vening connective tissue, may be the seat of the inflammatory action; consequently there are two forms—parenchymatous and interstitial.

The *parenchymatous* may occur in two forms; in isolated patches, or generally diffused. When a large part of the organ is attacked, there is a marked change in its appearance. The muscular tissue has a reddish color, is puffy in appearance, and the pericardium is spotted with points of ecchymoses, is cloudy, and coated here and there with a delicate exudation. The muscular tissue, on microscopical examination, is found to be cloudy, granular, and swollen, and the striæ indistinct or absent, or the fibers are broken up into granular fragments, are crowded with fat-granules, and ultimately are replaced by rows of fat-granules. When the change is far advanced, the muscle is brownish in color, and almost or quite pulpy in consistence. This change may extend over large parts of the organ, or may be confined to spots or isolated patches, and certain parts of the heart are especially apt to suffer, as the apex of the left ventricle, and, at the base, the posterior wall; next, the aortic valves adjacent to the septum, then the papillary muscle, and, on the right side, the muscular trabeculæ.

Interstitial myocarditis also occurs in two forms: the suppurative and the sclerotic; the former being acute, the latter chronic. Suppurative interstitial myocarditis usually coincides with the parenchymatous; and, between the muscular elements disintegrating with acute fatty degeneration, is seen more or less extensive dissemination of pus, or distinct and isolated collections, or abscesses. When the suppuration is due to emboli, the purulent collections are small, and there are usually several; when the result of interstitial inflammation, there is usually a single large one. An abscess may rupture outwardly into the sac of the pericardium, or inwardly into the cavity of the heart. If situated in the septum, by the discharge a communication is established between the two ventricles, or it may cause a rupture of a segment of the semilunar valve, an example of which has fallen under the author's observation. Again, an abscess in the walls discharging into the ventricle, forms a sac which, bulging outwardly under the blood-pressure, becomes an "aneurism of the heart," so called.* The interior of such a sac becomes lined with successive layers of fibrin, which protects the cavity from rupture, but only for a brief period. When an abscess discharges into the pericardium, a fatal pericarditis results; when the purulent matters and shreds of broken-down tissue enter the ventricular cavity, they produce the disastrous results of multiple embolisms.† Rarely, the pus is absorbed, and a mass of connective tissue and a puckered cicatrix remain to indicate the nature of the disease.

* "Transactions of the Pathological Society of London," vol. xix, p. 149 (with plate).

† Ibid., vol. xx. "A case of abscess of the heart bursting into the left ventricle." Boy of eleven years had a fall injuring his shoulder; delirium, wakefulness, fever, and a very rapid pulse, but no cardiac symptoms succeeded. He died on thirteenth day.

The *chronic interstitial myocarditis* is sometimes called sclerosis of the heart, or fibroid degeneration (Legg) of the heart. It consists in a proliferation—an overgrowth—of the connective tissue and an atrophy of the proper muscular elements. There may be small bands of connective tissue stretching between the muscular fibers, or larger, firm bands, or indurated masses, which take the place of muscular tissue entirely. These bundles or masses of connective tissue occur in the papillary muscle of the left ventricle and in the walls, but more toward the apex than at the base. Two evils result from the presence of these bands and masses of connective tissue and from the resulting muscular atrophy: the propelling power of the heart is reduced and stasis occurs in the venous system; the walls yield at those places composed of the connective tissue, and form the so-called "partial aneurism of the heart." It is especially at the apex of the left ventricle (eighty-five in eighty-seven cases) that these aneurisms form. They vary in size from a pigeon's to a hen's egg, are irregular and divided by partitions and often have diverticula attached, and they contain old deposits of fibrin and recent soft coagula. The walls of these partial aneurisms are composed of the sclerotic material, the endocardium, and the visceral layer of the pericardium with, it may be, the parietal layer attached.*

Symptoms.—The existence of myocarditis can hardly ever be anything but a presumption, based on negative rather than positive signs. If maladies are present, as rheumatism, pyæmia, puerperal fever, etc., in the course of which myocarditis may be expected, if the symptoms of cardiac failure come on suddenly, and if they can not be referred to an endocarditis or pericarditis, then the existence of inflammation of the heart-substance may be suspected. When this disease occurs as secondary to rheumatic endo- or pericarditis, the patient passes rapidly into that condition of profound adynamia known as the *typhoid state*. When an abscess discharges its contents into the cavity of the heart, the symptoms of multiple embolisms are produced; there are repeated violent chills, very high febrile temperature, profuse sweats, icterus, swollen spleen, albuminuria, delirium, or the disturbances due to embolism of the cerebral vessels, etc. The yielding of the sclerosed tissue and the formation of the so-called aneurisms are announced by failure of the heart; the pulse becomes thready, the lips blue, the face anxious, livid, and cyanosed, the respiration embarrassed, the surface cold, the weakness extreme, death occurring in a short time in syncope.

