Transient auriculoventricular dissociation with varying ventricular complexes caused by digitalis / Henry A. Christian.

Transient Auriculoventricular Dissociation with Varying Ventricular Complexes Caused by Digitalis

HENRY A CHRISTIAN, M.D.
BOSTON

Reprinted from the Archives of Internal Medicine
September, 1915, Vol. xvi, pp. 341-355

CHICAGO
AMERICAN MEDICAL ASSOCIATION
FIVE HUNDRED AND THIRTY-FIVE NORTH DEARBORN STREET
1915

TRANSIENT AURICULOVENTRICULAR DISSOCIATION WITH VARYING VENTRICULAR COMPLEXES CAUSED BY DIGITALIS*

HENRY A. CHRISTIAN, M.D.
BOSTON

In 1913, Cohn[1] and Oppenheimer and Williams[2] described cases of heart-block in which the form of the ventricular complex showed frequent change.

In Cohn's case the heart-block was transient. On Aug. 22, 1910, the patient showed partial heart-block with a 3-2 rhythm as shown by polygraphic tracings. On Feb. 10, 1911, this patient was studied again and showed a partial heart-block with a 2-1 rhythm. On February 11 and 13 there was a complete auriculoventricular dissociation. At this time electrocardiographic tracings were made and it was found that successive ventricular complexes changed their outline so that no two were precisely alike. Two general types, however, could be distinguished, the one resembling the type of beat arising from stimuli originating in the wall of the right ventricle and the other those arising in the wall of the left ventricle. Gradual variations were made out between these two types. With the change in type there was also a change in interval between successive ventricular complexes. This dissociation did not persist, for shortly after this time the rhythm was restored to a normal one. Later examinations on Dec. 19, 1911, and Feb. 18, 1913, showed that the normal rhythm had been maintained except for an occasional premature ventricular contraction.

This was a patient with cardiac decompensation, the etiology of which is not clear from the history. It appears to have been a slowly developing myocardial insufficiency. It seems not unlikely that the heart-block observed at the various times was the result of digitalis therapy.

In the case of Oppenheimer and Williams the heart-block was permanent during the period of observation from Feb. 26, 1912, to the day of the patient's death, Dec. 31, 1912. Histologic examination of the heart, however, revealed no organic lesion to account for the

---

* From the Medical Clinic of the Peter Bent Brigham Hospital, Boston.

1. Cohn: Heart, 1913, v, 5.
2. Oppenheimer and Williams: Proc. Soc. Exper. Biol. and Med., 1913, x, 86.

block in the auriculonodal junction, the node of Tawara or the main stem and its branches. The nodal artery was sclerotic. The variations in the ventricular complexes were seen not only from one examination to the next but often from beat to beat. The waves Q, R, S and T all showed variations; for example in Leads 1 and 2 the R waves were sometimes upright and sometimes inverted. In Lead 3 the wave R was always inverted. The patient had marked Cheyne-Stokes respiration and the auricular rate was strikingly reduced during the dyspneic period, while the ventricular rate showed little change.

Case 1.—The patient was in the Peter Bent Brigham Hospital on two occasions; the first time (Peter Bent Brigham Hospital Medical No. 426) from Oct. 18 to Dec. 18, 1913; the second time (Peter Bent Brigham Hospital Medical No. 939) from March 11 to April 5, 1914, on which day he died.

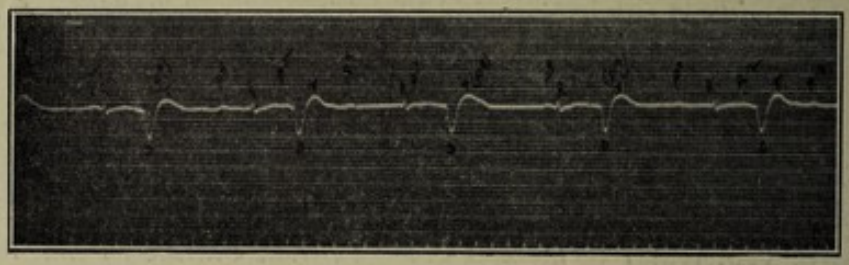


Fig. 1, Case 1.—Lead I, taken March 20, 1914.

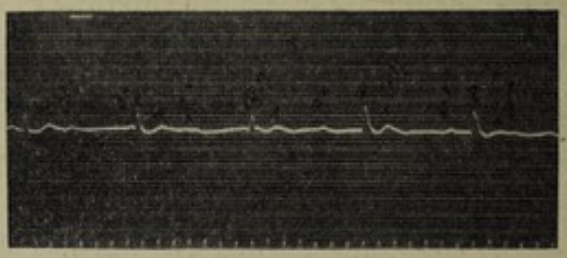


Fig. 2, Case 1.—Lead I, taken after Figure 1, March 20, 1914.

The patient, a man aged 48, had been somewhat short of breath for the past two or three years, but not enough to keep him from his work, that of a barber. He had had more or less swelling of his ankles for ten years. During the winter prior to admission he had a chronic cough. About two months before admission he noticed that he was sleeping poorly and shortly after this his breath became very short, particularly at night, so that he was obliged to sleep sitting propped upright in bed. When he first entered the hospital the right border of cardiac dulness was at the right sternal margin; the left border 9 cm. to the left of the midsternum. The apex beat was not seen or felt. Cardiac sounds were distant and a faint, blowing systolic murmur was heard, loudest at the apex. There were four or five beats of the same length followed by an extra beat, evidently an extra systole. The abdominal wall was edematous and brawny without shifting dulness. There was a brawny edema of the arms and legs.

The patient responded well to digitalis, and had a marked diuresis with disappearance of his edema. There was considerable evidence that in addition

to cardiac decompensation the patient had hypothyroidism. With thyroid extract in addition to cardiac therapy he improved markedly and left the hospital Dec. 18, 1913, in good condition, though unable to undergo much exertion.

After leaving the hospital the patient remained at home on account of weakness and shortness of breath. After a short time he became again very dyspneic and orthopneic, especially at night. He had considerable cough and raised a small amount of frothy sputum. Late in February, 1914, his feet began to swell. About the middle of April his abdomen became prominent. Symptoms of cardiac decompensation increased until he was readmitted on March 11, 1914, in an almost pulseless state.

