blood, the left ventricle may undergo hypertrophy, is that the contractile energy expended is necessarily increased, because of the obstacles in the circuit. The pulse is small, its tension low, and its rhythm

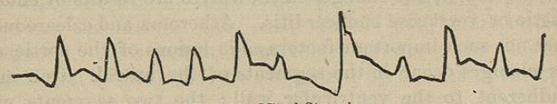

Fig. 25.—Mitral Stenosis.

irregular,* but the irregularity is not constantly present, and is a sign rather of rupture of the compensation. There are much cough, difficulty of breathing, bronchorrhœa, often bloody sputa, sometimes hæmorrhage, red-brown induration and hæmorrhagic infarctions; dilatation of the right cavities; general venous stasis, cyanosis; enlargement of the liver, ascites; albuminous urine, and general dropsy. By enlargement and hypertrophy of the left auricle, by dilatation and hypertrophy of the right ventricle, and by the distention of the veins, the stenosis is for a brief period compensated. But the conditions present bring about a slow rupture of the compensation, without the introduction of new disturbances. The changes in the muscular tissue of the right heart, the degeneration of the walls of the dilated vessels, and the alterations produced by the congestion of the liver, intestinal canal and kidneys, suffice to bring on the group of disorders above mentioned, which belong to the mitral lesions. The rupture of the compensation is much facilitated by overwork of the heart, by pulmonary diseases, or by intercurrent febrile maladies. On inspection, rather wide diffusion of the apical impulse is perceived, if there be an apical impulse strong enough for recognition. It is rather a widespread undulation than an impulse at a special point. It extends from within the mammillary line to the right border of the sternum and downward to the epigastrium. It may be absent. On palpation the apical impulse is found to be weak and unresisting, and a purring tremor is felt which may be diastolic or presystolic. If there be regurgitation, a purring tremor may also be felt synchronous with the systole. Both absolute and relative dullness are increased.

The transverse dullness is more increased than the vertical, and extends to the right border of the sternum, even beyond, and over the xiphoid appendix. A murmur is audible in the mitral area, of a rather harsh, grating, or blowing character, and occurring with the diastole and extending on up to the systole. The murmur may be presystolic —that is, occurring just before and extending in to the systole, but there are differences of opinion in respect to the time of this murmur. The murmur is usually heard with greater distinctness when the patient

* Balfour, "Diseases of the Heart," "Extreme Irregularity," p. 126.



sits upright leaning forward, or to the left. No murmur may be audible in some cases under any circumstances. Then the rational signs of mitral lesions possess a high degree of significance, and deserve attentive study, and a failure to appreciate their value and overweening attention to the physical signs are fruitful sources of error, under these circumstances. While, when present, the murmurs are heard in the mitral area with the greatest distinctness, they are propagated toward the apex, and lost toward the base. In a few cases of stenosis, another sign is to be heard over the apex, and at the pulmonary area, namely, reduplication of the second sound. Various explanations of this phenomenon have been offered, but the most probable is that the aortic and pulmonary valves do not close in the same instant of time, owing to the difference in tension of the aorta and pulmonary artery, the tension of the latter being relatively greater and therefore closing before the former. There is a sharp accentuation of the second sound in the pulmonary area, when the reduplication does not occur, owing to the high tension under which the valves are filled and closed. This characteristic of the second sound will disappear when the tension of the vessels declines from any cause or when the tricuspid becomes incompetent.

