ing impulse is felt at the end of the sternum, especially its right border, and in the epigastrium. In the third and fourth intercostal spaces to the right of the sternum, the impulsion of the hypertrophied auricles may sometimes be felt. On *percussion*, the area of præcordial dullness can be demonstrated. The *absolute* or superficial dullness is that derived by percussion over that portion of the heart uncovered by the lung—a triangular space ; the *relative* or deep dullness is that obtained by strong percussion over that portion of the heart covered by the lung. The dull space extends from a point internal to the upper border of the second rib at its junction with the sternum, obliquely downward to the left to the apex-beat, thence transversely to the right border of the sternum. This is an irregularly triangular or ovoidal space which returns, on percussion, the forms of dullness mentioned above. The area of absolute dullness is increased by hypertrophy of the heart, if the patient is percussed when erect and inclined slightly forward. The relative dullness is increased more when the patient is recumbent, by the heart sinking backward. In hypertrophy of the left ventricle, the dullness is parallel to the long axis of the heart ; in hypertrophy of the right, the dullness is over the lower extremity of the sternum.

When pure hypertrophy is the condition under examination, *auscultation* furnishes no important information. The sounds of the heart are somewhat affected in their timbre. In hypertrophy of the left ventricle, the first or ventricular sound has a rather metallic quality, and the second sound is strongly "accentuated" ; in hypertrophy of the right, the same facts exist, but the sounds are less intense. At the apex, a peculiar metallic "click" is sometimes heard, and is doubtless due to the vibration in the chest-well, produced by a very strong impulse. It is much louder when the stomach is distended with gas.

Dilatation.—When dilatation occurs in any of its forms, the propulsive power of the heart is diminished ; less so, however, in *active dilatation.* The result of this is a condition of ischæmia in one set of vessels, and of stasis in the other system. Thus, when the left ventricle is dilated, there is a lowering of tension in the aortic system, and an increase of pressure and abnormal fullness of the pulmonary; when the right ventricle is dilated, there are diminution of tension, and ischæmia of the pulmonary artery, and elevation of pressure with stasis in the peripheral venous system. The ultimate effects of the disturbance in the vascular system are the same when one ventricle is dilated as if both were, for, taking as an example the most common dilatation, that of the right side of the heart, the stasis in the peripheral veins extends to the capillaries, to the arteries, thence to the left side, and *vice versa.* When, however, dilatation of the right ventricle coincides with hypertrophy of the left, the excess in power of the one compensates for the deficiency in the contractile energy of the other. The results of dilatation of all the cavities are these : the vessels receiving blood from

the heart—efferent vessels—are in a condition of ischæmia, or diminished blood-supply, while the vessels conveying the blood to the heart —afferent vessels—are constantly abnormally full, or in a condition of hyperæmia and exaggerated tension. When the right heart is dilated, there are ischæmia of the pulmonary vessels, producing habitual dyspnœa, insufficient hæmatosis or aëration of the blood, and stasis in the general venous system. The peripheral veins are turgid with blood, there is cyanosis from deficient aëration, and a constant hyperæmia of the liver, spleen, kidneys, and intestinal canal. Increase of pressure in the renal veins causes *albuminuria ;* in the hepatic veins, jaundice and ascites ; in the veins of the extremities, œdema and general dropsy, and thrombosis. The rational symptoms of these functional disturbances are, palpitations of the heart ; frequency and irregularity of the pulse ; deficiency in the arterial blood-supply to the brain, and manifest in vertigo, ringing in the ears, attacks of faintness or actual syncope, etc. ; deficiency in the blood going to the lungs, and causing cough, dyspnœa, etc. The composition of the blood is impaired by the excess of carbonic acid ; the lessening of the oxidation processes diminishes the production of heat, and hence the general temperature is low ; the vessels themselves, the heart, and the tissues, undergo nutritive changes in consequence of insufficient energy in the process of tissue metamorphosis. A cachectic state, with lowered vitality of the tissues, so that they ulcerate under the least irritation, is the necessary outcome of these changes. There is not only a lowered state of the assimilative functions, but elimination is imperfectly carried on, and excrementitious materials are retained in the blood—carbonic acid and urea—causing hallucinations, delirium, eclampsia, coma, etc. The ill results of these nutritive alterations are also exhibited in increased damage to the heart-muscle, and consequently an exaggeration of the mechanical effects of the dilatation. Inspection furnishes no information of value, except, when dilatation of the right cavities render the valves incompetent, a venous pulse will be visible in the neck. On palpation, the area of cardiac impulsion is as wide as in hypertrophy, but the apical impulse is feeble, and may not be felt when the patient is recumbent. When there is hypertrophy of the right heart to compensate for dilatation of the left cavities, the apical impulse will be feeble, while the pulse of the right cavities at the border of the lower sternum will be comparatively strong. On percussion the extent of dullness is made out as in hypertrophy. On auscultation, the sounds are feeble, as a rule ; on the other hand, they may have a more clear and resonant quality. A soft-blowing murmur sometimes takes the place of the first sound. This murmur is situated in the mitral and tricuspid areas, and is due to the insufficiency of the valves to close the auriculo-ventricular orifices.