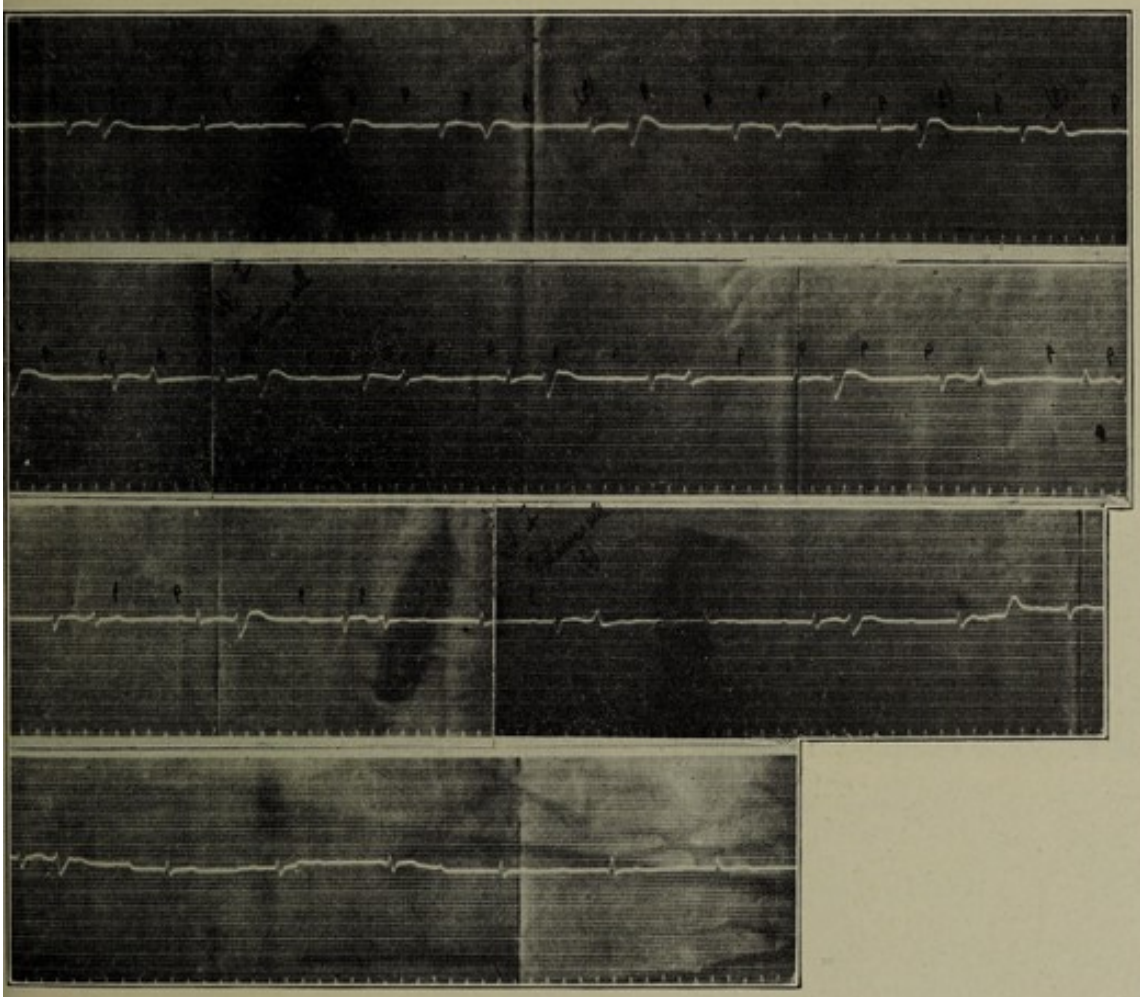


Fig. 3, Case 1.—Lead I, March 21, 1914. The strips form a continuous record.

On March 11 physical examination showed the patient lying propped up in bed, moderately dyspneic and cyanotic with moderate edema of the legs below the knees and moderate edema of the abdominal wall and of the thorax as high up as the nipples. The apex beat of the heart could not be felt. The borders of cardiac dulness were made out with great difficulty owing to hyperresonance of the chest. Heart sounds at the apex were barely audible; at the base the sounds were a little more distinct but were heard with great difficulty. No murmurs were heard. The heart action was regular. No pulse could be made out at the wrist. The lungs showed dulness at both bases with a few moist râles. The abdomen showed shifting dulness. The liver was enlarged, tender and pulsating. On March 12 the patient had gradually

improved, his respiration had become Cheyne-Stokes in type but he was less dyspneic. On March 14 the patient had improved further and there had been marked diuresis with appreciable diminution of the edema of the extremities and the ascites. The heart sounds were stronger; still there was no murmur. On March 17 a systolic murmur was heard over the whole precordium. By April 2 the patient's condition had grown less good. He seemed stuporous. There was marked Cheyne-Stokes respiration. On April 4 he had grown distinctly worse and on April 5 he continued to fail and died at 4:45 a. m. The cardiac condition had been regarded as one of chronic myocarditis in an individual with moderate hypothyroidism and moderate chronic nephritis.

