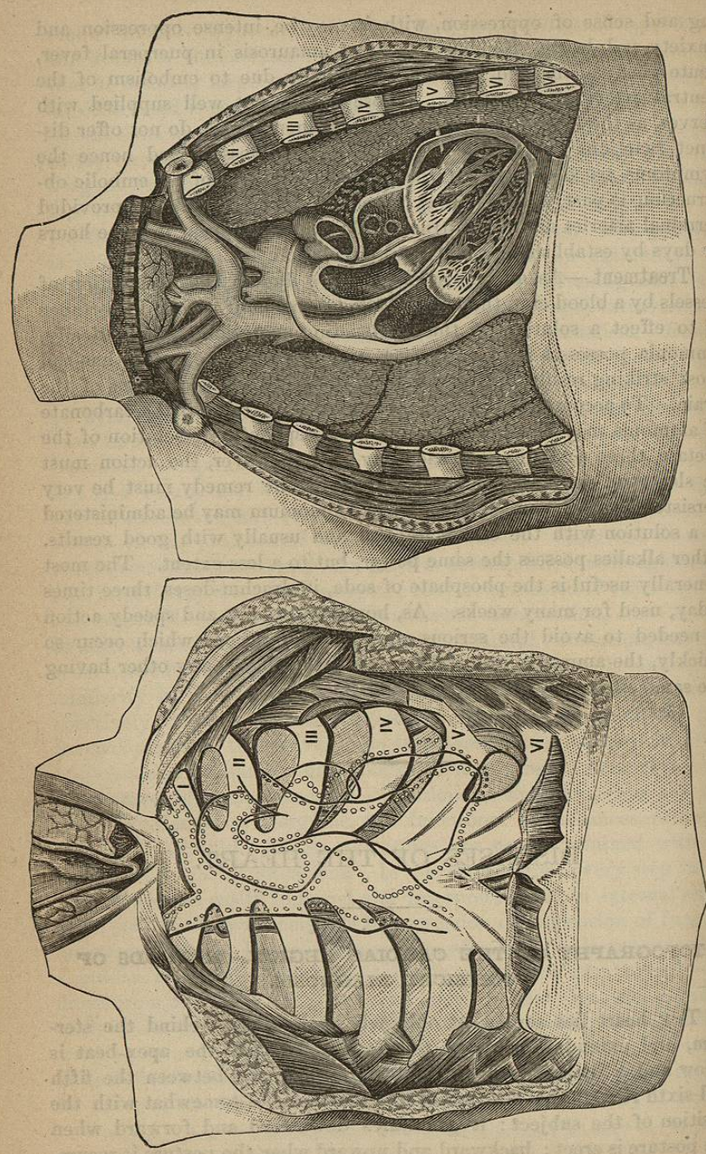

Fig. 19.—The Relation of the Valves and Orifices of the Heart to the Ribs, Sternum, and External Surface of the Thorax. Modified from Rüdinger.

bent. In general terms, the base of the heart is opposite the upper border of the cartilage of the third rib ; the most inferior part of the right auricle and the apex rests on a line parallel with the upper border of the sixth rib,* and in contact with the upper surface of the diaphragm (Figs. 19, 20).

It is important to note the position of the valves with reference to exterior points of the thorax.