**Symptoms of Regurgitation or Insufficiency, Rational and Physical.**—So long as the compensation continues, the patient may be comparatively free from discomfort, but the existence of these circulatory derangements leads to pathological changes which effect a rupture of the compensation—e. g., the pulmonary disorders, which are thus brought about, the myocarditis which attacks the walls of the right ventricle, or an intercurrent disease of some kind. Præcordial uneasiness, palpitation, cough, and dyspnœa are the first symptoms experienced when the compensation is ruptured. The pulse becomes soft, small, rapid, and irregular, and while the sphygmographic trace exhibits these features there is nothing distinctive in its form. The

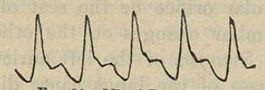

Fig. 26.—Mitral Insufficiency.

legs, presently, become œdematous, the cavity of the abdomen fills, the liver is disordered, the urine is loaded with albumen, and the patient ultimately dies drowned in his own fluids. The physical signs are characteristic. As in insufficiency of the mitral, there is more or less, usually considerable hypertrophy of the left ventricle, enlargement of the cavity and thickening of the walls of the left auricle, hypertrophy and dilatation of the right ventricle; the total result is that the heart is much enlarged, and lies lower and deeper than is the normal condition. The area of dullness, absolute and relative, vertical

and transverse, is enlarged, and the cardiac impulse diffused. On auscultation a systolic blowing murmur is audible in the mitral area, is propagated toward the apex, and may be most intense at the very extremity of the apex. This systolic *bruit* may also, when loud and strong, be heard over the whole cardiac area, and posteriorly under the angle of the scapula ; it may take the place of the first sound, or be heard with it. Usually the murmur can be separated from the proper systolic sound, by very carefully raising the head from the stethoscope so that the ear but touches it. Sometimes the *bruit* is heard with the greatest intensity in the second intercostal space, external to the left border of the sternum, in the position of the appendix of the left auricle, and because of the regurgitating blood like "the fluid in veins producing sonorous vibrations louder at the point of impingement than at that of origin" (Balfour). This, the explanation of Naunyn, is now generally admitted. If there be obstruction as well as regurgitation at the mitral orifice, there will be, as already set forth, a presystolic murmur, extending up to the systole, or under some circumstances a diastolic murmur. In regurgitation, as in stenosis, there is marked accentuation of the pulmonary second sound, until, at least, dilatation of the cavity and incompetence of the tricuspid introduce new conditions.

The diagnosis of mitral disease must rest on a careful survey of the rational and physical signs. Too strict attention to the physical and neglect of the rational signs are frequent sources of error. Exact localization of the murmurs to the areas to which they belong is most important. The history of the case necessarily enters into the question of its nature. When the indications afforded by the history of the case and the rational and physical signs coincide, any serious error is hardly possible.

**AFFECTIONS OF THE TRICUSPID VALVE AND ORIFICE.**— Only once or twice, in one hundred cases of endocarditis, will the right auriculo-ventricular orifice be the seat of mischief, and then in association with similar changes on the other side of the heart, at the mitral orifice. Stenosis of the left auriculo-ventricular orifice and obstructive diseases of the lungs cause distention of the right ventricle and produce that kind of insufficiency which is known as relative insufficiency. Regurgitation takes place through this orifice, because, being enlarged, the valves become unable to close it during the systole. Over-distention of the auricle and hypertrophy result from the regurgitation, and the tension rises in the venæ cavæ and venous system, while there are ischæmia and diminished tension in the aortic system. The right ventricle also undergoes hypertrophy, because it is filled under the increased pressure of the high tension in the veins and the hypertrophy of the auricle. Regurgitation is often due to changes in structure that are congenital, and stenosis