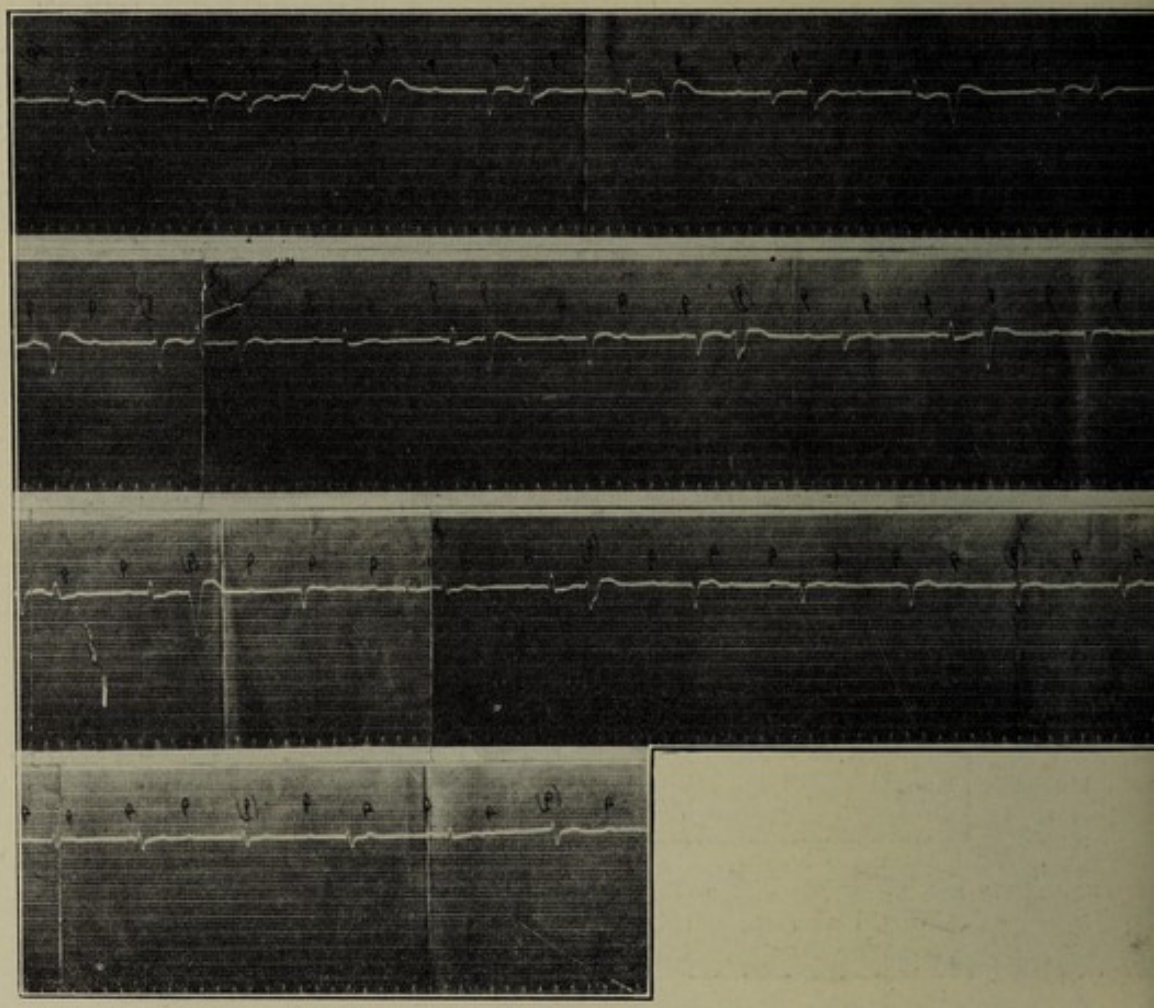


Fig. 4, Case 1.—Lead II, March 21, 1914. The strips form a continuous record.

During the second period in the hospital electrocardiographic studies were made. Some electrocardiograms taken on March 20 showed complete dissociation[3] with an auricular rate of 78, a ventricular rate of 66, with paired ventricular beats coming in quite a regular relation to each other (Fig. 1), the first one of the pair appearing to have its origin in the bundle of His, and the

3. In these cases the curves have been interpreted as showing complete dissociation. They might be interpreted instead as partial block with "P" waves at times in close association with ventricular complexes because contractions originating below the auricles have stimulated auricular contractions instead of the auricles and ventricles beating entirely independently of each other.

second one to have its origin in the ventricle. This condition, however, did not persist constantly, inasmuch as in some leads there was no pairing of beats in a regular sequence, while in others (Fig. 2), both auricular and ventricular rates were quite regular though completely dissociated and all ventricular complexes were of supraventricular origin.

On the next day, March 21, the same general appearance persisted (Figs. 3, 4 and 5) though there was greater variation in the form of the various ventricular complexes. There was a distinct tendency to an alternation of the character of the paired beats, in which the first ventricular complex of the

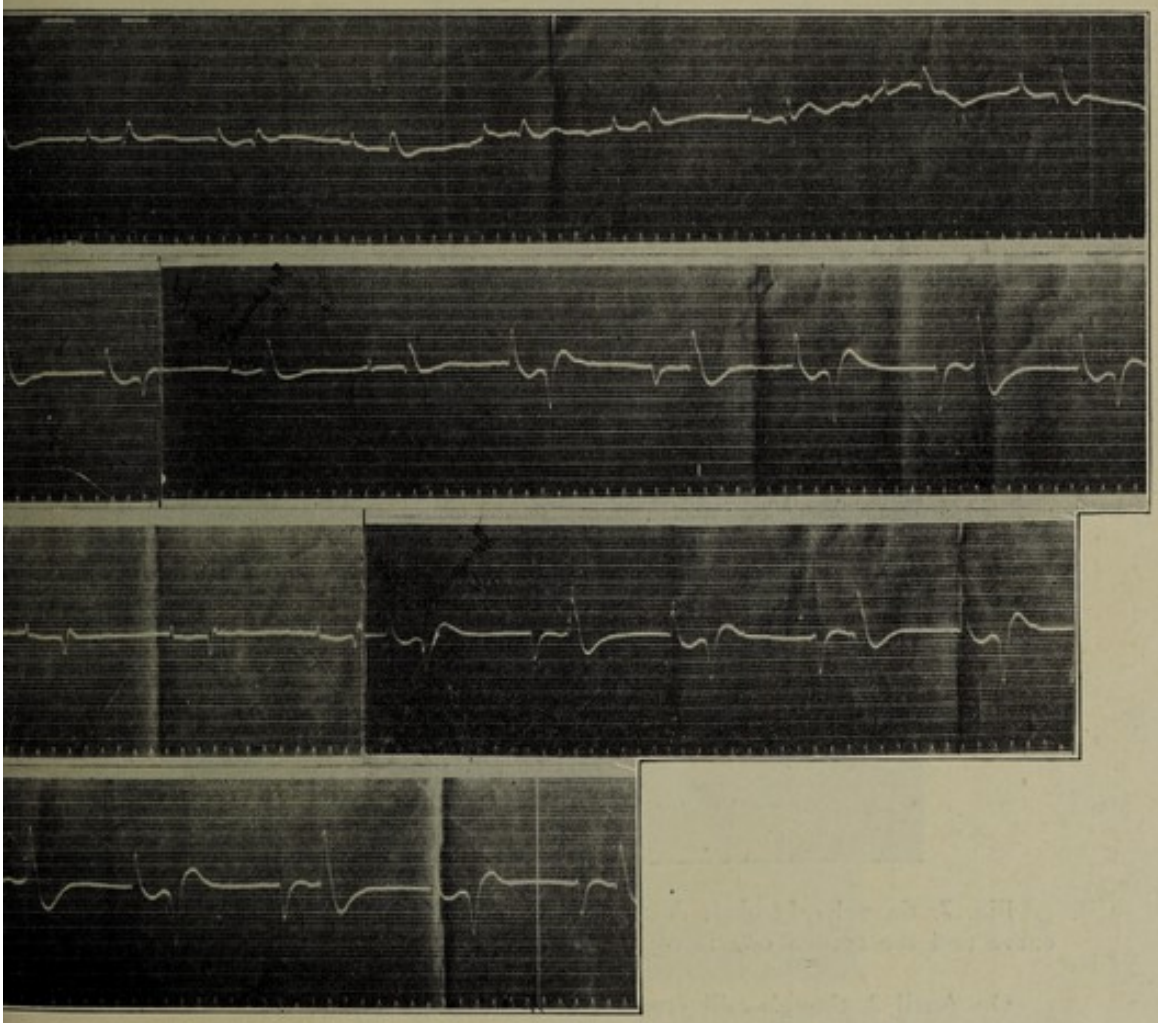


Fig. 5, Case 1.—Lead III, March 21, 1914. The strips form a continuous record except for a short break in the third strip.

pair gave an upward curve, and the second one a downward curve, and this arrangement changed in the next group so that the first complex was down and the second one up, and there was a considerable tendency to a regular alternation in this arrangement. In addition there were frequent minor changes in ventricular complexes as they succeeded each other, so that a great variety of complexes appear in any given curve.