Those cases of myocarditis in which the symptoms of embolism

* Ponfick, Virchow's "Archiv," Band lviii, p. 528.

are wanting, and aneurismal dilatations have not occurred, are characterized by the presence of the following signs: The movement of the heart is feeble, and the apical impulse unfelt; the pulse is small, weak, irregular, and intermittent. The great diminution which has taken place in the propulsive power of the heart manifests itself in stasis, pulmonary engorgement and œdema, cyanosis of the face, swollen veins, vertigo, delirium, etc. In the so-called chronic partial aneurism, there may be no symptoms for a time to indicate the existence of the lesions. We have here the same groups of symptoms, due to the diminished propelling power of the heart, as in the preceding paragraph, when sufficient damage has been done to cause yielding of the cardiac wall.

Course, Duration, and Termination.—The course of the acute form of myocarditis is very rapid, and the duration from two or three to eight days, but some of them terminate in a few hours. Death may be due to rupture of the heart, to cerebral emboli, to pulmonary œdema, to paralysis of the heart, etc. Chronic myocarditis pursues a very latent course. The development of the lesions may be slow, and hence the duration may be prolonged, but not indefinitely. Dilatation of the cavities, feebleness of action, and stasis, will bring on fatal lesions in a few months, or, at most, a year or two.

Treatment.—The treatment must be largely symptomatic, and for parenchymatous carditis is to the last degree inefficient, since the causes are not to be removed. Interstitial inflammation, like the same disease elsewhere, is little influenced by remedies. Minute doses of chloride of gold, or of corrosive chloride of mercury, quinine, and digitalis, offer the best prospect of improvement. The utmost quietude of mind and body must be maintained. A generous diet and means to promote digestion are necessary to improve the quality of the blood.

FATTY DEGENERATION OF THE HEART.

Definition.—A distinction must be drawn between fatty degeneration and fatty substitution: the former implying a change in the structure of the muscular tissue; the latter, a displacement of the muscular tissue, in which atrophy of the muscular elements may take place, by the pressure.

Causes.—The nutrition of the heart is impaired by a variety of causes, intrinsic and extrinsic. Among the intrinsic are pericarditis and myocarditis, which set up an inflammation of the heart-muscle; diminished blood-supply due to atheroma; compression, etc., of the coronary arteries; fat substitution, which, encroaching on the proper tissue of the organ, causes absorption, etc. Among the extrinsic causes are impaired nutrition in general, originating in various ways—cancer, tuberculosis, scrofula, prolonged suppuration, prolonged lactation, etc. Most of the foregoing causes induce atrophy by setting up a fatty degeneration. Anæmia, especially when extreme and long-

continued, has a strong tendency to induce this change. This has been demonstrated experimentally by Perl,* and clinically by Ponfick† and others. In the various causes above given it is the condition of anæmia induced by them which is responsible for the changes in the heart's muscular tissue. Infectious diseases, fevers, and certain poisons, notably phosphorus and alcohol, bring on fatty degeneration. The same result is produced by the mineral poisons in general, but to a less degree, and some other substances. Fatty deposition sometimes takes place to a dangerous extent in the obese, along the sulcus, and penetrating to the endocardium. Furthermore, in the anæmia of the obese, sometimes a very marked condition—fatty degeneration of the heart-muscle—comes on.