almost always. Very rarely is stenosis produced by acute endocarditis, and, when it does occur from this cause, the anatomical changes are precisely those which have been described as taking place on the other side. The results of stenosis are the same as those of regurgitation, and need not, therefore, be repeated ; but stenosis never exists alone, and is always associated with changes on the left side. The pulse is small, weak, but not otherwise altered. A very characteristic symptom is the occurrence of a pulsation in the jugular, synchronous with the cardiac movement. It ought not to be forgotten that waves are caused in the jugular by the respiratory movement—by the expiratory pressure. The true venous pulse does not extend beyond the bulb of the jugular, if the valves of the vein are intact, but by distention they become so, when the venous pulse is perceived along the whole extent of the vessel, extending even to the external jugular. It is synchronous with the contractions of the heart. The pulsation may be double, produced by the contraction of the auricle, and by the beating of the aorta, the vena cava superior lying in close proximity to that vessel. There is a feeble venous pulsation when there is regurgitation at the mitral orifice, a stronger one with coincident insufficiency of the tricuspid, and with the latter alone. That this pulsation is produced by the lesions above mentioned, and is not an oscillation in the blood-current caused in the various ways already described, is determined by merely compressing the vessel with the finger, when the following facts will be elicited : If the pulsation be due to the heart-movements (regurgitation), when the vein is compressed at its middle, it will continue below the point of compression and cease above ; if due to the beating of the carotid, it will continue above the point of compression, and cease below. If due to the respiratory movements, the pulsation will be synchronous with those movements ; if to the heart-movements, sychronous with them ; if respiratory, they will cease with the suspension of breathing; and, if cardiac, will continue. There is an equally characteristic venous pulse of the liver, which is felt immediately on the occurrence of the changes on the right side of the heart, because the hepatic veins are not provided with valves. The pulsation, synchronous with the cardiac movements, may be felt over the whole organ, or be confined to the right lobe. The venous pulsation in the neck may appear and disappear under the variations in the fullness of the right cavities and the force of the ventricular contractions. The hepatic pulsation is affected by effusions in the abdomen, as well as by the state of distention of the vena cava and the hypertrophy of the right ventricle. So long as the valves of the jugular remain intact, the increased tension under which their closure is effected causes a murmur, humming and clacking combined, which is audible in the bulb. The hypertrophy existing chiefly to the right, the area of impulse must be seen to the right, and is rather diffused.

Dullness on percussion, due to the enlarged right auricle, can be developed to the right of the sternum from the second to the fourth rib, and the dullness due to the right ventricle, to the base of the sternum, to the xiphoid appendix, and to the central and right portion of the epigastric region. A pulsation produced by the right auricle can be seen and felt sometimes in the right, second intercostal space. On auscultation in the tricuspid area—the lower segment of the sternum—we hear a blowing murmur, systolic in time, and most intense at the junction of the intercostal space between the fourth and fifth rib and the sternum; sometimes, most intense over the xiphoid appendix. This is the characteristic murmur, but there are associated with it the valvular mitral murmurs which almost always are present, and are audible with the greatest intensity at the mitral area and toward the apex. These are both systolic, presystolic, and diastolic, as has been pointed out. In the affections of the right auriculo-ventricular orifice, the pulmonary second sound is weak, because of the diminished tension in the pulmonary artery, unless there is coincident obstruction or regurgitation at the mitral orifice, which causes an accentuation of the pulmonary second sound. The mechanical effect of the lesions on the right side is immediate, and compensation is possible to a very limited extent. Extreme venous stasis soon occurs, with the attendant symptoms of hepatic disturbance, ascites, albuminuria, general dropsy. The prognosis is therefore unfavorable. The diagnosis is difficult because of the coexistent mitral lesions, but the lesions of the right auriculo-ventricular orifice are established by the determination of these physical signs: a well-marked, true venous pulsation of the neck; a systolic murmur, audible with the greatest intensity at the junction of the intercostal space between the fourth and fifth rib with the right border of the sternum, and a weak, pulmonary second sound.