The curve taken on March 25 showed a perfectly regular rate of 90, with all impulses transmitted from auricle to ventricle and ventricular complexes of normal form. There was a considerable delay, however, in the "P-R" interval, which reached its maximum on the next day, March 26, and then gradually

decreased, though never came quite to the normal time of transmission. The heart rate increased to 90 on March 26, to 104 on March 27, to 108 on March 28, and to 120 on March 30, and with this increase in rate of contractions the heart remained regular, the complexes normal in form, but the "T" and "P" waves gradually approached nearer and nearer until they became superimposed, which feature was very well shown by some curves taken on March 30, in which, following vagus irritation, there were some greatly increased intervals between successive "R" waves, and during these the "T" and "P" waves became separated (Fig. 6).

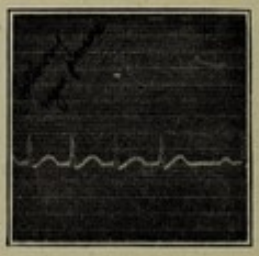


Fig. 6, Case 1.—Lead II, March 30, 1914. Curve shows a pause caused by vagus pressure.

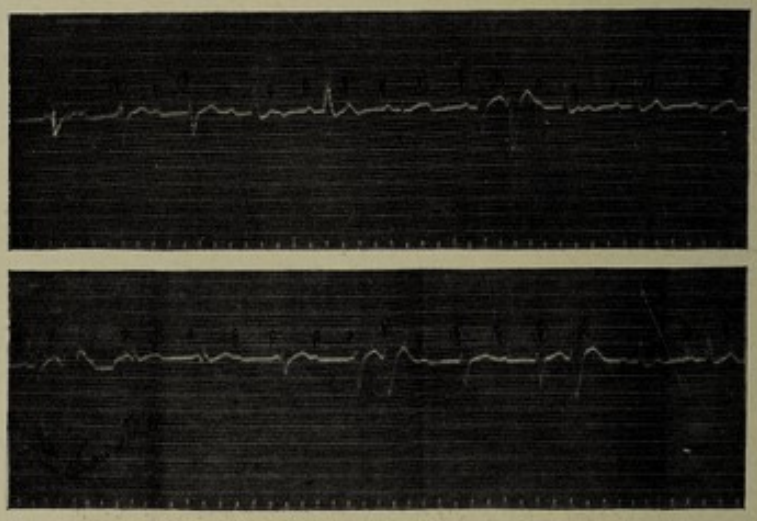


Fig. 7, Case 1.—Lead I, April 3, 1914. The two strips form a continuous curve and the second one is continuous with the curve in Figure 8.

On April 2, though still regular, the rate of the heart slowed down to 84, and the "P-R" interval increased slightly in length.

On April 3 heart-block again occurred with an auricular rate of 126 and a ventricular rate of 72. In Lead 1 (Figs. 7 and 8) this was present throughout, and at times there was such a pairing of the ventricular beats and changes in ventricular complexes as occurred when the heart was previously in block on March 20 and 21. This block, however, was not maintained, because in the interval between taking Lead 1 and Lead 2 the ventricular rate increased to 120, the ventricular complexes became normal in form, the "T" and "P" waves coincided (Fig. 9). This condition persisted while Lead 3 was being taken. After Lead 3 was taken, Lead 1 was again taken, and at this time there was an auricular rate of 120 and a ventricular rate of 60, a partial heart-block with 2-1 rhythm. There was a "P-R" interval when transmission was not blocked varying between 0.2 and 0.24 of a second.

April 4 electrocardiograms showed a rate of 120 in Lead 1 with a superimposed "P" and "T" wave, and a "P-R" interval of 0.24 to 0.28 of a second. At one point in this curve there was a blocked auriculoventricular transmission and a pause of about double the length of the preceding intervals between "R" waves. Lead 2 on April 4 showed no blocking, whereas Lead 3 (Fig. 10) showed a complete block and an auricular rate of 120 and a ventricular rate of 60. This apparently was not a partial block but a complete one, notwithstanding the fact that the auricular rate is twice that of the ventricular. This is brought out by the great variation in the time interval between the beginning of the "P" wave and the beginning of the next following "R" wave. At first glance, however, it would seem to be a partial block, with a 2 - 1 rhythm, the first beat not followed by a ventricular contraction, and the second beat fol-

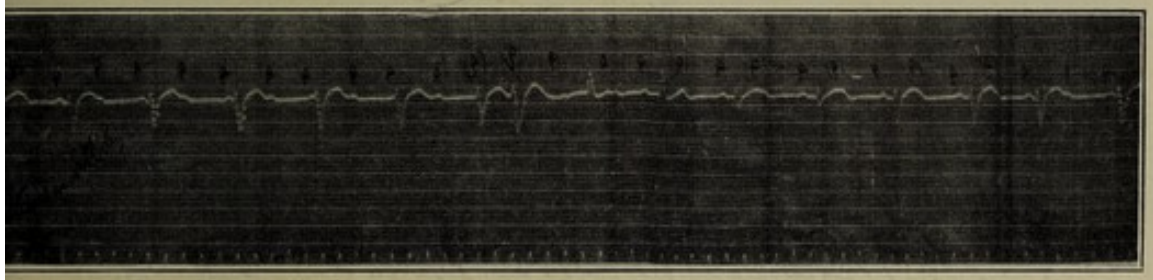


Fig. 8, Case 1.—Lead I, April 3, 1914. This strip is continuous with the second strip in Figure 7.

lowed by a ventricular contraction. Here again changes in form of ventricular complexes occur. A little later another tracing was taken which in Lead 1 shows a ventricular rate of 66 with an apparent complete block; however, the "P" waves are not distinct enough to justify any definite statement. Leads 2 and 3 have an auricular rate of 114 and a ventricular rate of 72. The complete blocking is much more evident as shown by the variations in conduction time.

The patient died during the night following these last tracings and at necropsy the heart showed the following condition:

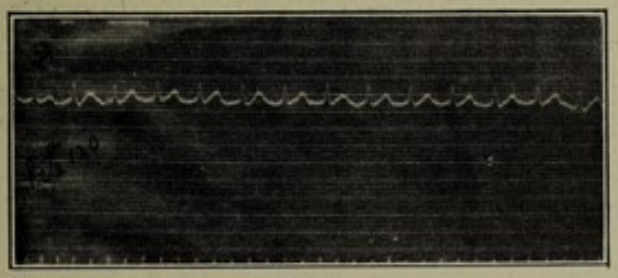


Fig. 9, Case 1.—Lead II, April 3, 1914. Curve taken almost immediately following that shown in Figure 8.