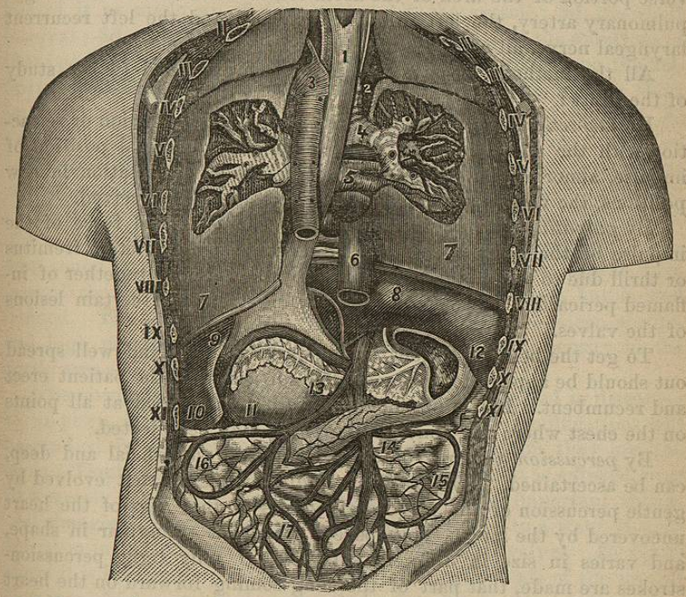

Fig. 20.—Posterior View.—1, Œsophagus; 3, Aorta; 4, Right Primary Bronchus; 5, Right Pulmonary Artery; 6, Ascending Vena Cava; 7, Lungs. After Rüdinger.

The aortic orifice lies under the junction of the cartilage of the third left rib with the sternum at its lower margin. The orifice of the pulmonary artery is at the left edge of the sternum, opposite the second intercostal space. The left auriculo-ventricular (mitral) orifice is somewhat less than an inch from the left margin of the sternum, and is beneath the third costal cartilage. The right auriculo-ventricular (tricuspid) orifice is under the center of the sternum, opposite the upper border of the fourth intercostal space. It follows that, if the stethoscope be placed on the junction of the third left costal cartilage with the sternum, its circumference will bisect each of the cardiac valves.

* Henle's "Handbuch," Dritter Band, "Gefässlehre," p. 38 ; also Rüdinger, *op. cit.*

The ascending portion of the arch of the aorta extends about one fourth of an inch beyond the right margin of the sternum, and the vena cava lies half an inch to the right of the aorta, both vessels being behind the right second costal cartilage and the first and second intercostal spaces. The trunk of the pulmonary artery is beneath the sternum, to the left of the middle line, and extending somewhat more than half an inch beyond the left border of the sternum. The transverse portion of the arch of the aorta comes into relation to the right pulmonary artery, the left primary bronchus, and the left recurrent laryngeal nerve, all of which pass under it.

All the methods of physical diagnosis are applied to the study of the heart:

By *inspection*, the position of the apex-beat, protrusion or retraction with the apical impulse, area of impulse, abnormal position of impulse, abnormal pulsations in proper position, or pulsation in new positions, the venous pulse in the neck, etc., may be perceived.

By *palpation* can be ascertained the character and force of the impulse of the heart, the rhythm of its movements, friction fremitus or thrill due to the vibration imparted by the rubbing together of inflamed pericardial surfaces, and the purring tremor of certain lesions of the valves.

To get the best indications from palpation, the hand well spread out should be applied to the precordial surface, with the patient erect and recumbent. Palpation should, in turn, be practiced at all points on the chest where any abnormal pulsation may be detected.

By *percussion*, the area of cardiac dullness, superficial and deep, can be ascertained. By *superficial dullness* is meant that evolved by gentle percussion of the precordial region—of that part of the heart uncovered by the lung. This space is somewhat triangular in shape, and varies in size in different subjects. If only light percussion-strokes are made, that part of the lung coming forward on the heart is thrown into vibration, hence imparting the pulmonary quality to the tone. In determining the presence or absence of effusion into the pericardial sac, or hypertrophy, this superficial dullness has more significance than the deep. When strong percussion is practiced, the body of the heart is reached, the vibrations have a different character, and hence the production of *deep dullness*. The superficial dullness is not only more significant, but is also more easily determined. The condition of the pulmonary parenchyma, of the stomach and colon, and the degree in which the left lobe of the liver extends to the left, modify the deep dullness and increase the difficulty of defining it accurately.

The position, shape, amount, and degree of the deep cardiac dullness must be noted. The relation of these modifying circumstances to morbid states of the heart will be pointed out when the individual maladies are under consideration.

By *auscultation* we obtain the most exact information of the state of the heart. Certain facts regarding the normal condition of the organ must be clearly fixed in the mind in advance of the study of pathological states. That these may be the more readily comprehended and remembered, all statements not immediately utilizable are avoided.