**AFFECTIONS OF THE PULMONARY VALVES AND ORIFICE.**—These may be congenital or acquired. When acquired they are produced by endocarditis, or are due to calcareous deposition and atheromatous degeneration, but acquired changes are extremely rare. The results of stenosis and insufficiency are the same, and consist of dilatation of the cavity and hypertrophy, leading to insufficiency of the tricuspid. In *insufficiency* of the pulmonary valves the resulting conditions are the same as in the corresponding change at the aortic orifice. The pulmonary artery and its divisions undergo dilatation, the intima becomes the seat of the nutritive changes already described, and lobular pneumonia and hæmorrhagic infarctions occur in the lungs. The rational signs are dyspnœa, deficient aëration of the blood and cyanosis, distention of the superficial vessels, dropsy, palpitation of the heart, præcordial oppression, sudden attacks of suffocative feeling, with præcordial pain and intense anxiety, etc. The physical signs are those of enlargement of the right cavities, a loud diastolic murmur heard with great intensity at the left border of the sternum and the upper margin of the third rib, and propagated toward the middle of the sternum, opposite the fourth rib and downward, and is lost going toward and over the great vessels at the base. There may be also a systolic murmur. These symptoms only occur when the compensation is ruptured, for the hypertrophy of the ventricle walls and the dilatation of the cavity compensate very fully for the mischief done.



*Stenosis* is a more important condition than insufficiency, but it is congenital stenosis with which we have to deal chiefly, the acquired condition being exceedingly rare. In congenital stenosis the changes consist in constriction of the pulmonary artery, unclosed foramen ovale, unclosed ductus Botalli, stricture at the ductus Botalli, with hypertrophy of the right cavities. The importance of these congenital defects, besides the damage to the heart, consists in the frequent association of these anatomical anomalies with tuberculosis of the lungs. The right ventricle enlarges to a remarkable extent, the walls attaining in thickness to the dimensions almost of the left. The result is, there are present the physical signs of hypertrophy of the right ventricle—an increased area of cardiac dullness to the right; a blowing, systolic murmur, audible in the pulmonary area, and propagated not toward the base and great vessels, but somewhat to the left and a little downward, the point of greatest intensity being the junction of the third rib, upper border, with the left border of the sternum; weak or inaudible second sound. The rational symptoms correspond to the anatomical conditions. The compensation effected by dilatation and hypertrophy of the right ventricle suffices to maintain a condition of comparative comfort, but unusual physical exercise, obstructive pulmonary diseases, and other causes bring about a rupture of the compensation, when there ensue difficulty of breathing, cough, cyanosis that may be very intense, but general dropsy and albuminuria occur only when the right ventricular wall weakens by myocarditis.

The duration of these cases of congenital defects in the structure of the heart varies with the degree of deformity and the circumstances in life. The compensation may be so perfect that the heart is equal to the needs of a quiet existence, and comparative comfort may be enjoyed by youths who possess even a considerable degree of cyanosis. But the degree of cyanosis is usually a measure of the success of the efforts at compensation. The subjects of congenital pulmonary stenosis are otherwise imperfect in organization—they are comparatively weak, develop slowly, have soft, flabby muscles, bones do not unite, and the nutrition continues poor. Beside the cyanosis, which is usually most strongly marked in the extremities, they have cold hands and feet, and possess but little endurance of cold, are subject to asthmatic attacks, to

giddiness and vertigo, to epileptoid attacks, etc. The duration of life in these congenital cases varies from a few months to twenty or thirty years.

**Treatment.**—We quite agree in the statement of the late Dr. Flint,* that caution is necessary in the expression of opinion to the subjects of cardiac mischief; the importance of recognizing the fact that some murmurs have no pathological nor clinical significance; the good results obtained from the treatment of associated morbid states in cases of undoubted valvular disease; and, finally, the striking relief derived from the timely use of "digitalis and active hydragogue purgation repeated from time to time."

