

TERMINAL CARDIAC ARRHYTHMIAS

REPORT OF THREE CASES *

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Because of the frequency with which death of the individual is dependent on cardiac failure, the mode of death of the heart is a matter of considerable interest. Moreover, it is highly probable¹ that one of the mechanisms found to occur in gradual cardiac death, that is, ventricular fibrillation, is responsible for many sudden deaths of otherwise unknown cause, as suggested by McWilliam.² At the present time, the electrocardiogram offers our best means of attacking this problem. Despite the interest in the subject and its importance, there are but few clinical records of terminal mechanisms and fewer of ventricular fibrillation. The paucity of clinical electrocardiographic records of lethal exitus needs no explanation. That ventricular fibrillation is not better known clinically results from the fact that this mechanism is necessarily fatal, if it persists beyond a very few minutes. Experimentally, the condition is rather well known³ since it can be produced without difficulty by faradization of the ventricles,⁴ by occlusion of a coronary artery and by intoxication with digitalis, potassium, chloroform and other agents. In experimental work, death often occurs with the onset of ventricular fibrillation preceded by no known cause. The onset of experimental fibrillation is usually gradual, following ventricular extrasystoles, ventricular tachycardia and a disturbance resembling flutter, in the order named. When fibrillation is established, there are scarcely any movements of the ventricles; the heart is distended and the blood pressure at zero. The galvanometric record is characteristic, showing continuous slow movements of the string which are grossly irregular in amplitude and rhythm, with minute irregular deviations which are perhaps traces of auricular

* From the Cardiographic Laboratory of the Johns Hopkins Hospital and University.

1. Lewis, Thomas: Mechanism and Graphic Registration of the Heart Beat, London and New York, 1920, p. 320.

2. McWilliam, J. A.: On the rhythm of the Mammalian Heart, *J. Physiol.* **9**:167, 1888.

3. Levy, A. G.: Genesis of Ventricular Extra-Systoles Under Chloroform, etc., *Heart* **5**:299, 1913.

4. McWilliam, J. A.: Fibrillar Contraction of the Heart, *J. Physiol.* **8**:296, 1887.

activity. Recovery of the ventricles in experimental animals is not infrequently seen, and it is interesting that this occurs more often in smaller animals.⁵ It is probable that the occurrence of recovery is related to the size of the heart and the mass of the tissue involved.⁶

The earliest clinical record suggesting ventricular fibrillation is Hoffmann's.⁷ This is taken by Lewis as a transitional stage of tachycardia antecedent to actual fibrillation. The patient recovered. Rohmer⁸ reported terminal records of three fatal cases of diphtheria in which complete dissociation occurred. In 1912, Robinson⁹ presented a group of seven cases with fatal outcome, with an electrocardiographic study of exitus. In two instances the records showed the occurrence of ventricular fibrillation after all signs of life recognizable clinically had ceased, and in one of these cases fibrillation was followed by a regular rhythm. Various other arrhythmias, including heart block, ventricular extrasystoles and tachycardia, occurred, without any characteristic sequence. It was noted that there was a tendency for the amplitude of R to diminish while its duration and the amplitude of T increased and R and T gradually became fused. It is to be noted that these patients died as a result of acute infectious diseases without primary cardiac involvement. There was no one point in the heart found to succumb last. Halsey¹⁰ reported a terminal record of a patient ill with pneumonia, in which death coincided with the development of ventricular fibrillation. Robinson and Bredeck¹¹ obtained a record showing ventricular fibrillation, or a stage just short of that condition, from a patient suffering from heart disease, who recovered and lived thirty hours after the cessation of fibrillation. It is of interest that the records of this case showed ventricular extrasystoles with marked variation in the type of the Q R S complex probably indicating a high degree of irritability and abnormal paths of conduction.

The present cases are reported because of the occurrence of interesting arrhythmias determined electrocardiographically shortly before and during the interval immediately preceding death.

REPORT OF CASES

CASE 1.—W. W., male, colored, aged 48, first admitted Oct. 16, 1920, and discharged Oct. 27, 1920.

5. Gunn, J. A.: Ventricular Fibrillation in the Rat's Heart, *Heart*, **5**:1, 1913.
6. Garrey, W. E.: Nature of Fibrillary Contraction of the Heart, *Am. J. Physiol.* **33**:397, 1914.
7. Hoffmann, A.: Fibrillation of the Ventricles, *Heart*, **3**:213, 1911.
8. Rohmer, P.: Ueber das Elektrokardiogramm des Diphtherieherztodes, *München. med. Wchnschr.* **58**:2358, 1911.
9. Robinson, G. C.: Study with the Electrocardiograph of the Mode of Death of the Human Heart, *J. Exper. Med.* **16**:291, 1912.
10. Halsey, R. H.: A Case of Ventricular Fibrillation, *Heart*, **6**:67, 1915.
11. Robinson, G. C., and Bredeck, J. F.: Ventricular Fibrillation in Man with Cardiac Recovery, *Arch. Int. Med.* **20**:725 (Nov.) 1917.

