of administration, and partly by the love of the novel, clinicians have from time to time experimented on their patients with various of the glucosides which have been found in the digitalis leaves. At present digitoxin holds the foreground among these substances; but this much is clear, that if there be any single principle which represents completely the therapeutic virtues of digitalis that principle is *not* digitoxin. In the first place Fraenkel has shown that of all the digitalis glucosides digitoxin is the most dangerous and the most cumulative in its action; secondly, the work of Gottlieb and Magnus would indicate that digitoxin is decidedly more active in its vasomotor effects than either digitalis itself or strophanthus; and, thirdly, Kakowski experimenting with digitalin, digitalein, and digitoxin, was able with none of them to produce the increase in the contracting power of the heart that is brought about by digitalis.

ATAXIA OF THE HEART MUSCLE.

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SINCE the simultaneous graphic registration of venous and arterial pulses has been generally adopted, following the pioneer work of Mackenzie,¹ we have learned that dissociations in the activity of the different heart chambers occur very much more frequently than we formerly supposed. The dissociation of auricular and ventricular contractions has been generally accepted since the publications of His, Mackenzie, and many other writers. The anatomical cause has been shown to consist in a lesion of the bundle of His, reproduced experimentally by Hering² and Erlanger³ and shown pathologically by Stengel,⁴ and Ophuls and myself.⁵

Only lately the discussion of the possibility of bemisystole, an isolated contraction of one side of the heart, while the other remained in diastole, has been taken up.

Twenty years ago von Leyden⁶ reported several cases in which he tried to explain by a bemisystole the phenomena observed in cases of mitral disease. In those cases two apex beats and two pulsations of the jugular vein corresponded to one arterial pulse; the first apex beat with a corresponding arterial and venous wave was supposed to correspond to a complete contraction of both heart chambers; while the second apex beat with a venous wave, but without an

¹ Study of the Pulse.

² Jour. of Exper. Med., 1906.

³ Deut. Arch. f. klin. Med., vol. lxxxvii.

⁴ Pflüger's Archiv., 1905, cviii.

⁵ AMER. JOUR. MED. SCI., 1905.

⁶ Ztschr. f. klin. Med., vol. lxi.

of administration, and partly by the love of the novel, clinicians have from time to time experimented on their patients with various of the glucosides which have been found in the digitalis leaves. At present digitoxin holds the foreground among these substances; but this much is clear, that if there be any single principle which represents completely the therapeutic virtues of digitalis that principle is *not* digitoxin. In the first place Fraenkel has shown that of all the digitalis glucosides digitoxin is the most dangerous and the most cumulative in its action; secondly, the work of Gottlieb and Magnus would indicate that digitoxin is decidedly more active in its vasomotor effects than either digitalis itself or strophanthus; and, thirdly, Kakowski experimenting with digitalin, digitalein, and digitoxin, was able with none of them to produce the increase in the contracting power of the heart that is brought about by digitalis.

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Only lately the discussion of the possibility of hemisystole, an isolated contraction of one side of the heart, while the other remained in diastole, has been taken up.

Twenty years ago von Leyden⁶ reported several cases in which he tried to explain by a hemisystole the phenomena observed in cases of mitral disease. In those cases two apex beats and two pulsations of the jugular vein corresponded to one arterial pulse; the first apex beat with a corresponding arterial and venous wave was supposed to correspond to a complete contraction of both heart chambers; while the second apex beat with a venous wave, but without an

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arterial pulse, was thought to correspond to an isolated contraction of the right ventricle.

This view advocated by von Leyden and defended again recently, has not been generally accepted. Riegel especially tried to show that the phenomena could be more easily explained by the theory that the second beat corresponded to a premature contraction of the whole heart, and that the absence of the arterial pulse could be explained by the fact that the left ventricle during the short diastole had not recovered sufficient strength to overcome the arterial pressure and open the aortic valve. This view has been generally adopted, and by the work of Wenckebach, Hering, Cushny, and Mackenzie has been shown to be the cause of the supposed hemisystole in the great majority of cases.

The possibility of a hemisystole has been denied on physiological and anatomical grounds, especially by Hering who admitted the occurrence of hemisystole only as a symptom of agony in a dying heart. Notwithstanding the authority of such an excellent observer as Hering, the occurrence of hemisystole has been very widely discussed in recent years.