Heart: Weight, 560 gm. In situ the heart borders measured 5 cm. to the right and 11 cm. to the left (horizontal measurements) of the midline. The right auricle was slightly dilated. The appendages were free. The pericardium was everywhere glistening. The tricuspid valve measured 12.5 cm. The line of closure of valves showed no abnormalties; the valves everywhere were delicate. The chordae tendineae were not thickened. There was slight dilatation of the right ventricle and the endocardial surface was everywhere glistening, showing no abnormalities. The wall measured 6 mm. The pulmonary valve measured 8.5 cm., the semilunar cusps were normal; the line of closure of cusps showed no roughening. The pulmonary artery was incised in situ; it showed no clots. There was a small amount of clot in the chambers above

described. The left auricle was not dilated. The auricular appendage contained a small irregular mass of fibrinous material, very fragile; this was somewhat whiter than normal but apparently was clot. The mitral valve measured 11.65 cm. The valve was thickened throughout. The chordae tendineae were somewhat thickened. There was no apparent change along the line of closure though superficially it was roughened. The wall of the left ventricle measured 1.6 cm. In the region of the septum, about 4 cm. below the aortic valve, was a mass which extended down to the apex, it being 4 cm. in length, adherent to the endocardium, irregular in outline, reddish in color and covered by a definite thin whitish membrane. The heart muscle was dark red in color, was of fairly good firmness; on cross section it was homogeneous and did not show macroscopic

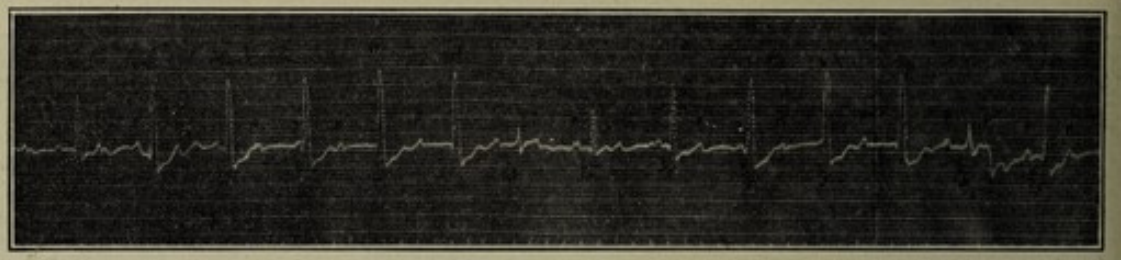


Fig. 10, Case 1.—Lead III, April 4, 1914.

increase in connective-tissue elements. There was definite thickening of the endocardium, especially of the left auricle.

Coronary arteries: The orifices were open. There was marked thickening of the walls of the vessels; this was most marked down to the level of the bifurcation of the transverse and descending branches. In portions the lumen was practically obliterated. The walls were firm but not brittle. In one portion of the left anterior coronary there was apparently total obliteration of the lumen. Sections of heart muscle taken for microscopic study showed no interstitial fibrosis. There was no evident lesion of the conduction system.

Case 2.—In this case the patient was in the Peter Bent Brigham Hospital (Medical No. 1717) from Oct. 3, 1914, to Nov. 29, 1914, on which day she died. The patient was a woman, aged 29, who about seven months before admission, while in the seventh month of pregnancy, began to be troubled with

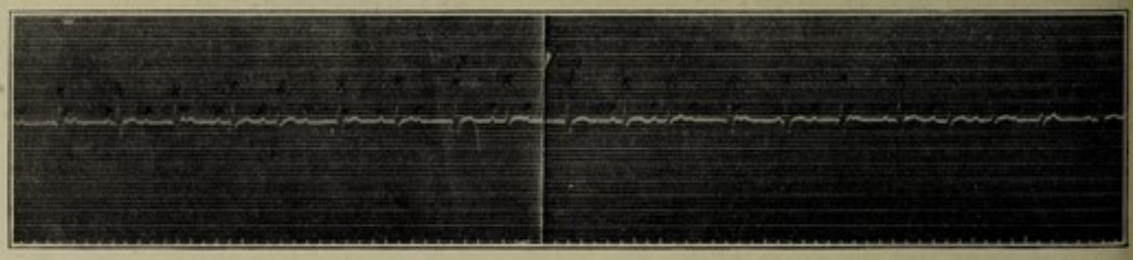


Fig. 11, Case 2.—Lead I, Nov. 23, 1914, 9:25 a. m.

shortness of breath and soon became unable to lie down flat on account of dyspnea. About three weeks after this onset she went into labor and was delivered of a stillborn child. After remaining in bed for two weeks she got up and went about her work though she felt very weak. One month later she developed bronchitis attended with cough and expectoration, at times blood tinged. She began to have palpitation about three months before she came to the hospital and her feet began to swell, the swelling gradually extending up her legs. About three weeks before admission the swelling had involved her abdomen. Notwithstanding these symptoms she had remained up doing her housework as best she could.

When she entered the hospital, Oct. 3, 1914, she was moderately dyspneic and her lips and skin were somewhat cyanotic. She had marked edema of the legs, moderate edema of her forearms, marked edema of her abdominal wall and the lower part of the thorax. Her abdomen contained fluid. There appeared also to be a moderate amount of fluid in each thoracic cavity. The right border of cardiac dulness was 5 cm. to the right of the midsternal line; the left border 16 cm. to the left of the midsternal line. The heart action was rapid and regular with a distinct presystolic gallop rhythm over the base, There was a short systolic murmur heard in the apex region. The pulmonic second sound was markedly accentuated. The systolic murmur present on admission at times disappeared, though it was usually present. Her condition grad-

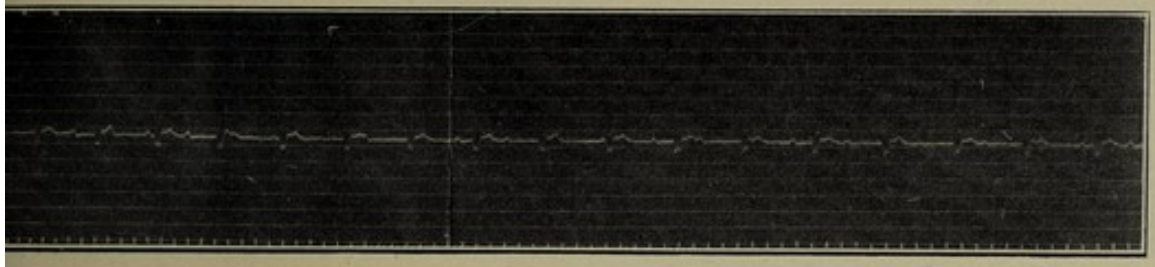


Fig. 12, Case 2.——Lead II, Nov. 23, 1914. Record taken very shortly after that shown in Figure 11.

ually improved, then remained stationary with a varying amount of edema. She received digitalis from time to time with no very marked effect on cardiac rate or rhythm except in the period beginning on November 22 in the afternoon. Up to the day of her death her general condition had not very essentially changed from that at the time of her admission. On November 29 she appeared as usual sitting propped up in bed, and was brightly talking to her neighbors when she suddenly died. We had considered the condition clinically as chronic myocarditis. No necropsy could be obtained.