Pathological Anatomy.—The change may be general or diffused, or *exist in spots and patches.* The color becomes yellowish, the tissue soft and easily torn, and on the touch makes, in advanced cases, a distinctly greasy impression. The initial change is in the primitive bundles, which become cloudy, granular, and their striæ disappear. Minute oil-globules appear, and are soon seen in rows, but they presently coalesce; large globules are formed, and nothing is then visible in the sarcolemma but a multitude of fat-drops. With this change in the fibrillæ of muscle, an œdematous condition of the sub-serous connective tissue occurs, and the nutrient vessels are advanced in calcareous degeneration. The fatty change may occupy the walls of the left ventricle, or be confined to isolated patches here and there in the walls of the heart, the papillary muscle, the trabeculæ, the septum, etc. In the cases of fatty substitution, the whole heart may be enveloped in a dense layer of fat, which also pushes its way into the muscle, following the inter-muscular planes and the connective tissue, causing such compression that the muscular fibers undergo atrophy, and are pale, thin, and wanting in contractile power.

Symptoms.—Weakening of the heart, produced by fatty change in its muscles, causes the disturbances due to ænemia of the organs and to venous stasis. The rational are more significant than the physical signs. On palpation, the apical impulse is weak. On percussion, there is nothing distinctive, except an increase of the area of absolute dullness, if the organ is enlarged by dilatation of its cavities. As there is venous stasis, and as the right cavities yield more than the left, the area of dullness is increased over the lower end of the sternum to the xiphoid appendix. On auscultation, if there be fatty degeneration of the papillary muscle, a systolic murmur is audible in the mitral area. The sounds of the heart are dull, confused, almost inaudible, and there is often a failure of synchronism in the closure of the valves, causing double sounds. The pulse is small, irregular, intermittent, weak, and

* "Ueber den Einfluss der Anämie auf die Ernährung des Herzmuskels," Virchow's "Archiv," Band lix. p. 39.

† Ponfick, "Berliner klin. Woch.," "Ueber Fettherz," Nos. 1 and 2, 1873.

easily compressed, and may be very slow, falling to forty, often even as low as twenty ; but this is exceptional. A very formidable symptom, which, however, occurs under other circumstances, is a peculiar alteration of the respiratory rhythm, known as the Cheyne-Stokes breathing, in which at intervals the respiration becomes slower and shallower, until finally it seems to cease—is suspended for some seconds, half a minute, for a minute—and then is resumed, slow and shallow, but gradually attaining its normal amplitude. This may be kept up for some time, then disappear, to occur again. The diminished propulsive power of the heart, causing anæmia of organs, induces characteristic symptoms. Sudden anæmia of the brain, faintness, and actual fainting, often occur on rising up suddenly from a recumbent posture, stooping, turning around quickly, etc. These subjects experience constantly, or nearly so, a sense of fullness and distention about the ensiform cartilage or lower sternum, which is associated with præcordial anxiety, and they have attacks of angina pectoris.* They experience difficulty of breathing on slight exertion, and can not ascend elevations or stairways without experiencing great distress. The veins of the neck are habitually distended, and the countenance looks dusky and anxious. The legs become œdematous ; next, the body generally; the liver enlarges, ascites forms, the urine becomes albuminous, etc.

Course, Duration, and Termination.—Acute fatty heart, produced by the action of poisons, terminates early ; but the cases due to the ordinary causes proceed more slowly, and may last during several years. Their development is obscure, and there are no pronounced symptoms until those of failing heart come on. The termination is in general dropsy, or death is caused by œdema of the lungs, or takes place suddenly by paralysis of the heart, or by rupture of the organ.

Diagnosis.—If the causes of fatty degeneration have existed, and symptomsof cardiac weakness come onslowly,the existence of fattyheart may be regarded as probable, but the diagnosis is largely the balance of probabilities, and is not to be arrived at by exclusion with certainty.

Treatment.—As anæmia plays so important a part in the causation of fatty degeneration of the heart, the treatment should be directed to the enrichment of the blood. Iron, manganese, and strychnine (the sulphates), is an excellent combination. The author has seen good results from the phosphate of iron, quinine, and strychnine, in the form of the elixir. Jaccoud prefers caffeine to digitalis as a heart-tonic in these cases; See urges its use in the same condition, and the author has found it to be as excellent a remedy as its advocates assert. It must, however, be given in sufficient doses, from five to fifteen grains *ter in die.* The efficacy of opium, or, better, small doses of morphine, as a tonic of the heart, is too little understood, especially in the form of hypodermatic injection. Inhalations of oxygen-gas, the internal use of

* J. Lockhart Clarke, "St. George's Hospital Reports," vol. iv, p. 1.

cod-liver oil, and general faradization of the muscles, are expedients of high utility. Nitro-glycerin is a remedy of the greatest value in these cases, or when the heart is weak from any cause. By lowering the peripheral tension of the vascular system, it lessens the work of the heart, and thus obviates a serious danger. By dilating the arterioles of the muscular substance of the heart, it promotes the nutrition of the organ, and in this consists one of the most valuable results of its administration. It relieves the sense of precordial distress, the faintness and vertigo, and other unpleasant consequences of the cerebral anæmia. When the blood-pressure is low because of a relaxed state of the arterioles, barium chloride (gr. $\frac{1}{4}$—gr. $\frac{1}{2}$) is the most efficient of the remedies now in use.