It was again Mackenzie who first published some tracings in which there was an incongruence in the number of apex beats, indicating the action of the left ventricle, and the number of venous pulsations. He considered these tracings as very exceptional, and admitted the incongruence of the left and right heart action to be a symptom of beginning death.

The question of hemisystole was taken up again by F. Kraus and G. Nicolai,⁷ who published a preliminary communication on this question. They studied the question by the graphic method in the perfused heart and by the electrodiagram in the human heart, and reached the following conclusions: If ventricular extrasystole is produced by direct stimulation, the ventricle in which the extra stimulus is produced contracts more strongly than the ventricle to which the stimulus has to be transmitted. If one diminishes the intensity of the stimulus, the contraction of the second ventricle becomes weaker, until a point is reached at which the first ventricle contracts, while the second one does not register any change in the intraventricular pressure. Such conditions arise in the human heart, if the extrasystole proceeds from the left ventricle. Here all the variations may be observed, deviation in the strength of the contraction and deviation from the synchronism of the contraction, until complete hemisystole occurs.

A second method by which the occurrence of hemisystole could be shown consisted in the electrocardiogram taken by Einthoven's method. Krause and Nicolai assert that the electrocardiograms of the left and the right ventricle are different from each other and

⁷ *Deut. med. Woch.*, 1906, p. 1.

are characteristic of the contraction of either left or right heart. The electrocardiogram taken over the heart during a ventricular extrasystole shows by variations in its form the part which the left and the right ventricle are playing. The form of the electrocardiogram also indicates the point from which the extrasystole starts, whether from the left or from the right ventricle. In some cases the electrocardiogram assumes the form of a purely right-sided cardiogram; under those conditions a hemisystole of the right ventricle may be diagnosed.

The views expressed by Krause and Nicolai and the arguments for their validity cannot be judged at the present date, as the experimental evidence for their conclusions is not fully published, and as the value of the electrocardiogram, its interpretation, and especially the differentiation of the left and the right ventricular curves are not fully established and accepted.

Recently Wenckebach^a has reported a case in which he was able to show the existence of dissociation in the activity of the ventricles. He drew attention to the fact that in cases of perpetual arrhythmia the disappearance of murmurs in extrasystoles points to the fact that not the whole ventricle is contracting, but only certain parts of it. The same conclusion can be reached by the fact that the height of the pulse does not depend upon the length of the preceding diastole, as under normal conditions. In several tracings of the apex beat obtained in a case of perpetual arrhythmia a superposition of three systoles could be demonstrated, each following systole beginning before the preceding had ended. As during systole the heart muscle is refractory to any stimulus, the conclusion had to be reached that the second systole began in a part of the heart muscle which had not contracted during the preceding systole. The case, therefore, proves the existence of partial contractions of the ventricle. Similar dissociations in the activity of the two auricles and the venous sinus were reported in the same exceedingly interesting paper of Wenckebach.

According to modern literature, therefore, we must distinguish between two varieties of dissociation of the activity of the ventricles: (1) Hemisystole consisting of alternating contractions of the left and the right chambers of the heart; and (2) partial contractions of the heart muscle, for which I propose the name of "ataxia of the heart."

Dissociation of the activity of the ventricles seems to be very rare, as very few convincing observations of this condition have been published. I have encountered it only once amongst a very great number of tracings obtained from heart cases. As the condition may be of some interest I shall briefly describe the phenomena observed in the patient:

^a Archiv f. Anat. and Phys., Phys. Abteil.

E. B., aged twenty-seven years, Italian, entered the hospital on November 29, 1907, complaining of swelling of the legs, shortness of breath, cough, and pain over his heart. He had rheumatism about seven years ago in his right knee, incapacitating him for about three or four days, but otherwise he has never been ill. In April, 1907, he had a first attack of insufficiency of the heart with shortness of breath and cough lasting for about two months; lately these symptoms have returned, aggravated by marked sleeplessness and pain of an anginoid character. In the last few weeks he has noticed swelling of the legs.