Diagnosis.—Hypertrophy is to be distinguished from dilatation of

the heart, from pericardial effusions, tumors of the mediastinum, etc. The force of the impulse, the accentuation of the second sound, and the state of the systemic circulation, enable the differentiation to be made from dilatation, and also from effusion; besides, in the latter, the dullness has been preceded by a friction-sound, and, when the effusion comes on, the heart-sounds weaken and disappear. The seat of the dilatation is determined chiefly by the position of the dullness. Hypertrophy and dilatation are differentiated from tumors in the mediastinum, by the displacement of the heart occasioned by the latter, and by the persistence of the normal heart-sounds. The pressure of a tumor on the great vessels and important nerves introduces into the symptomatology of the case new symptoms quite foreign to either hypertrophy or dilatation. From pleuritic effusion in the neighborhood, retained by adhesions—the so-called encapsulated—the dullness due to hypertrophy or dilatation may be difficult to separate, but effusions displace the heart without altering the character of its impulse and its murmurs; when the pleural effusions are unconfined, the ready distinction consists in the change of the position of the patient, shifting the dullness.

Course, Duration, and Termination.—The course of these affections is chronic, but hypertrophy continues much longer than dilatation. Hypertrophy, uncomplicated, exists unchanged for many years, and is important rather on account of the complications which may grow out of it than of itself, yet changes in the heart-substance and in the vessels must eventually result. Over-supply of blood to organs leads to nutritive alterations in them. Rupture of vessels may take place, but disease of the arterial tunics is necessary also; hence the importance of hypertrophy of the heart as a factor in cerebral and in pulmonary hæmorrhage. Dilatation of the cavities is much more rapid in its course and important in its results than hypertrophy, but simple and passive dilatations are more serious than the active form. The heart is much weaker, its tissues become diseased, and death may be sudden by paralysis or by rupture, or in attacks similar to angina pectoris. The stasis in the circulation, the pulmonary, hepatic, and renal troubles, and the general dropsy which result from dilatation, are the usual sequelæ, and death ultimately occurs from the combined effect of these disturbances.

Prognosis.—The prognosis is necessarily grave, but it should always be guarded. Simple hypertrophy may exist for years, without any apparent interference with function. In dilatation, the hope of any lengthened period of freedom from ill results can not be encouraged. When dropsy appears, it becomes a question of the physical endurance largely, for death can not, then, long be delayed.

Treatment—*Hypertrophy.*—When hypertrophy is compensatory or compensated, there is no need of therapeutical measures. It may, however, be necessary to combat the hypertrophy, or its results in the organism at large, if the force of the heart and the pressure in the vascular system are so great as to threaten serious consequences. The most direct method is the abstraction of blood, either by venesection or by leeches, and this is allowable in vigorous subjects. Purgatives lower the blood-pressure, especially the saline purgatives, which draw off by the intestinal mucous membrane more or less fluid. They are much less objectionable than bloodletting, are more easily handled, and are more permanent in results. Next to saline purgatives in efficiency is the tincture of aconite-root. Tincture of veratrum viride is more powerful, but less easily managed, for its effects are quickly produced and not easily confined within the prescribed limits.