Complaint.—Shortness of breath, cough and swelling of the ankles.

Past History.—Typhoid fever at 7 years of age. Influenza in March, 1918. No history of rheumatic fever or sore throat. Neisserian infection at 21 and again at 42. Nocturia for twenty years. The patient denied syphilis and alcoholism.

Present Illness.—The onset was in January, 1918, with swelling of the ankles. About two weeks after this the patient, on running for a car, became extremely dyspneic. The dyspnea increased until he could not sleep lying down. The patient suffered from cough, marked palpitation and periodic night sweats. For two years puffiness of the eyelids had been quite noticeable in the morning. The swelling of the ankles had been persistent.

Physical Examination.—Temperature, 98.8 F.; pulse, 88; respiration, 22. Tonsils scarred. No general glandular enlargement. In the chest the signs of an old tuberculous process of the right upper lobe were present. Many moist râles over both bases. There was a forceful cardiac apical impulse in the fourth interspace, 14 cm. to the left of the midsternal line. There was a diastolic shock over the base. No thrills felt. The relative cardiac dullness extended 15 cm. to the left and 4 cm. to the right of the midsternal line. There was retromanubrial dullness which reached 5 cm. to each side of the midline. The first sound was followed by a faint high pitched musical murmur at the apex. The second sound was reduplicated. The pulse was regular except for occasional extrasystoles. The peripheral arteries were thickened and tortuous. The blood pressure was 220/140. The liver edge was 8 cm. below the costal margin. No edema of the extremities was present. Ophthalmoscopic examination showed the optic disks to have hazy margins. The arteries were tortuous and the veins distended. No hemorrhages or exudate.

Laboratory Findings.—Blood examination negative. Blood urea, 18.5 mg. Phenolsulphonaphthalein, 60 per cent. in two hours. Wassermann reaction negative. Urinary output, from 1,600 to 2,000 c.c. Sp. gr., 1.010 to 1.028. Occasional trace of albumin, with a few hyaline and granular casts.

Course.—Uneventful. No digitalis therapy. Two electrocardiograms taken during this admission. The patient was discharged free from cardiac failure.

The patient was readmitted Dec. 31, 1920. While out of the hospital he worked part of each day. About three weeks after discharge his ankles began to swell, his dyspnea returned and his eyelids became puffy. There were numerous attacks of vertigo.

Physical Examination.—Marked orthopnea. Slight generalized edema. The breath sounds were suppressed and many moist râles were heard over the base of both lungs. The relative cardiac dullness measured 18 by 4 cm. The pulse was regular. Blood pressure 220/150. In the abdomen shifting dullness and a fluid wave were present. Ophthalmoscopic examination showed several hemorrhages and some exudate.

Laboratory Findings.—Wassermann reaction negative. Blood urea, from 35.8 to 47.2 mg. Phenolsulphonaphthalein, Jan. 5, 1921, 47 per cent. in two hours; Jan. 18, 1921, 26 per cent. in two hours. Urinary output averaged 500 c.c. Albumin and casts constantly present.

Course.—The edema persisted. The patient had constant tachycardia, nocturnal paroxysms of dyspnea and periods of Cheyne-Stokes respiration. The temperature was often subnormal, and the blood pressure varied remarkably. The patient grew progressively weaker, failing to respond to any therapy and died, March 21, 1921. During this admission electrocardiographic records were made twice, and finally just before and after the death of the patient.

Necropsy Findings.—The principal necropsy findings¹² may be summarized as follows: The heart weighed 720 gm. The myocardium showed yellowish

12. Necropsy findings in Cases 1 and 2 are presented by courtesy of Dr. W. G. MacCallum, pathologist to the Johns Hopkins Hospital.

white opacities scattered diffusely throughout, and distinct areas of depressed scars were seen in the surface. There were no valvular lesions observed. Many mural thrombi were present in the left ventricle. Both ventricles were dilated and hypertrophied. The aorta was not enlarged. It was fairly elastic but showed many raised plaques of beginning sclerosis. The lungs showed widespread bronchopneumonia. The spleen contained a recent organizing infarct. The kidneys had adherent capsules; there were several small infarcts and the cortex was thinned. The provisional diagnosis was chronic diffuse nephritis, cardiac hypertrophy and dilatation. Myocarditis with mural thrombi. Anasarca. Infarcts of spleen and kidneys. Edema of lungs. Bronchopneumonia. Pleurisy.