The *cardiac cycle*, or the whole period of the heart's movement, has been variously divided, but it suffices for practical purposes to regard it as made up of four parts: 1st, the systole; 2d, the short interval; 3d, the diastole; 4th, the long interval. If the durations of these periods, respectively, are expressed in tenths, they have the following relations to each other: the systole, $\frac{4}{10}$; the first or short interval, $\frac{1}{10}$; the diastole, $\frac{2}{10}$; and the second or long interval, $\frac{3}{10}$. Certain sounds coincident with the systole and diastole are audible on application of the ear to the chest. These sounds are designated the *first* or *systolic sound*, due to the muscular contraction and the tension of the mitral and tricuspid valves, and the *second* or *diastolic sound*, due to the sudden stretching of the aortic and pulmonary valves. The first sound, audible with the greatest intensity in the mitral area, toward the apex, is rather dull, low pitched, prolonged, but is well defined. The second sound is much shorter in duration, clear, abrupt, and high pitched, and is audible with the maximum intensity in the aortic area—at the base.

The normal sounds of the heart are variously changed in disease—as to pitch, duration, quality, and rhythm. These modifications will be studied in connection with the maladies of which they are the signs. Doubling of the sounds, or *reduplication*, as it is entitled, may be a merely functional condition, or it may signify important structural changes. This phenomenon has been referred to a want of synchronism in the actions of the tricuspid and mitral valves. By Guttmann it is held to be the separation into distinct sounds of the several elements which, combined, make up the first or second sound, respectively—a lack of synchronism in the contraction of the papillary muscles acting on the segments of the mitral or tricuspid valve. This explanation is accepted by many as an adequate explanation of the phenomenon.

INFLAMMATION OF THE PERICARDIUM—PERICARDITIS.

Definition.—The term *pericarditis* means an inflammation of the pericardium. The inflammation may be limited to the parietal or visceral layer, or to a part of either, or it may involve the whole of both surfaces. In the former case, it is *partial* or *circumscribed;* in the latter, *general* or *diffused*. The inflammation may also be either *acute* or *chronic*.

Causes.—Idiopathic or primary pericarditis may arise from traumatism or from cold. In those cases supposed to be produced by changes of temperature there is usually, probably, a diathetic condition—as albuminuria—which escapes notice. Secondary pericarditis is more common, and is due to two causes: to an extension of inflammation from neighboring parts—pneumonia, left pleurisy, pulmonary tuberculosis, caries of the sternum or ribs, aneurism of the aorta, endocarditis, etc.; to the rheumatic dyscrasia. The dependence of pericarditis on rheumatism has been very differently stated by the different authorities. That in about one third of all the cases this complication arises is the opinion of Bamberger, and is doubtless a close approximation to the truth, but Thompson* says sixteen per cent. The severity of the cases, but not the position of the joints affected, has some influence in determining the frequency of the complication. The first attack is more liable to this complication; the second attack stands next. In Thompson's forty-three cases of pericarditis, twenty-five happened during the first attack and thirteen during the second. The author has seen three cases in which the pericarditis preceded the joint affection. Usually this complication arises during the period of greatest severity of the disease—during the second week, the favorite days being the ninth and tenth (Thompson). Pericarditis also occurs during the course of certain eruptive fevers, as scarlatina, variola, in puerperal fever, in albuminuria, scorbutus, etc., but there are no numerical data for an exact statement of the relative frequency. As regards the period of life in which pericarditis happens, there are differences in the two sexes—women being more liable during the period of puberty, thirteen to twenty, and men from twenty to thirty, the average being respectively nineteen and twenty-five (Thompson). Men are somewhat more liable to the disease than women, but the difference is slight.