On admission the patient presented the picture characteristic of combined failure of the left and right ventricles. His skin was extremely pale and seemingly exsanguinated, spotted at the ear, cheeks, and lips by deeply cyanotic areas; there was quite an appreciable amount of oedema extending to the knees. The respiration was quite labored, varying between 30 and 40 per minute. The pulse was very irregular in force and rhythm; comparing it with the apex beat one could easily notice that a large number of heart beats did not reach the wrist. The veins of the neck were markedly distended and pulsated. The apex beat could be felt, very heaving in character, in the seventh interspace in the anterior axillary line; the right heart border was 2 cm. to the right of the right sternal border, the upper border at the third rib. Over the whole area of the heart a systolic thrill could be felt. On auscultation such a multiplicity of murmurs were heard, that it was at first impossible to come to a clear conception of the valvular lesions. After the heart had slowed down under the influence of digitalis, a long drawn-out systolic murmur was audible at the apex, preceded by the sharply clicking sound of mitral stenosis. The second sound was very indistinct, and was followed by a long drawn-out diastolic murmur extending to the beginning of next systole. Over the tricuspid area a systolic murmur could be heard, while at the aortic area a sharp systolic murmur was present and was carried through the aorta up to the arteries of the neck. The diastolic sound over the aorta was normal, while distinctly accentuated and reduplicated over the pulmonary artery. The liver was distinctly enlarged, extending 5 cm. below the costal margin, and was easily palpable. Pulsation of the liver, however, could not be made out. The urine was very scanty and contained a small amount of albumin.

The diagnosis was made of aortic stenosis, mitral insufficiency and stenosis, and tricuspid insufficiency.

Under large doses of digitalis the heart regained sufficient strength, the oedema, the cyanosis, and the albumin in the urine disappeared. Three days after beginning the digitalis medication the pulse frequency fell below 60; digitalis was stopped, but the frequency of the pulse kept on falling until on the fifth day it fell to 28; comparison

with the apex showed that each radial pulse corresponded to a bigeminal heart beat, of which the second was abortive.

The patient made an uneventful recovery. The tricuspid murmur disappeared with the slowing of the pulse. Six weeks afterward the patient left the hospital with a heart sufficiently compensated for light work.

At the beginning of January the patient returned for treatment, with almost the same condition that prevailed when he first entered the hospital. He reacted again very nicely to digitalis medication; under small continued doses of digitalis the heart has kept in good condition ever since.

Certain auscultatory phenomena at the time of the first examination attracted our attention to the possibility of dissociation between the action of the two ventricles. During the first few days the heart action was so rapid that no definite inferences could be drawn; as the heart began to slow down, however, it appeared that a certain number of the beats not leading to a radial pulse were distinctly different from the normal systoles and produced auscultatory phenomena very different from the usual cardiac sounds. While in successful contractions the first sound started with a clicking sound, indicative of mitral stenosis, in other contractions only a very soft systolic murmur could be heard followed by a normal second sound. The absence of the clicking sound seemed to show that the mitral orifice did not close and that, therefore, the left ventricle during certain phases of the heart action did not contract. Occasionally another modification of the heart sounds was observed: It seemed as if both the first and the second sound started an echo; the first sound was followed by another sound too far separated to be taken for a simple reduplication; then a second sound could be heard followed by another second sound.

Both these phenomena seemed to point to dissociation between the action of the right and the left ventricle; tracings taken at that time corroborated this view. Under the influence of digitalis these auscultatory signs disappeared entirely; the right and the left heart seemed to work together. Later on the dissociation recurred, but without any auscultatory signs indicating a dissociation. The most painstaking auscultations of the heart did not reveal any sounds, while the jugular vein still kept on pulsating.

A great number of tracings were taken during the four months the patient was under observation. The difficulty in arriving at an understanding of the waves present in the tracings obtained called for further observations; but at no time except at the time of the acute digitalis poisoning, could anything be obtained resembling a normal phlebogram.

Figs. 1, 2 and 3 are parts of tracings selected as best illustrating the condition constantly found. Fig. 1 was taken at slow speed, Figs. 2 and 3 at high speed, as may be seen from the markings corresponding to $\frac{1}{4}$ seconds.