The treatment of dilatation must pursue the opposite direction. The general nutrition must be maintained at the highest point, to promote the nutrition of the cardiac muscle. A generous diet, moderate exercise in the open air, the inhalation of oxygen, are important agencies to accomplish the objects just mentioned. Bitters to increase the appetite and iron to improve the quality of the blood are strongly indicated. To tone up the heart and raise the tension of the vascular system, there is no remedy so efficient as digitalis. It should be given with quinine, which is also an excellent heart-tonic. The most remarkable effects attend the use of minute doses of morphine hypodermatically in these cases. When there is extreme dyspnœa, the heart very feeble, the fluid everywhere gaining, the effect of the injection is almost magical. It sometimes happens that the symptoms are too urgent to await the slow action of digitalis, or it may be the stomach will not tolerate the digitalis in any form, then the injection is most opportune—the patient is relieved by it—time is gained for the action of the various remedies that may be substituted for it, or the stomach will bear it better. A decided change has taken place in the state of professional opinion on the subject of exercise in heart-diseases. From absolute quiet, or the avoidance of anxiety and fatigue, opinion has gone in some extreme instances to mountain climbing. It need hardly be said that the medium is the true ground to take. Daily, systematic, walking exercise, such as will improve the forces of the body, contributes to improve the heart nutrition and the increased muscular action its muscular power.

Careful attention should be given to the diet of these patients. In many instances acidity of stomach causes reflex palpitations of a very hurtful kind, and any articles difficult of digestion, especially if nausea follows, is apt to excite paroxysms of pseudo angina pectoris.

ENDOCARDITIS—INFLAMMATION OF THE ENDOCARDIUM.

Definition.—Acute inflammation occurs in two distinct forms: plastic, or simple exudative inflammation; ulcerous, or diphtheritic

inflammation. The plastic form is either acute or chronic, but these differ merely in degree and rate of progress.

Causes.—Primary or idiopathic endocarditis, except in the ulcerous form, is extremely rare. Plastic endocarditis is usually a secondary affection : secondary to pleuritis, pneumonia, pericarditis, myocarditis, etc., but, very much more frequently, secondary to acute rheumatism. The relative frequency of endocardial inflammation in acute rheumatism is differently stated by different observers. According to some, one half, others one third, of the cases are complicated by endocarditis, but the real number is, no doubt, lower than one third. The source of error is the occurrence of a soft-blowing murmur in cases of rheumatism, due not to inflammation of the endocardium but to the condition of the blood. The more severe the type of rheumatic fever the greater the danger of cardiac complications, but there are numerous exceptions to this rule. The pericardial and endocardial inflammation may precede the joint-troubles.

Pathological Anatomy.—The initial lesion is hyperæmia, which involves the sub-serous connective tissue as well as the membrane itself. The stasis in the vessels induces rupture of the capillaries, here and there, and minute extravasations are thus formed. Migration of white corpuscles, exudation of fibrinogenous and germinal matter, now takes place into the affected membrane, and the cells of the endothelium become cloudy, loosen, and undergo proliferation. The membrane, which in health is thin, transparent, and glistening, becomes, as a result of these changes, rough, opaque, and thickened. The roughness of the membrane is due, further, to the formation of lamelliform or conical vegetations, the product of the activity in cell proliferation at particular parts, or, according to Rindfleisch, they are composed of an homogeneous fibrinous exudation from the vessels. If the changes in the structure of the membrane do not go beyond this point, it is probable that complete restitution may occur. Proceeding from this point the inflammation may take the *plastic* or the *ulcerous form*. We are now concerned with the former only. The exudation on the auriculo-ventricular valves (mitral) is found chiefly at the free border, where the tendons are inserted ; on the semi-lunar valves (aortic) on the lateral border where the segments come in contact, yet the corpora arantii may also be the seat of abundant exudation. The vegetations projecting from the surface of the membrane entangle masses of fibrin whipped out of the blood, which may project from the valves, swinging to and fro like a polypoid excrescence. The chordæ tendinæ may be affected in a manner similar to the valves. Softened by the inflammatory process, the chordæ may give way, permitting a segment to become adherent to a neighboring one. Adhesion of the semi-lunar valves may occur at the side where they are in contact. The adhesions undergo organization, and thus the most serious changes are