Pathological Anatomy.—In the first stage of the inflammation there are two pathological conditions present: an alteration of the tissue, the seat of the inflammation; and an effusion into the pericardial sac. The inflamed membrane is marked by an arborescence of minute vessels, or is of a deep-red color, in consequence of the general stasis, and contains here and there spots of extravasation from rupture of over-distended vessels. The membrane becomes dull, cloudy, and at first dry, and also swells from interstitial exudation, and its resistance is diminished by the separation of the connective-tissue elements. The stage of hyperæmia and suspended secretion is of short duration—lasting from a few hours to twenty-four, the shorter rather than the longer period. Rarely a case occurs in which there is no other than the interstitial exudation, no moist exudation on the surface, nor effusion

* "St. George's Hospital Reports," vol. iv, p. 31.

into the cavity. Usually, after a variable period of a few hours, the membrane which was dry becomes coated, especially the visceral layer about the origin of the great vessels, with an exudation of fibrinous substance, having, it may be, a thin, pellicular character, or thicker and more consistent, but soon extending over both surfaces. Sometimes the exudation is reticulated, sometimes it forms conical or filiform projections—pineapple heart, *cor villosum*, *cor tomentosum*, etc. These peculiar appearances are due largely to the movements of the heart and the friction of the exudation on the two surfaces. When the exudation is sero-fibrinous, more or less straw-colored serum, having flocculi of lymph or masses of fibrinous substance floating in it, is contained in the cavity. Instead of being straw-colored the fluid may retain so much of the solid exudation churned up with it as to have a creamy consistence and a yellowish color; or it may have a reddish tint from a slight admixture of blood, or be composed largely of blood (hæmorrhagic pericarditis). The serous fluid may also have a yellowish tint from the presence of leucocytes, or the exudation may have from the beginning a purulent character. The latter is the case in pericarditis occurring during pyæmia, puerperal septicæmia, variola, etc. The hæmorrhagic exudation occurs in chronic alcoholismus and in scorbutus. There are, therefore, sero-fibrinous, hæmorrhagic, sero-purulent, and purulent exudations. A strictly serous exudation is found in general dropsy, in dropsy of the pericardium, etc., but not in true pericarditis.

Effusions may be entirely removed, even those consisting largely of solid exudation. The fibrinous matter breaks up into a granular mass, which gradually becomes fatty; the cells also undergo a fatty metamorphosis; the watery part is quickly taken up and the fatty emulsion undergoes slow absorption. A complete restoration of the parts to the normal may ultimately take place, but this is an exceptional result. It is to be expected only when the exudation is largely serous, or when the fibrinous substance is deposited on a small extent of surface and is thin. Usually the watery part of the exudation is taken up; the migrated white-blood corpuscles in the mass of fibrinous exudation assume a fusiform shape, unite end by end, and form canals or blood-vessels, and thus an exudation becomes organized. The epithelium takes part in these changes, by the proliferation of its cells, and the mass of solid exudation is composed not only of fibrinous substance, but migrated leucocytes, and proliferating epithelium, mixed with a basis substance, composed of germinal matter.* Projecting masses of exudation, uniting from the two sides, form bands, which organize by the formation of vessels, and remain permanently. There may be a thin band or bands connecting the visceral and parietal layers, or larger and broader bands which, uniting, form sub-

* Rindfleisch, *op. cit.*, p. 265.

divisions of the sac, or, the two surfaces may be glued together, entirely obliterating the cavity of the pericardium. The union may be so perfect that the most careful dissection can not separate them. Calcareous deposits may subsequently form in the exudation, or the whole of it may finally become so completely calcified, by the deposit of lime salts, that the heart is inclosed in an apparently bony case. The adherent pericardium is not unfrequently reported in medical journal literature as a congenital absence of this sac, and the calcification of an exudation, as the formation of a true bony envelope of the heart. The fluid exudation may persist notwithstanding the formation of neo-membrane and bands of adhesion, and it changes in quantity, now increasing while fresh deposits of fibrinous substance is occurring, now diminishing with a temporary amendment; sometimes assuming a hæmorrhagic character, but more frequently becoming purulent. The more solid and unorganized exudation, crossed here and there by bands of adhesion, assumes a grayish color, and undergoes ultimately a caseous transformation.