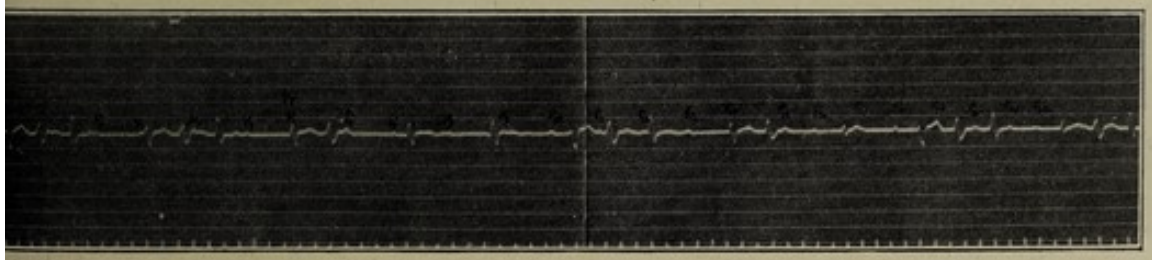


Fig. 13, Case 2.—Lead I, Nov. 23, 1914, 9:45 a. m.

During her stay in the hospital electrocardiographic studies were made. From October 4 to November 22 numerous electrocardiograms showed essentially the same type of tracing with a "P - R" interval of about 0.16 of a second and normal except for a contour of ventricular complexes indicating a moderate degree of right ventricular hypertrophy. During all this period the patient had taken a considerable amount of digitalis as mentioned above, without any very definite effect of any kind except occasional nausea, at which time the digitalis was stopped.

On November 22 after having been on 10 c.c. of an infusion of digitalis three times a day from November 11 to 16, and four times a day from November 16 to 21, on which day the dose was increased in the afternoon to 12 c.c. four times a day, the patient became nauseated at 4:30 p. m., and the digitalis was omitted.

Electrocardiograms were taken on the morning of November 22 and were similar to those taken on the preceding days. That afternoon the pulse fell from 110 to 90 and in the evening to 60, which was its rate next morning, November 23. An electrocardiogram was taken at 9:15 a. m. November 23, and showed at this time a rate of 93 with a long "P - R" interval, at times 0.34 of a second, in Lead 1. In Lead 2, taken a short time after Lead 1, the "P - R" interval had decreased to 0.22 of a second in the first part of the lead. In the latter part of the curve it lengthened out and there was a blocked impulse followed by two ectopic ventricular beats with blunt notched downward waves. In Lead 3 conduction time was similar to the first part of Lead 2. Ten minutes later there was complete auriculoventricular dissociation with an auricular rate

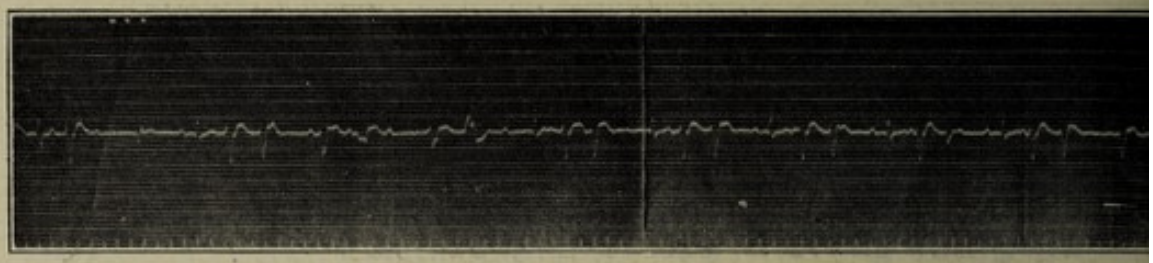


Fig. 14, Case 2.—Lead III, Nov. 23, 1914. Record taken shortly after that shown in Figure 13.

of about 99 and a ventricular rate of about 70. In Lead 1 (Fig. 11), succeeding ventricular complexes showed a change from complex to complex with two general types of waves; one with a moderately high "R" wave and a very slight "S" wave; the other with a slightly higher "R" wave and an "S" wave nearly as great as the "R" wave. In Lead 2 (Fig. 12) there appeared with persisting complete dissociation a succession of broad notched downward waves in the ventricular complex, in form suggesting defective conduction in the right

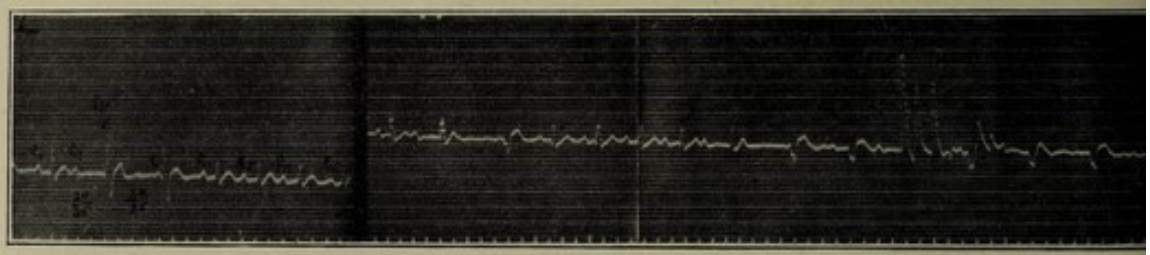


Fig. 15, Case 2.—Leads I and II, Nov. 23, 1914, 3 p. m.

branch of the bundle of His. After a brief interval another record of Lead 2 showed an admixture of ventricular complexes similar to those just described with complexes of normal supraventricular origin or with origin in the junctional tissue. Apparently complete dissociation had been maintained. In Lead 3 most of the ventricular complexes were of normal contour but there were occasional ones in which most of the complex was made up of a rather large downward deflection instead of the usual upward deflection. These downward deflections were not blunt or notched. Twenty minutes later complete dissociation apparently had persisted with now a larger number of ectopic beats in all three leads (Figs. 13 and 14), giving a considerable variation in the type of succeeding complexes.

At 3 p. m. on November 23 the curve in Lead 1 (Fig. 15) was very similar to that which was obtained at 9:25 a. m. on the same day, but the rate was slightly

more rapid. In Leads 2 (Fig. 15) and 3 (Fig. 16) there were considerably more ectopic ventricular beats of apparently various points of origin, and at places these occurred in rapid succession so as to suggest in places temporary ventricular fibrillation.

On the next day, November 24, at 11 a. m., at 3:45 and at 3:50 p. m. the rate was regular and the complex similar to those during the early stay in the hospital, while at 4 p. m. it had become irregular with one or two ectopic beats following the normal beat, consequently a bigeminal or trigeminal (Fig. 17) rhythm.