wrought in the structure and functions of the valves. Also, large masses of fibrin may be entangled in them, and they may be the cause of thrombotic deposits around them. When the inflammatory process passes to the chronic stage, characteristic changes take place in the exudation : it loses some part of its water, solidifies, and subsequently contracts. The connective tissue undergoes hyperplasia, especially the connective tissue of the borders of the valves, but the membrane, generally of the valves, may be affected by the same change. As a result of the tendency of the new material to contract, the valves become much deformed, thick, and inflexible, and, of course, their functions are correspondingly impaired. Calcareous changes occur in the deposits, and fatty degeneration also takes place. Patches of softening may occur in the valves, the membrane yields, and pouches or aneurisms form, which ultimately give way, and thus a valve is perforated. This process, occurring at various points, imparts to the valve a sieve-like appearance. Vegetations detached, or bits of adherent fibrin cast off, constitute emboli, which, entering the blood-current, will be deposited in distant parts—on the left side of the brain, in the kidneys, spleen, etc. The orifices of the valves undergo similar changes. The connective-tissue transformations take place, and hence rigidity, deformities, and contraction result.

Symptoms.—When endocarditis is idiopathic, which is very rare, its onset is marked by the usual symptoms of an acute febrile or inflammatory affection. There is a chill, followed by fever, a coated tongue, anorexia, nausea, sometimes vomiting, and general *malaise*. As it occurs in the course of another disease, the additional disturbance induced by it may altogether escape recognition, and it is only by persistent watchfulness, under such circumstances, that it is discovered. This is true of its onset in rheumatism, Bright's disease, the eruptive fevers, etc. On the other hand, the commencement of endocarditis may be manifest by very obvious signs. For example, if during the course of acute rheumatism endocarditis comes on, there will occur an increase in the temperature, the thermometer rising a degree or two, the pulse will become more rapid, and the general condition less favorable, than before the complication arose. The fever does not pursue a special type, and the pulse exhibits no characteristic quality. The other rational symptoms are equally indefinite. There may or may not be some uneasiness in the region of the heart, some præcordial oppression, and some palpitation. There may occur, also, increased impulsion of the heart, more rapid and tumultuous beating of the carotids, headache, noises in the ears, some dyspnœa, etc. After a time the action of the heart becomes less energetic, the strength of the pulse declines, the function of hæmatosis is impaired, and hence the functions generally, especially the cerebral, are less energetically performed. The physical signs are much more distinctive than the ra-

tional ; the changes in the valves and at the orifices necessarily modify the character of the murmurs, or add new sounds. The period and position of the murmur are determined by the valve affected and by the time, in the cardiac revolution, when the blood-current passes the affected orifice. In mitral insufficiency a *bruit* or murmur is audible with the first sound (systolic) at the apex, and with the second sound (diastolic), or after it (presystolic), if there is obstruction at the mitral orifice. In aortic obstruction the murmur is audible with the first sound (systolic) at the base, and with the second sound (diastolic) if the aortic valves are insufficient. If the lesions occur on the opposite or right side of the heart, which is very rare, the same rules obtain, but the position at which the sounds are heard is different. To hear the sounds at the right auriculo-ventricular orifice, the ear must be placed over the ensiform appendix, and, for the pulmonary valves, at the junction of the third right rib with the sternum. Percussion affords but little information. If there be aortic obstruction, some distention of the heart is occasioned, which increases the area of dullness in the vertical direction ; if mitral obstruction, the right cavities will be somewhat dilated and the dullness increased in the transverse direction. The facts may be formulated as follows : In acute endocarditis the same physical signs characteristic of chronic valvular diseases of the heart occur suddenly ; and, further, the sudden development of the symptoms of mitral insufficiency is the most characteristic sign of acute endocarditis (Jaccoud). Obstruction or regurgitation at the mitral orifice increases the pressure of the blood in the pulmonary artery, and hence a physical sign of this condition is accentuation of the pulmonary second sound. More or less congestion of the lungs and stasis in the venous system are necessary consequences of mitral disease.