Electrocardiographic Records.—Table 1 presents a summary of the electrocardiographic records of this patient. The first was taken Oct. 16, 1920, the day of admission, and the second (Fig. 1 A) twelve days later. These two records are similar in every respect and present no striking abnormality save a slight prolongation of the Q R S interval

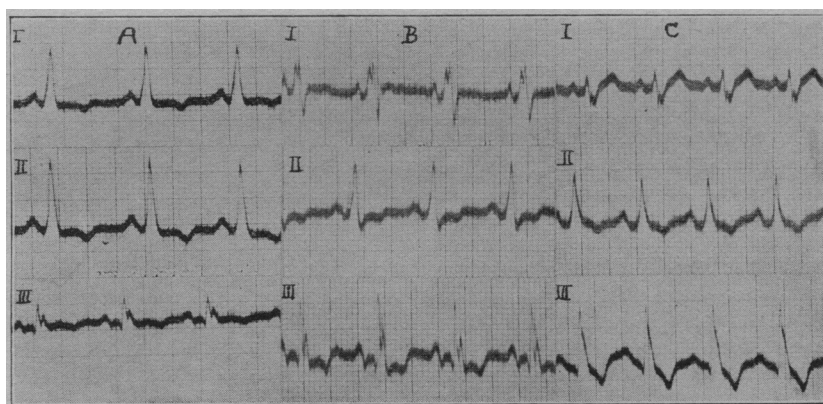


Fig. 1.—Case I. A, Oct. 28, 1920. B, Feb. 1, 1921. C, March 21, 1921 (day of patient's death). The Q R S complex changes remarkably.

and notching of the R wave in Leads I and III. The third record, which was taken Jan. 18, 1921, three months after the first, showed no significant change, except a longer Q R S interval, while in the fourth record (Fig. 1 B), taken February 1, the notching in Leads I and III became more conspicuous. There is a remarkable progression in the type of the Q R S complex from the first to the fifth record. Analysis of the movements of the electrical axis shows that it pursues a fundamentally similar course throughout the records. The explanation and significance of the changes is at present obscure, but the hypothesis of increasing delay in the spread of the impulse throughout the left bundle would explain the outstanding features.

The remaining records were taken practically continuously on the day of the patient's death, beginning about ten or twelve minutes before all clinical signs of life ceased and ending shortly thereafter. Fig. 1 C

shows the three usual leads at the beginning of this period. A marked sinus tachycardia, lengthened P-R and Q R S intervals are the chief new characteristics. The later figures are all of Lead III (the measurements in the table are of Lead III for all the records). The rate is gradually slowed from 120 to 65 in Figure 2. This figure is of great interest because of the changes in the "P" wave shown in it. In the early cycles of this record "P" is definitely positive, then it flattens out, disappears and finally is definitely negative, while the P-R interval is progressively shortened and the rate slowed. These changes may be interpreted either as a migration of the pacemaker within the sino-auricular node or as a shift from the sino-auricular to the auriculo-ventricular node.¹² In Figure 3 but few P waves appear and those which are seen are at a very slow rate and irregular. It would seem that most of the complexes from this point on are nodal since they have approxi-

TABLE 1.—MEASUREMENTS OF ELECTROCARDIOGRAMS OF CASE 1 (LEAD III)

Film No.	Fig. No.	Date	Rate	Rhythm	Duration, Sec.			Amplitude, Mm.			Notes
					P-R	QRS	Q-T	P	R	T	
3077-1	...	10/16/20	85	Reg.	0.15	0.08	0.32	0.75	6.0	-1.0	All "T's" —
3077-2	1A	10/28/20	94	Reg.	0.15	0.09	0.33	1.0	5.0	-0.5	All "T's" —
3077-3	...	1/15/21	95	Reg.	0.15	0.10	0.32	1.75	-9.0	-1.25	T ₁ +, T ₂ —, T ₃ —
3077-4	1B	2/ 1/21	105	Reg.	0.15	0.10	0.29	1.75	12.0	-1.5	T ₁ +, T ₂ —, T ₃ —
3077-5	1C	3/21/21	120	Reg.	0.16	0.10	0.27	1.0	11.5	-3.5	T ₁ +, T ₂ —, T ₃ —
3077-6	2	3/21/21	86-65	Irreg.	0.21-0.15	0.11	0.29	1.5	11.0	-3.0	Nodal rhythm
3077-7	3	3/21/21	60	Irreg.	0.42	0.12	0.32	1.0-0.0	13.0	-2.0	Nodal rhythm
3077-8	4	3/21/21	60	Irreg.	0.12	0.44	3.0	4.0	Ventricular fibrillation
3077-9	5	3/21/21	55	Irreg.	0.12	0.42	2.5	7.0-3.0	Ventricular fibrillation
3077-10	...	3/21/21	47.5	Irreg.	0.08	0.26	2.0-0.0	0.5	

