PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Gallop Rhythm of the Heart.—FRIEDREICH MÜLLER (*Münch. med. Woch.*, 1906, liii, 785). Gallop rhythm of the heart consists in the interposition of a third sound in the cardiac cycle. This sound occurs in diastole and is associated with a definite shock which is visible and easily palpable and makes a marked impression on the cardiographic record. In the cardiogram this impression may appear immediately before the systolic rise—the presystolic type, or in the first half of diastole, shortly after the second sound—the proto-diastolic type. Both of these waves are visible in the record of a normal apex-beat, although they are then very small elevations. They are pictured and studied by Marey in his classical work on the circulation and are particularly well illustrated in the tracings of Edgrem. It has been noted, too, that in cases of acute pericarditis in which there is certainly no change in the heart muscle, that the friction rub has a triple rhythm entirely analogous to the gallop rhythm. The occurrence of the first or proto-diastolic wave corresponds with the period of greatest relaxation of the ventricular muscle and is synchronous with the negative wave or drop in the jugular pulse tracing; it marks the time when the blood begins to flow from the auricle into the ventricle. The presystolic elevation comes immediately before the ventricular contraction, is synchronous with an elevation in the jugular tracing, and is due according to Marey, not to the auricular contraction, but to the increased velocity of the blood flow from this contraction.

The numerous curves which Müller presents show the justice of Potain's division of gallop rhythm into the presystolic and the proto-

diastolic type. In some curves the third sound occurs just before the systole and is marked on the cardiogram by an accentuation of the normal pre-systolic elevation, and occurs synchronously with a rise in the jugular pulse. The occurrence of this presystolic sound might he due to an unusually large amount of hlood being thrown into the ventricle or to a loss of tone and elasticity in the ventricular wall. Potain assumes that a relaxed or sclerosed ventricular wall lends itself more easily to dilatation and oscillation (erschütterung) than a normal one, and he gives his support to the second view. Although such a notion is purely hypothetical it draws some support from the observation that dilatation is nearly always present when there is gallop rhythm. Chauveau, who favors the other view, insists that since gallop rhythm is so frequently a transient feature its occurrence cannot depend upon a permanent change in the ventricular wall. That the presystolic sound, on the other hand, is due to an exaggerated activity of the auricle, is suggested hy the frequent finding postmortem of dilatation of the auricle and a considerable hypertrophy of its wall. Such a finding, however, is not constant. Frank has shown that the pressure increase caused by the auricular contraction when the auricle is better filled and more distended is plainly shown in the ventricular curve, and Marey, too, points out that the presystolic wave in the cardiogram is more marked when the auricle discharges its blood into a ventricle already filled, or in other words, a ventricle incompletely emptied. The evidence indicates that the presystolic gallop rhythm depends upon an increased discharge of blood from the auricle into the ventricle. It is interesting to note that, as a rule, in presystolic gallop rhythm the presystolic wave is further away from the ventricular systole than normal—that is, the time between the auricular and the ventricular contraction is increased. This might be explained upon the characteristics of the heart muscle as brought out by Englemann, namely, the retarded conductivity in dying muscle or in damage to the auriculoventricular bundle. Or one might think that when the ventricle empties itself only incompletely a greater tension is required in the auricle to overcome the resistance of an already filled ventricle and that the auriclar contraction is thus prolonged, just as the ventricular systole is prolonged in the face of increased arterial tension.

Müller also shows curves of typical proto-diastolic gallop rhythm in which the elevation occurs early in diastole and is synchronous with a venous negative pulse. In many of these tracings the presystolic elevation is likewise well-marked, and the close relation between the two types is further indicated hy the ease with which one passes into the other. It is frequently noted that as conditions grow worse an existing presystolic gallop rhythm will change to a proto-diastolic. Barié insists that the latter is the more serious form and that it is always associated with dilatation of the ventricle. The time in the cardiac cycle at which the proto-diastolic sound occurs, the fact that the two waves appear so frequently in the same curve, and that the two sounds so repeatedly change from one to the other, lead one to assume a similar cause for hoth, namely, an increased flow of blood from the overdistended auricle into the ventricle. As a general rule, the presystolic type occurs with a slow, forcible heart action, the proto-diastolic when the heart action is more seriously disturbed, as evidenced by rapidity and irregularity and signs of circulatory stasis.

Another feature in Müller's curves is the relative length of systole as compared with diastole. This lengthening of systole is apparently due to a delayed relaxation of the ventricular wall which requires a more rapid outflow of the auricular blood when relaxation is complete. As Barie has observed, gallop rhythm occurs principally in conditions in which the blood pressure is high, but the fact that it occurs, too, in infectious diseases in which the pressure is low and in which there may be evidence of disease of the heart muscle, and that in cases of nephritis and arteriosclerosis and myocarditis it is not infrequently present at a time when the pressure is low, indicates that high tension is not the all-important factor that Barie considers it. The condition depends more probably upon the disturbance of the adjustment between the heart's strength and the resistance to be overcome. Gallop rhythm is found almost always associated with evident signs of cardiac insufficiency, no matter whether this insufficiency be due to an increase in peripheral resistance or decrease of cardiac power. As the circulatory conditions become readjusted the gallop rhythm tends to disappear. That the ventricle is in some instances struggling against a resistance which it can overcome only with great difficulty is indicated in some curves by a definite prolongation of the time of closure or the time between the beginning of the ventricular contraction and the opening of the semilunar valves.

Besides the increased resistance and the lessened cardiac power the rapidity of the heart-rate is also of importance in causing gallop rhythm. Schiff was able to produce gallop rhythm in dogs by cutting the vagus. In Basedow's disease and in tumultuous heart action from any cause the same tendency to gallop rhythm is noticed. In these cases it is due probably to the very short duration of diastole. The systoles crowding in one upon another, the auriculoventricular bundle has not time to recover its conductivity, and the distance between the auricular and the ventricular beats becomes so great that it may almost seem that the auricle is beating after the ventricle.

The prognosis of gallop rhythm is not always bad. Many patients live comfortably for years after its onset. Its occurrence, however, should always demand our earnest attention, and it is frequently an indication for active therapeutic measures, particularly for the administration of digitalis.

Gout and its Causation.—HALL (*The Practitioner*, 1906, lxxvi, 361) discusses the causation and treatment of gout and reviews the recent literature on the subject. His own work on the metabolism of gout is so well known and trustworthy that the following paragraph may be appropriately quoted:

"Uric acid cannot be regarded as a distinct factor in the causation of disease. We have still much to learn concerning its circulation in the tissues, and its capabilities for combinations therein. We need also further information as to its manner of excretion from the human body, and its varying behavior in individuals presenting different metabolic powers. Under properly arranged conditions, the rate and extent of its excretion may be utilized for the interpretation of the personal tolerance for exogenous nucleins, and for the elucidation of some of the intermediary stages in metabolism; but as an etiological entity, uric acid must be definitely discarded. It is high time that every practitioner made a point of fully educating the public in this subject."