Course, Duration, and Termination.—The course of acute plastic endocarditis is necessarily brief. The patient either partially recovers by the disease assuming the subacute and chronic phase, or he dies from the immediate consequences and complications. When the case passes from acute to chronic, the fever ceases, compensation takes place, by which the disorders of circulation are obviated for a time, yet the physical signs of valvular mischief continue. Death may result from a gradual weakening, terminating in paralysis of the heart, or heart-clot may form, or a cerebral embolism occur. Pericarditis, myocarditis, and pneumonia, may also intervene and take life. That a cure of actual lesions may happen is admitted, but the examples of such a fortunate termination are extremely infrequent. The duration of the acute attack is short ; of the subacute and chronic form, indefinite.

Diagnosis.—The differentiation consists in the application of the physical signs. It should not be forgotten that a murmur exists of a soft-blowing character, not due to valvular lesion, and which disappears on the subsidence of the acute symptoms.

Prognosis.—The acute form is not very dangerous to life, and hence a favorable prognosis may be expressed. As regards the ultimate results of valvular lesions, the prognosis is grave.

Treatment.—The character of the associated malady and the condition of the patient must enter largely into the consideration of remedies. As it is a fundamental principle to keep the suffering organ quiet, remedies capable of effecting this are very important—these are, ice and digitalis. An ice-bag should be applied to the præcordial region, and a tablespoonful of infusion of digitalis given every four hours. Flying-blisters should be applied to the axillary region. In the incipiency, before much damage has been done, there can be no doubt of the great efficacy of the hypodermatic injection of morphia, or the internal administration of morphia and quinine—one quarter grain of morphia and ten grains of quinia every four hours until three or four doses are taken. When considerable exudation has occurred, besides the remedies to quiet the heart, ammonia should be given freely, with the view to exert a solvent action. The best form for administration is the carbonate (ten grains) in the solution of the acetate (half an ounce) every four hours, or half the quantity every two hours. If there be much depression in the progress of the case, quinine and digitalis should be prescribed in combination, or caffeine, strophantine, chloride of barium, and other remedies of the kind should be given.

ULCERATIVE ENDOCARDITIS—DIPHTHERITIC ENDOCARDITIS.

Definition.—This is a peculiar form of disease, in which ulcerations and diphtheritic exudations, with colonies of micrococci, develop in the endocardium, followed by septic infection of the blood and multiple embolisms.

Causes.—A peculiar state or type of constitution seems necessary to develop this disease. It occurs during the course of some cases of acute rheumatism, of puerperal fever, of diphtheria, etc., and now and then this process attacks the valves in cases of chronic plastic endocarditis, the new material undergoing rapid and destructive ulceration. This disease makes its appearance between puberty and forty. A depressed condition of the vital forces, due to bad hygienic influences, seems to be very influential in determining the occurrence of this disease in youths. The close analogy between the diphtheritic process and this ulcerous disease of the left heart and the frequent coincidence of the two affections render it highly probable that the diphtheritic poison is the chief if not the only factor in its causation.

Pathological Anatomy.—The initial lesions are the same as those described under the head of plastic endocarditis. The lesions are chiefly on the left side of the heart, and attack by preference the anterior flap of the mitral and the semi-lunar valves of the aorta ; next the walls of the appendages to the left auricle ; and, lastly, the walls of