Fig. 1, taken at slow speed, demonstrates the impossibility of finding a definite relation between the arterial pulse and the jugular pulse. The heart is beating irregularly but slowly, as can be seen from the tracing of the apex beat; the radial pulse corresponds in

FIG. 1.—Taken at slow speed; the marks indicate the beginning of systole in the radial artery and the corresponding point in the venous pulse.

frequency to the apex beat. If two heart beats follow each other quickly, as systole 1 and 2, the radial pulse corresponding to the second beat becomes very small and is considerably retarded compared to normal beats. This is another illustration of the Extra-

FIG. 2.—The same as Fig. 1, but at high speed.

verspätung described by Hering, due to the occurrence of premature beats. The heart muscle, which has not recovered sufficient strength in the short diastole, opens the aortic valves at a later period of the systole; the extra wave travels in the arterial system not as quickly as a normal wave, for reasons sufficiently discussed by Hering.

If one tries to analyze the venous pulse occurring between one apex and radial beat and the next, one is impressed by the number of waves corresponding to one heart evolution. In systole 2 there are seven waves occurring between radial beat 2 and 3; in systole 5 there are ten waves. Similar conditions prevail in Figs. 2 and 3, taken at high speed. In systole 3 of Fig. 3 there are five waves corresponding to one radial pulse.

The interpretation of the venous pulse offers great difficulties, since the normal waves of the venous pulse cannot be identified. In Fig. 3 corresponding points are marked, the first mark of each systole corresponding to the beginning of the radial pulse; the second mark shows the beginning of diastole indicated by the dicrotic wave. While the points on the apex beat and radial tracings correspond very nicely in the different systoles, no relation can be found between

FIG. 3.—Taken at high speed; shows in systole 3, 5 venous waves corresponding to one arterial pulsation.

the waves of the venous pulse and the radial pulse. Sometimes the *c* wave corresponds to a very sharp wave, sometimes to a very slow wave, sometimes to a depression. In Fig. 3, systole 3, a diastolic wave is entirely missing, showing that the diastole of the right ventricle did not occur at the same time as the diastole of the left, thus proving the dissociation in the action of the two ventricles.

The explanation of the venous waves occurring during the diastole of the left ventricle is very difficult. Waves occurring during diastole known so far are: (1) The *v* wave occurring synchronously with the beginning of diastole. (2) The wave *a* produced by the contraction of the right auricle preceding ventricular systole. (3) Lately, A. Hirschfelder⁹ has drawn attention to a wave *h* occurring

⁹ Johns Hopkins Hosp. Bull., vol. xviii.

in diastole and caused, according to his interpretation, by the closure of the mitral valve. Gibson later described the same wave and connected it with a third heart sound which occasionally may be heard in a slowly beating heart. Einthoven has encountered the same wave in his electrodiagrams.

Four waves, therefore, may correspond to one arterial pulse: The *a* wave corresponding to auricular systole; the *c* wave due partly to the transmitted carotid wave, and partly to a systolic venous wave; the *v* wave setting in at the beginning of diastole; and the *h* wave occurring in full diastole, and the cause of which is still under discussion.

To one evolution of the left ventricle only four venous waves can possibly correspond. The recurrence of seven to ten waves during one arterial pulse can be explained in one of the following ways: (1) In case the waves are caused by contraction of the auricle, (a) by block between the auricle and the ventricle, and (b) by fibrillations of the auricles. (2) In case the waves are ventricular in origin, by dissociation between the action of different parts of the ventricle. To decide which of these explanations should be adopted, tracings of the heart action were taken from the œsophagus; at the same time

FIG. 4.—Oesophageal cardiogram.

the apex beat was registered (Fig. 4). To understand the meaning of this tracing I must discuss the meaning of the normal œsophageal cardiogram, the explanation of which we owe to Rantenberg¹⁰ and Hewlett.¹¹ Fig. 5 is taken from Rantenberg's work. The wave preceding the apex beat is evidently due to the action of the left auricle as *s*; at the beginning of the ventricular systole the œsophageal tracing also shows a raising line *vs*, followed by several secondary waves, until the beginning of diastole is indicated by a sudden sharp fall *D*. At this moment the diastole begins and the mitral valves open; the left auricle which is in contact with the registering apparatus suddenly collapses, leading necessarily to a sudden fall of the tracing.

¹⁰ Archiv. f. klin. Med., 1907, xci.