RUPTURE OF THE HEART.

Definition.—Under the designation of *rupture of the heart* is meant the so-called spontaneous rupture, in contradistinction to rupture by wounds and injuries.

Pathogeny and Symptoms.—That rupture shall occur it is necessary that the walls of the heart be weakened by disease. The most frequent cause is fatty degeneration, for, in twenty-four cases, this condition of the muscular tissue was found in nineteen.* Next in importance as a cause is the softening produced by acute myocarditis, especially the suppurative form, or the aneurisms, so called, due to the changes of chronic myocarditis. Diseases of the coronary artery, tumors, echinococci, by destroying muscular tissue, lead to rupture. It is more common in men than in women, and in old age—after sixty years. As to the site of the rupture, statistics show that the left ventricle, at or near the apex, next the right ventricle, then the right auricle, are the most usual ; but the preponderance is immensely on the side of the left ventricle—forty-three times in fifty-five cases. There is usually but a single rent, but there may be several, and, as they follow the direction of the muscular bands and the line of least resistance, they are tortuous, somewhat jagged in their margins, and the two orifices are not opposite. The size of the rent varies from an inch to the whole length of the cavity. The pericardial sac contains more or less blood, according to the size of the opening. The rupture may be gradual, a part yielding at a time. Death may take place almost instantaneously. Usually, a groan or a cry is uttered, the face grows deadly pale, the individual falls unconscious, there is some shuddering, and he is dead. The dying may extend over several days—the patient experiencing the symptoms of *angina pectoris* several times with intervals of partial relief, death occurring suddenly at last. In such cases, it is assumed that successive portions of the heart-wall yield, or that clots temporarily obstruct the rent.

The treatment, when there is time for it, is purely symptomatic.

* "Berliner klinische Wochenschrift," 1873, p. 15 ; Ponfick, "Ueber Fettherz."

HYPERTROPHY AND DILATATION OF THE HEART.

Definition.—By hypertrophy of the heart is meant an increase of size of the organ, because of an addition to its substance. This enlargement takes several directions, as follows :

Simple hypertrophy means an increase in size without alteration of the cavities ; *concentric hypertrophy* means increase in thickness of the walls, the cavities becoming smaller ; *excentric hypertrophy* means increase in the thickness of the walls, the cavities becoming larger.

The dilatations of the heart correspond in arrangement as follows :

In *simple dilatation*, the cavities are enlarged while the walls remain normal ; in *active dilatation*, which corresponds to excentric hypertrophy, the cavities are enlarged, and the walls are increased in thickness ; in *passive dilatation*, the cavities are enlarged and the walls are thinner. This is the most usual form.

The conditions attendant on hypertrophy and dilatation are, in some respects, the same, so that it is an economy of space, and contributes to clearness of conception, to study them together.