On November 25 and 26 no electrocardiograms were taken but the heart action judging by the pulse as felt appeared to be normal. On November 27

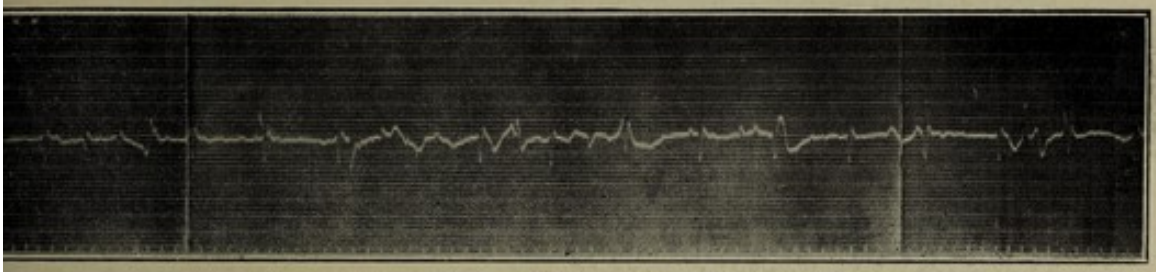


Fig. 16, Case 2.—Lead III, Nov. 23, 1914, 3 p. m.

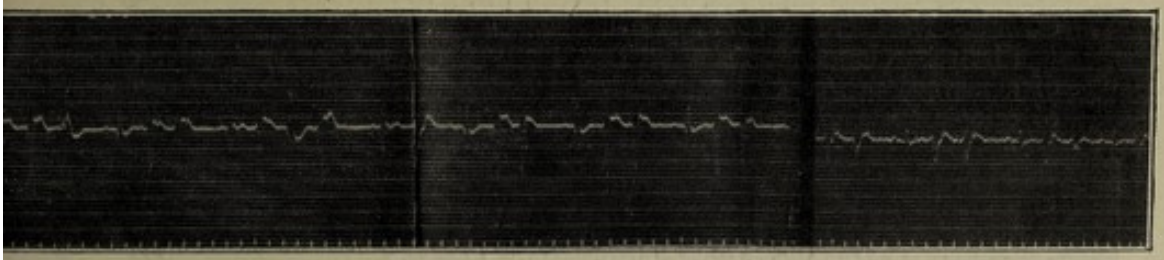


Fig. 17, Case 2.—Lead III, Nov. 23, 1914, 4 p. m.

a normal curve was obtained. No others were taken between this time and the death of the patient on November 29 but in this period no pulse irregularity was noted.

Case 3.—In this case the patient was in the Peter Bent Brigham Hospital (Medical No. 2043) from Dec. 15, 1914, to March 9, 1915, on which day death occurred. This patient was a woman, aged 58, who about Christmas time,

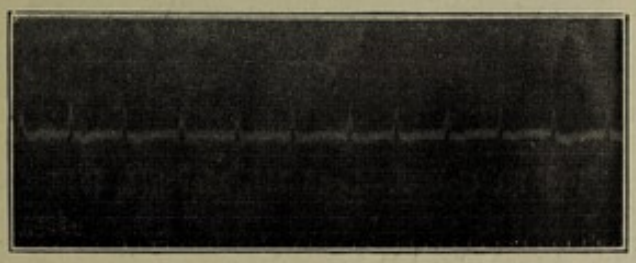


Fig. 18, Case 3.—Lead I, March 5, 1915.

1913, had a rather severe attack of indigestion and shortness of breath. She appears to have had similar symptoms for some time prior to this but never to such a marked degree. At this time dyspnea was severe enough to cause her to remain sitting up in a chair throughout the night. Under treatment she

improved so that she was in good condition until the middle of September, 1914, when the symptoms of Christmas, 1913, reappeared and in addition her legs swelled. These symptoms gradually increased and she became much weaker and feebler and lost considerable weight. When she came to the hospital her area of cardiac dulness extended 3 cm. to the right of the midsternal line and 16 cm. to the left. The heart rate was occasionally interrupted by extra systoles. No murmurs were heard. Her systolic blood pressure was 250. There was marked edema of the legs, abdomen and the lower back. Her eyes showed the picture of albuminuric retinitis. She had the urine of a chronic interstitial

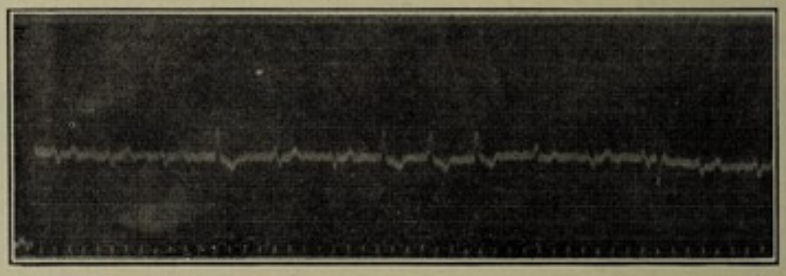
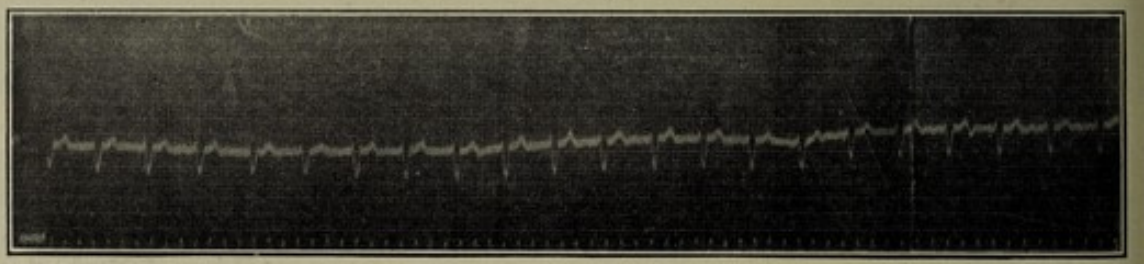


Fig. 19, Case 3.—Lead II, March 5, 1915.

nephritis and functional tests showed that this was of an advanced degree. She gradually grew worse and died on March 9. Clinically it had been a case of chronic interstitial nephritis, hypertension, heart hypertrophy and chronic myocarditis. No necropsy could be obtained.

Electrocardiographic study on Dec. 16, 1914, showed the ventricular contour of a marked degree of left ventricular hypertrophy with a moderate number of ectopic beats of both auricular and ventricular origin. Electrocardiograms



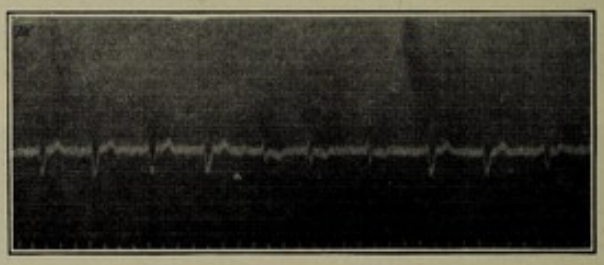


Fig. 20, Case 3.—Lead III, March 5, 1915.

on December 18 showed only ectopic auricular beats. Other electrocardiograms showed no ectopic beats. On March 1, 1915, electrocardiograms showed a typical picture of auricular fibrillation with no ectopic beats. On March 5, 1915, electrocardiograms showed at times a variety of ventricular complexes (Figs. 18, 19 and 20), often changing in contour from beat to beat. In this curve no definite "P" waves could be made out though in places there were suggestions of "P" waves. The ventricular rate was regular as compared with the curve of March 1 when there was definite auricular fibrillation. Con-

sequently it cannot be said definitely what was taking place in the auricles. It may be that there was auricular fibrillation with a regular idiopathic ventricular rate or it may be that the auricles were beating and auriculoventricular dissociation was present, but the curves did not give distinct evidence of this.