When the mischief done to the heart is recent, and the newly formed connective tissue is contracting, it is highly important to give the heart "physiological rest," to enable the damage to the valves to be repaired as completely as can be effected. The rest is best secured by maintaining the recumbent posture much of the time during the period of convalescence, by the careful administration of veratrum viride, or chloride of barium, to keep the revolutions of the heart at about fifty to sixty per minute, and by iron and suitable diet to improve the quality of the blood, or by the use of nitro-glycerin to lessen the work of the heart in overcoming the peripheral resistance, and to improve the nutrition of the organ when the general vascular tension is low. When compensation is effected and the heart is equal to the task of overcoming the obstacles, no medicinal treatment is necessary. Every effort must be directed to the maintenance of the compensation, by quietude of mind and body, and by avoidance of all causes of diseases. Nevertheless, daily open-air exercise and exposure to sunshine are necessary to maintain health at the proper standard; for, if the blood is impoverished by an in-door life and the want of appetite and imperfect sleep, which are necessary results, the rupture of the compensation must then take place. Formerly severe exercises were strongly condemned as hurtful to a weak heart, but now, by some medical extremists, are as strongly approved as means to promote the nutrition and to increase the muscular power of the organ. In the natural course of events in valvular affections, the nutritive alterations which occur in the tunics of the vessels and in the heart-muscles ultimately effect a rupture of the compensation. Anæmia not only hastens the pathological processes taking place in the vessels and in the heart, but it actually inaugurates similar changes. It is, therefore, a measure of the highest importance to keep the appetite, digestion, and blood-making process in the most efficient state. When the heart is behaving badly in consequence of the anæmic condition, the organ is relieved by attention to the nutrition. Unless, therefore, under such circumstances there is plain need of digitalis and the other "heart-

* The "Medical News and Abstract," January 7, 1880.



tonics," so called, they should be avoided, for they disturb the stomach and interfere with digestion. When, in women especially, the compensation is not ruptured, but great distress is experienced from anæmia or the chlorotic state, the indications clearly are not to treat the heart, but those nutritive disturbances on which the functional troubles depend. When such subjects are not relieved by stomachic tonics, iron, and a generous diet, the system of rest, forced feeding, massage, and muscular faradization proposed by Weir Mitchell may be resorted to with advantage. Besides the measures necessary to prevent or overcome anæmia, the dietetic management requires the patient with compensated valve-mischief to avoid such cardiac stimulants as tea, coffee, tobacco, and alcohol in any form, except a little wine allowed at dinner provided it improves digestion. The choice of a suitable chalybeate can be made from a long list of preparations. It is a rule that combinations of iron with a mineral acid are more effective and often better borne than the milder and supposed more easily assimilated citrates, tartrates, and carbonates. German therapeutists much prescribe the ethereal acetated tincture of their pharmacopœia. The tinctura ferri chloridi is, probably, the most generally useful and efficient of the ordinary preparations. It should be given always well diluted with water after meals, and should be taken through a glass tube or a straw. An excellent stomachic tonic is tincture of nux vomica—ten drops to twenty—*ter in die* and before meals, or the milder tinctures of colomba or gentian may be preferred. A combination of great value in these cases is the elixir of the phosphates of iron, quinine, and strychnine (Aitken). The nutrition in cases of compensated valvular lesions often fails slowly, from the gradual congestion of the liver and of the intestinal mucous membrane. The digestion is slow and insufficient, the appetite fails, and the absorption of aliment is seriously interfered with by the hyperæmia and distention of the vessels. Timely recognition of this state and the use of appropriate means will prevent serious trouble. Excellent remedies are arsenic, euonymin, and salol, and under some circumstances, calomel; they are stomachic tonics, and, in sufficient quantity, powerfully stimulate the hepatic functions and deplete the portal system. The treatment should be commenced by free action of the intestines procured by the compound jalap powder, elaterium, podophyllin resin, and similar agents. Then stomachic tonics, chalybeates, and digestives, as pepsin and lactopeptine, are indicated. The kidneys should be kept active, and this is best accomplished by the simultaneous but not conjoint use of a chalybeate and a diuretic, as tincture of iron and solution of bitartrate of potassa—the iron to be taken after meals, and the potassa solution to be drunk freely between meals. An excellent method of managing these cases, when a rupture of the compensation is threatened, is to give two or three times a week some efficient doses of iridin or euonymin, and to prescribe iron,