Causes.—*Hypertrophy.*—Simple hypertrophy, which is by no means common, arises from over-action of the cardiac muscle, without there being any disease of the circulatory apparatus. The over-action is due to the abuse of such stimulants as coffee, tea, tobacco ; to moral emotions and intellectual effort, when excessive ; to repeated muscular fatigue, etc. The hypertrophy resulting in this way is general. Any obstacle to the free circulation of the blood imposes additional work on the heart. Narrowing of the aortic orifice gives the left ventricle more work to do, and hence its muscular fibers undergo hypertrophy ; in the same way, hypertrophy of the right ventricle results from narrowing of the pulmonary orifice, of the left auricle, from mitral stenosis, and of the right auricle, from tricuspid stenosis. These are typical examples of partial hypertrophy. The causes of obstruction in front, inducing hypertrophy of the left ventricle, are several : stenosis and regurgitation at the orifice of the aorta ; narrowing of the artery at the duct of Botal ; aneurism, and compression of the vessel by tumors ; atheroma of the arterial system. Hypertrophy of the left auricle results from obstruction and regurgitation at the mitral orifice, especially narrowing of the orifice. Similar causes produce similar effects on the other side. Hypertrophy of the right ventricle is due to narrowing of the pulmonary orifice, to aneurisms, and tumors compressing the artery, to chronic pulmonary diseases which obstruct the circulation, as emphysema, caseous pneumonia, fibroid lung, large pleural accumulations, etc. Hypertrophy results from, or is an attendant on, Bright's disease. Various explanations have been offered of the nature of this relationship, but it is clear that, if hypertrophy of the muscular layer of the arterioles exist in front, the heart has increased resistance, which requires additional effort to overcome. Hypertrophy is, so to speak, a physiological result of the changes in the arterial system due to age ; for the calcareous deposition in the tunics of the aorta and of the vessels generally greatly increases the resistance of the arterial circuit by diminishing the elasticity.

Dilatation.—Simple dilatation of the heart occurs in delicate constitutions, especially of growing youths, subjected to over-exertion. This has been observed in armies on a large scale, and by civil physicians as well.* Maclean † has published observations on this point made in the English service ; Seits and others in Germany ; but Da Costa was the first to set the subject in its true light, by studies in our hospitals during the late rebellion, and preceded all other investigators in this line.

The right ventricle, being much feebler than the left, is more liable to suffer dilatation. This condition results from the increase of pressure due to insufficiency of the semilunar and tricuspid valves, and pulmonary lesions which hinder the circulation in the pulmonary capillaries, such as emphysema, chronic bronchial catarrh, chronic interstitial pneumonia, and tubercular and caseous infiltration. On the left side the most frequent cause is aortic obstruction and insufficiency ; but obstruction rather than insufficiency is more certain to produce the dilatation. Mitral insufficiency leads to dilatation of the right cavities by maintaining constantly an increased pressure in the pulmonary capillaries. The cavities yield under normal pressure of the blood when altered by disease. Pericarditis and endocarditis affect the condition of the muscular tissue, by setting up a myocarditis—a granular degeneration. Myocarditis arises under other circumstances also, and the heart-muscle is weakened, not by this disease only, but by fatty degeneration, fatty substitution, tumors, etc.

Pathological Anatomy.—In hypertrophy the change may be confined to one part, or the whole organ may be involved. To such enormous proportions does the heart attain sometimes as to be called *cor bovinum*—ox's heart. The walls of the left ventricle may increase to an inch, an inch and a half, or even two inches in thickness, and the walls of the other cavities undergo corresponding development. The shape of the heart is altered by hypertrophy. When there is hypertrophy of the right ventricle, the heart is widened transversely and the apex is blunted ; when the left ventricle is enlarged, the heart is elongated, and, if its cavity is at the same time enlarged, the septum is pressed over into the right ventricle. When both ventricles are enlarged, the heart assumes a globular shape. The position of the hypertrophied heart is more horizontal ; if the left ventricle is the seat of the change, the direction of the organ is to the left and downward. By reason of an increase in weight the heart in the recumbent posture sinks relatively lower, and hence the area of absolute dullness may appear smaller ; in the vertical position the heart descends, pushing

* Dr. O. Fräntzel, "Ueber die Entstehung von Hypertrophie und Dilatation der Herzventrikel durch Kriegsstrapezen," Virchow's "Archiv," Band lvii, S. 215.

† "The British Medical Journal," February 16, 1867.

the diaphragm before it, and making the epigastrium more prominent. In texture, the substance of the heart is firmer than normal, and when divided has sharp edges which remain apart. In color, the tint is brighter and fresher looking than in the healthy state. Subsequently, if fatty change begins in isolated patches, the reddish-brown hue of the muscle will be marked by spots of a faintly yellowish or reddish-yellow color. It seems to be well established that the increase in the muscular tissue of the heart is a true hypertrophy, and not a hyperplasia, that the existing elements are increased in size, but that no new elements are formed. Dilatation occurs chiefly in the auricles, which may be so stretched that the muscular elements undergo fatty degeneration, are absorbed and disappear, leaving the endo- and pericardium in contact, or separated by some connective tissue only. The size to which the auricles may be expanded is enormous. The right ventricle may be much dilated and its walls thinned ; the orifices may be much enlarged, the trabeculæ wasted, and the valves thinned. The left ventricle is rarely dilated merely, but the walls are also hypertrophied.