¹¹ Jour. Med. Research, 1907, xi.

These points, applied to the interpretations of the œsophageal tracing obtained in my case, show that the œsophageal registration concords absolutely with the cardiogram. The number of beats registered corresponds with the number of apex beats, and not with the venous tracings. On the other hand, no trace of auricular contraction could be obtained, proving that in this case the irregular heart action was complicated by auricular paralysis, as was to be expected in a case of perpetual irregularity.

FIG. 5.—Normal œsophageal cardiogram.

On the other hand, the absence of auricular waves proves that the right auricle was paralysed. We know from the work of Fredericq and Erlanger,¹² that the left auricle is almost invariably dependent upon the right auricle for its activity. Fibrillation of the right auricle with complete paralysis of the left auricle has never been observed, as far as I have been able to learn; the same reason also excludes the possibility of block between the auricle and the ventricle.

FIG. 6.—Showing partial contractions of the left ventricle at the points marked by X.

I am, therefore, obliged to assume that the multiplicity of the venous waves is due to dissociation in the action of the right and the left ventricle.

The mechanism of this dissociation is shown by the œsophageal tracing. In Fig. 6 two parts of the œsophageal tracings, together

with the corresponding apex beats, are reproduced. In the places marked by *X*, there are three instances in which both apex and oesophageal tracing differ quite distinctly from the other parts of the tracing. In both, following the fall which marks the beginning of diastole, there is a new ascending wave, corresponding in the apex tracing to a beat very much smaller in size than the usual apex beat. In the oesophageal tracing the wave due to this contraction seems to be slightly smaller than the usual wave: the principal difference from the ordinary beat, however, is due to the fact that the next beat begins before the fall of the curve indicating that the beginning of diastole has set in. In other words, the next contraction begins before the previous contraction has finished. This can take place in two ways: (1) In the form of partial contraction of the ventricle;

FIG. 7.—Showing at the points marked *X* partial contractions of the left ventricle interrupted before diastole by a complete systole.

that only one part of the whole ventricle responds to one stimulus, another part to a subsequent stimulus. (2) Another explanation may be found in the possibility that under certain conditions the law of the refractory period of the heart muscle does not hold good, and that under certain conditions a superimposed contraction may occur in the already contracting ventricle.

While the second view may be discussed, in view of the work of Rhode and Carlson, showing that under certain conditions the heart in systole is not absolutely refractory to new stimuli, such a supposition would not be justifiable in view of Fig. 3, which shows an absolute incongruence in the work of the right and the left ventricle. We, therefore, have to admit as an explanation the occurrence of partial contractions of the ventricles. It has to be conceded that such partial contractions do not occur only in the right ventricle; the

oesophageal tracings showed that similar partial contractions occurred also in the left ventricle. Fig. 7 shows also at the point X a partial contraction interrupted before its diastole by the following full-sized diastole.

I have already proposed the name of ataxia of the heart muscle for the phenomenon of partial ventricle contractions. As in locomotor ataxia the coördination of the different parts of the muscles of the limbs has been lost, so in ataxia of the heart the coöperation of the different parts of the heart muscle is lost. How the circulation can be kept up effectively under such conditions is a marvel to anybody who has carefully analyzed the action of such a heart. Evidently the partial contractions do not influence the blood stream which is entirely kept up by the full-sized contraction.

FIG. 8.—Showing typical digitalis action with normal phlebogram of ventricular type without any partial contractions.

The explanation of the phenomenon of ataxia is very difficult. One could at first think that the stimulus originating at the auriculo-ventricular ring is blocked in different places and reaches different places of the ventricle at different times. Such an explanation is impossible in view of the fact that the ataxia of the heart disappeared under digitalis (Fig. 8). Digitalis slows conduction and would aggravate any condition due to a lesion of conductivity. I believe the phenomenon to be due to different parts of the heart originating their own rhythm, as shown in Erlanger's experiments, in which every part of the heart muscle, even if separated from the region of the large veins or the His bundle, contracted rhythmically.

Investigation of ataxia of the heart will certainly show that partial contractions of the ventricle occur quite frequently in perpetual arrhythmias, and that a large number of symptoms observed in these cases can be explained only by partial contractions of the ventricles.