The muscular tissue of the heart becomes diseased by reason of the proximity of the inflammation—an acute myocarditis—which affects the muscular tissue in contact with the inflamed membrane. The muscular fibers become paler than normal, soften, and are infiltrated with fat-granules, so that the muscular contractility is impaired, and hence, if the lesion extends, the power of the heart will be greatly lessened. The extent of the pericarditis and the duration of the inflammation have a material influence on the extent of the myocarditis. In hæmorrhagic and purulent exudations, the damage to the heart is greater. The strain on the heart due to the increased exertion required in fever, and the compression of the exudation, interfering with the passage of the blood to the muscular tissue of the heart, also affect the nutrition of the organ, and favor degenerative changes. Endocarditis may result by an extension of disease from the inflamed pericardium, as has been experimentally and clinically established. In chronic pericarditis the myocarditis persists, the walls yield to the blood-pressure, and the cavities, the right especially, dilate.

Symptoms.—When an idiopathic pericarditis comes on, the initial symptoms occurring are those of any acute serous inflammation: *malaise*, chill, fever, increased respiration, loss of appetite, frequently nausea and vomiting. Pain of a dull, heavy character, or a feeling of soreness, is felt in the chest, but not invariably. Acute pain in the position of the pericardium is experienced only in those cases with pleuritis of the adjacent portion of the pleura, so that the real significance of any soreness or pain felt is ascertainable only on physical exploration. When pericarditis is secondary to an existing disease, there are no marked disturbances to indicate its onset—no distinctive increase in the temperature and pulse-rate, or in the respiratory

movements, but there may be some præcordial anxiety and oppression, so that, in all cases of diseases in which inflammation of the pericardium is liable to occur, systematic physical exploration of the chest should always be practiced.

The fever movement in simple idiopathic pericarditis is of the remittent type, but in the secondary disease it does not modify that of the existing malady. The state of the circulation varies from a condition of high tension, with full, strong pulse, to great feebleness, low tension, and small, irregular, and unequal pulse. A weak, irregular pulse is characteristic only of cases with considerable effusion, with myocarditis, or exhausted by the severity and duration of this disease. The rational signs of pericarditis possess but little value; but the physical signs are highly significant. In the young, a small amount of effusion may render the præcordial space prominent, but, in adults, only a large accumulation will push out the intercostal spaces sufficiently to produce bulging, unless the lung is shrunken, or there are pleuritic adhesions so situated as to prevent the outward expansion of the pericardium. When there is any considerable distention of the sac and anterior bulging, the nipple of the left side is thrown up higher than its fellow of the opposite side. In consequence of the effusion, the sac of the pericardium is enlarged, and the mobility of the heart on changes of position is increased. Hence, on palpation, this increased mobility is ascertained by the different positions in which the apex-beat can be felt. When the effusion is sufficient to force the heart to a more horizontal position, the apical impulse is farther out and upward. As the effusion increases, filling the sac, the apical impulse becomes weaker and weaker, and is finally no longer felt, as the fluid is interposed between the apex-beat and the chest-wall. When the systole of the heart is weakened by myocarditis, or exhaustion, the apical impulse disappears earlier, especially if there be interposed a thick layer of soft exudation; on the other hand, the apex-beat will be felt longer when there is hypertrophy of the heart, and may not disappear at all if old adhesions keep the apex against the chest-wall. A change of position, as bending the body forward, may cause the apical impulse to be felt again when it had disappeared on the dorsal decubitus. On palpation, for a brief period may occasionally be felt a vibration of the chest-wall, due to the rubbing of the roughened surfaces together. To develop this sensation, firm pressure must be made in the intercostal space with the finger-tips. It is exceedingly rare for this friction fremitus to be strong enough to excite vibrations of the chest-wall, which may be perceived by the hand laid on the præcordial space. It is a rough, jarring, rasping sensation, similar to but quite distinct from the *frêmissement cataire*, or purring tremor, and is not exactly isochronous with the cardiac systole and diastole, although a to and-fro movement.