the ventricle. Occasionally the same morbid process occurs on the right side, and, in one reported case, on the tricuspid only,* and its chordæ tendinæ, which were destroyed. After the initial changes already described, the nuclei of the connective tissue undergo rapid proliferation and form granulations of the surface; fibrinous deposits take place, and the whole forms a "felt-like" mass, intimately connected with the tissues beneath. A process of softening then begins in the interior of these masses; they crumble and fall away, and leave a ragged, irregular ulcer, which is the seat of fresh fibrinous deposits. Perforation of the valve may ultimately take place, and the margins of the perforation are rough, ragged, and ulcerated; and they are surrounded by granulations having the same structure as those which have already ulcerated. A distinctive peculiarity of this process is the presence early in the course of formation of the granulations, and in the midst of the proliferating connective-tissue corpuscles, of a finely granular material, the particles having various shapes, strongly refractive of light, and resisting the action of acids and alkalies. These granules, as Virchow was the first to point out, are micrococci, and the granular masses are colonies of micrococci. The losses of substance by thinning the valves lead to the formation of the so-called valvular aneurisms, and coagula forming in these are thrown off with patches of diseased tissue, when the aneurism gives way. Ulceration of the septum, induced in the same way, leads to communication between the cavities. The particles of ulcerating tissue, of fibrin and blood-clot, and the little masses of micrococci colonies thrown off into the blood-current, form multiple embolisms. Two results follow: either there is merely mechanical obstruction of vessels, or an infective process is set up the same as that of the original disease. The spleen, kidneys, and brain, are the organs in which these deposits take place from the left side of the heart. When the disease is in the right side of the heart, the emboli are swept into the lungs.† As these organs contain the "terminal arteries" of Cohnheim, there will occur hæmorrhagic infarctions and ichorous suppuration. All the organs of the body may, indeed, be the seat of abscesses from embolic deposits. The distribution of infective materials—specific micrococci—sets up a general infection of the blood. Wherever the micrococci are deposited they undergo rapid multiplication, and initiate the same morbid action as at the original source of infection. Numerous are the alterations occurring in various organs in ulcerative endocarditis. The spleen is very much enlarged, whether the seat of infarctions or not; in the kidneys are abscess formations, and the afferent vessels are blocked with colonies of migrating micrococci; in the brain there

* T. Whipham, M. B., "Transactions of the Pathological Society," vol. xxii, p. 118.
† C. J. Eberth, Virchow's "Archiv," Band lvii, "Ueber diphtherische Endocarditis."

are extravasations, especially of the meninges; in the lungs, abscesses from emboli; in the heart, myocarditis and pericarditis; and in the small intestine, swelling of the patches of Peyer and solitary glands, and ulcerations which differ from those of typhoid, in that they are not confined to the lower extremity of the ilium, are not opposite the insertion of the mesentery, and are not limited to the glands.*

Symptoms.—Cases of ulcerative endocarditis differ much in their objective symptoms, but they may be referred to two types: typhoid; pyæmic. In both, the cardiac symptoms are quite masked by the preponderating importance of the systemic state, and hence cases of primary endocarditis are apt to be overlooked. When there is an attack of rheumatism going on, suspicion of cardiac mischief will of course be excited by the sudden occurrence of a violent chill which inaugurates both forms. In the *typhoid form* succeeding the chill there is considerable fever, the range of temperature being rather of the remittent type; headache, vertigo, and extreme prostration, and sometimes a sense of præcordial oppression, are then experienced; the tongue is dry and brownish; there are nausea and vomiting, and the bowels are constipated, or diarrhœa is present. The prostration gains rapidly, and by the fourth day a condition of depression is reached comparable to the second week of typhoid. The resemblance to typhoid is all the greater, since the abdomen is swollen and tympanitic and the spleen is enlarged. Delirium (irritation) soon comes on, to be replaced in a few days by stupor and coma (depression). A severe diarrhœa now succeeds to constipation, if that condition has existed before, and the perplexity of the case may be enhanced by rose-spots and petechiæ appearing on the abdomen. Presently, the patient lying in a comatose state, the stools and urine are passed involuntarily. The urine has a smoky appearance, and contains more or less blood, and albumen is present. There is usually some bronchial catarrh, with cough and dyspnœa—the latter, however, may be due to blocking of vessels and infarctions. On auscultation, a rather loud, systolic murmer is audible, usually with greatest intensity in the mitral area, or with the second sound in the aortic area. The *pyæmic* form begins with a chill, which is a decided rigor, followed by a high fever and sweating. The chills recur sometimes with the regularity of an intermittent fever, but usually very irregularly, as is proper to pyæmia. A condition of profound and increasing adynamia is soon developed. There is often a yellowish hue of the skin; there may be jaundice, or there may occur petechial or hæmorrhagic spots, or a roseola may make its appearance. During the maxima of the temperature curves the heat may attain to 105° Fahr. and the pulse to 140. Dyspnœa and accelerated breathing may indicate pulmonary infarctions and pneumonia; enlargement of the

* Rudolf Maier, Virchow's "Archiv," Band lxii, "Ein Fall von primärer Endocarditis diphtheritica."