Symptoms—*Hypertrophy.*—The signs and symptoms of cardiac disease are divisible into two groups—*rational, physical.* The rational signs are presumptive, and consist of the functional disturbances which indicate the probable seat of the disease ; the physical signs are derived from physical laws and methods, and are positive in their results. As respects the rational symptoms, the first point to be noted is, that those vessels receiving their blood-supply from an hypertrophied ventricle obtain more blood and with greater force than in the normal condition, and hence the tension in these vessels is higher ; whereas, the vessels on the other side receive less blood with diminished force, and their tension is lower. When the left ventricle is hypertrophied, the tension is increased in the aortic system and diminished in the pulmonary. The opposite condition obtains when the right ventricle is enlarged, for then the pressure is greater relatively in the pulmonary system and less in the aortic. When both ventricles have undergone hypertrophy, the tension is increased in the aortic system and in the pulmonary artery. In consequence of the increased distributing power of the left ventricle, the blood-current is accelerated in the arterial system and communicating capillaries, and, as the pulmonary circuit has also a higher tension and greater celerity, the blood received from the great venous trunks is quickly disposed of, so that the tension falls

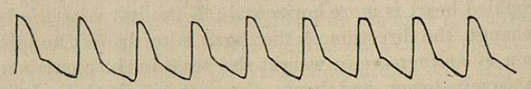

Fig. 22.—Hypertrophy.

in the venous radicles. The final effect of pure hypertrophy is an acceleration in the whole round of the circulation. The pulse is full, firm, and bounding. The ascent line of the sphygmographic trace is

vertical and abrupt, but the summit is rounded and the descent oblique, unless there be regurgitation at the aortic orifice. The face is red and congested ; the nose bleeds easily ; the head feels full, and aches a good deal, especially when any strong muscular effort is made ; there are more or less *tinnitus aurium* and dizziness. When the arterial walls are weakened by atheromatous degeneration, cerebral hæmorrhage may be a result of hypertrophy of the left ventricle ; but the way to rupture is prepared by gradual yielding of the arterial tunics, and the formation of minute aneurismal dilatations known as "miliary aneurisms." The strong beating in the superficial arteries is felt by the patient, and produces a disagreeable roaring and beating in the ears, especially when lying on the left side. The attacks of palpitation are frequent, but their severity is not in proportion to the extent of the hypertrophy, for the action may be very tumultuous when the enlargement is slight, and *vice versa.* There are pretty constantly felt by the patient a sense of præcordial anxiety, and, rarely, attacks of pain extending to the shoulder and arm, similar to *angina pectoris.* A sense of fullness in the chest, of oppression, and sometimes embarrassed breathing are experienced, but the pulmonary symptoms may be due to congestion of the bronchial mucous membrane, supplied as it is by the bronchial arteries, and not from the pulmonary. When the hypertrophy is confined to the right ventricle, no other lesion existing—an extremely rare condition—the symptoms present will be a sensation of fullness and oppression of the chest—possibly dyspnœa ; œdema and hæmorrhage may occur, and the production of interstitial inflammation and possibly other diseases promoted. The foregoing signs of hypertrophy are presumptive or rational ; the physical signs now to be considered establish the seat and character of the lesion. On *inspection* there is to be observed a prominence of the chest, greatest at the junction of the fourth and fifth ribs with the sternum. This has been denied; but, that it is often encountered in hypertrophy occurring in young subjects, the author's experience entitles him to affirm. When hypertrophy occurs later in life, the ribs having become rigid, no elevation of the chest-wall can be effected, how powerful soever may be the impulse of the heart. As in hypertrophy, the position of the heart is more horizontal and depressed to the left, on *palpation*, the apical impulse is felt near to the axillary line, and one, two, and possibly three intercostal spaces lower down, and it is stronger and more widely diffused. The force of the impulse is sufficient to raise the hand when placed on the cardiac region, or the head when applied in auscultation, and the whole left thorax may be felt lifted up and carried toward the left. This is entitled *the heaving impulse*, and is very characteristic of extreme hypertrophy. Instead of the impulse having a heaving character, sometimes it makes the impression of a sudden jar which is immediately arrested. In hypertrophy of the right ventricle the heav-