During the early part of the patient's stay in the hospital she had had from time to time small amounts of digitalis. From January 29 to March 2, no digitalis was given. On March 2, 8 c.c. of an infusion of digitalis was begun at 10 a. m. and continued three times a day until 7 p. m. on March 5 when it was omitted owing to the character of the electrocardiographic record obtained on that day. The general condition of the patient was growing much worse during and prior to this period of digitalis therapy. Only one electrocardiographic record was taken after March 5. It showed auricular fibrillation without ectopic beats. This was taken on March 8 slightly less than twenty-four hours before the death of the patient.

Case 4.—In this case (Medical No. 2361) the patient was in the Peter Bent Brigham Hospital from Feb. 18, 1915, to March 18, 1915, on which day he was discharged. This patient, a man aged 68, about a year before admission began to notice that he was short of breath. Gradually this grew worse and he developed a cough with tenacious sputum. A distressed feeling in the region of his stomach with failing appetite and constipation ensued. No edema of his legs, however, developed. When he came into the hospital the area of cardiac dulness extended 3 cm. to the right and 14 cm. to the left of the midsternal line. A soft systolic murmur was heard at the apex and the heart sounds were fairly strong. The rate was moderately irregular of the extra systole type of arrhythmia as judged by the finger. The systolic blood pressure was 165 and the radial arteries showed pronounced sclerosis. In the bases of the lungs there were many coarse râles. The liver was slightly enlarged. No subcutaneous edema was made out. The case was regarded as one of chronic myocarditis and chronic nephritis. The patient improved in the hospital and was discharged in good condition. He received 5 c.c. of an infusion of digitalis three times a day from February 24 to February 27.

Electrocardiograms on February 18, 20, 23 and 24 showed a moderate number of ectopic beats of both auricular and ventricular origin and the ventricular complex of left ventricular hypertrophy. Pulsus alternans was present. On February 26, having started on digitalis on February 24 as previously described, the electrocardiograms showed the varying type of ventricular complex which has been described in the previous cases. However, the variations in type of ventricular complex were far less marked than in the cases previously described. The relation of auricular to ventricular beat could not be made out very satisfactorily as the "P" waves were indistinct. However, in places the "P" wave came very close to or was partially incorporated in the "R" wave so that it seems reasonable to suppose that the relation of the contraction of the auricle to the ventricle was similar to that in the previously described cases. On the next day there were found slight variations in some of the succeeding ventricular complexes. Subsequently on March 1, 5, 10, 12 and 13 the electrocardiograms were similar to those of the first days in the hospital.

Cohn offers three possible explanations for the varying ventricular complexes met with in his case. The first assumes a single permanent site in the main stem of the conduction system, above its division into right and left branches, as a source of stimulus to production and that this site is supposed to discharge impulses at regular intervals. Under this assumption the impulses are conducted now in one branch of the system and now in the other, the varying ventricular complexes depending on which branch the impulse traverses. The passage of the

impulse over one branch would, according to this interpretation, be the result of a temporary functional disturbance in the other. It assumes that complete dissociation between the contraction of the auricle and ventricle may be present as a temporary derangement, probably toxic, in the conduction system. The derangement must be of such a nature that impulses could not be conducted from auricles to ventricles, but could be conducted along that portion of the system in continuity with the ventricles, that is to say the portion between the A-V node and the distribution to the ventricular muscle.

The second explanation assumes that the site from which the impulses to contraction come is not fixed, but wanders from one side of the heart, that is to say of the conduction system, to the other. Cohn thinks that impulses so widely scattered as would have to be assumed here may give rise to an orderly succession of contractions, but that such an occurrence is unlikely.

The third explanation assumes that the stimuli to contraction arise synchronously or almost synchronously in the auricle and in the wall of the left ventricle, but the existence of block in Cohn's patient makes this unlikely. On the whole Cohn favors an intoxication as the basic cause of the phenomenon of varying ventricular complexes, assuming that his patient had digitalis intoxication.

Oppenheimer and Williams suggest in their case that the divergent types of ventricular complexes arise from the fact that the intrinsic ventricular pace-maker was frequently shifting or that the different impulses started at the same point and traveled either along different routes or at varying rates along the same route. As they found no anatomic lesion in the auriculoventricular system in their case they say that the heart-block may possibly be of neurogenic or circulatory origin or may be ascribed to chemical agents, to asphyxia or to some hindrance of the passage of impulses from terminal arborizations of the conduction system to the ventricular musculature.

In the cases which I have described variation in form of ventricular complexes was present similar to that described in the cases of Cohn and Oppenheimer and Williams, though not so marked in degree. In addition in my first case there was pairing of beats, a well-recognized digitalis phenomenon. My first patient had received digitalis from March 11 to March 15 in doses of 0.1 gram of powdered leaves every four hours and again from March 30 to April 4 in doses of 0.1 gram of powdered leaves four times a day. The first electrocardiograms taken on this patient were taken on March 20, five days after digitalis was stopped. These and those taken the following day showed the changing ventricular complexes. Subsequently normal cardiac rhythm was restored. On April 3, however, under digitalis therapy the phe-

nomena which occurred on March 20 and 21 reappeared. At necropsy the heart showed no evident lesion of the conduction system, and during life the blocking had been transitory. In my second, third and fourth cases the patients developed these changes under digitalis therapy and the condition was again transient. So it would seem that in my cases as well as in the others the variations in form of ventricular complexes were the result of digitalis action in individuals with hearts decompensated from myocardial insufficiency. Auriculoventricular dissociation developed as a result of digitalis and there was a further disturbance in conduction or in myocardial irritability leading to changing ventricular complexes. It is interesting in this connection that in my second case with auriculoventricular dissociation there appeared temporarily to be defective conduction in the right branch of the bundle of His in addition to the block higher up.

SUMMARY

As an occasional result of digitalis there develops a cardiac disturbance consisting of variations in the form of succeeding ventricular complexes. Of the cases, one reported by Cohn, one by Oppenheimer and Williams and four by myself, all except possibly one of my cases showed auriculoventricular dissociation.[3] In one of my cases the curve obtained did not justify any statement as to auricular activity; there may have been a regular rate in the auricles with auriculoventricular dissociation. All cases were of the type of chronic myocarditis without any marked signs of valve lesions.

Peter Bent Brigham Hospital.