mately the normal outline. It is interesting that the coupling recorded is almost certainly due to occasional responses to impulses built up higher up in the auricular wall, since "P" waves appear at a constant P-R interval in connection with two of the coupled complexes, while the P which belongs with the third complex is hidden by the first R of that couple. Definite, moderately advanced ventricular fibrillation is shown in Figure 4 and again in Figure 5. Both periods of fibrillation are brief and are succeeded by series of abnormal complexes with no trace of P waves. The second period of fibrillation is a more advanced disorder than the first. With reference to the abnormal complexes, their origin cannot be definitely stated. The "T" waves are of high amplitude and positive whereas they were previously always negative. The tendency for the R and T waves to coalesce, pointed out by

12. Lewis, T.: Effect of Vagal Stimulation on Atrio-Ventricular Rhythm, *Heart* 5:247, 1914. Lewis, T., Meakins, J., and White, P. D.: Excitatory Process in the Dog's Heart, *Phil. Tr. Roy. Soc. Lond.*, B 205:375, 1914.

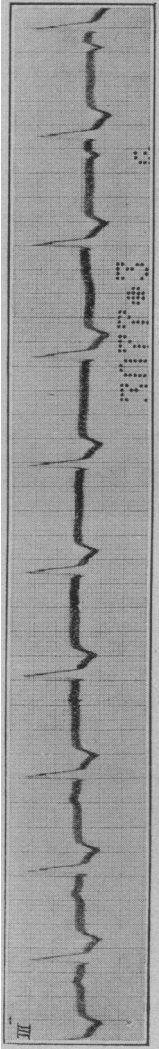


Fig. 2.—Case 1. Lead III. Ten minutes before the patient's death. Note the change in the P wave.

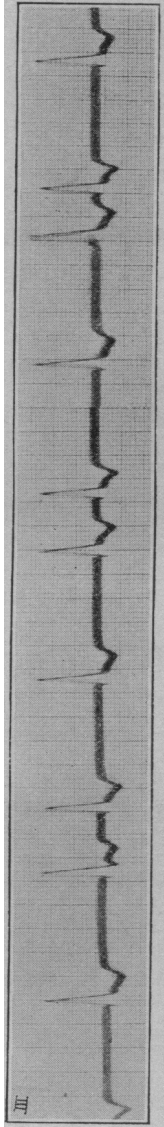


Fig. 3.—Case 1. Lead III. A few seconds later than Figure 2. Nodal rhythm with occasional coupling due to impulses of auricular origin.

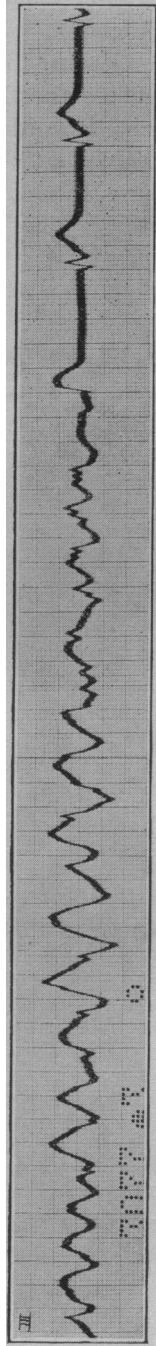


Fig. 4.—Case 1. Lead III. Five minutes after Figure 3. Ventricular fibrillation followed by regular complexes of ventricular origin.

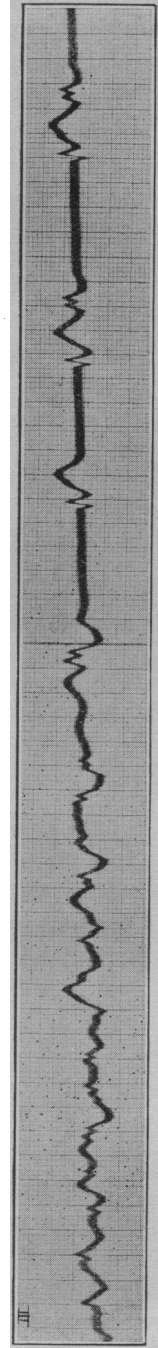


Fig. 5.—Case 1. Lead III. A few seconds later. Ventricular fibrillation followed by regular complexes similar to those of Figure 4 with occasional complexes of a second type.

Robinson,⁹ is shown to some extent. The amplitude of the R waves gradually decreases more and more rapidly toward the end of the record. No attempt to record the exact instant of "clinical death" was made, but this occurred shortly before the last record was taken.

To summarize the electrocardiographic findings of this case, we may say that the rate gradually slowed, while the site of impulse

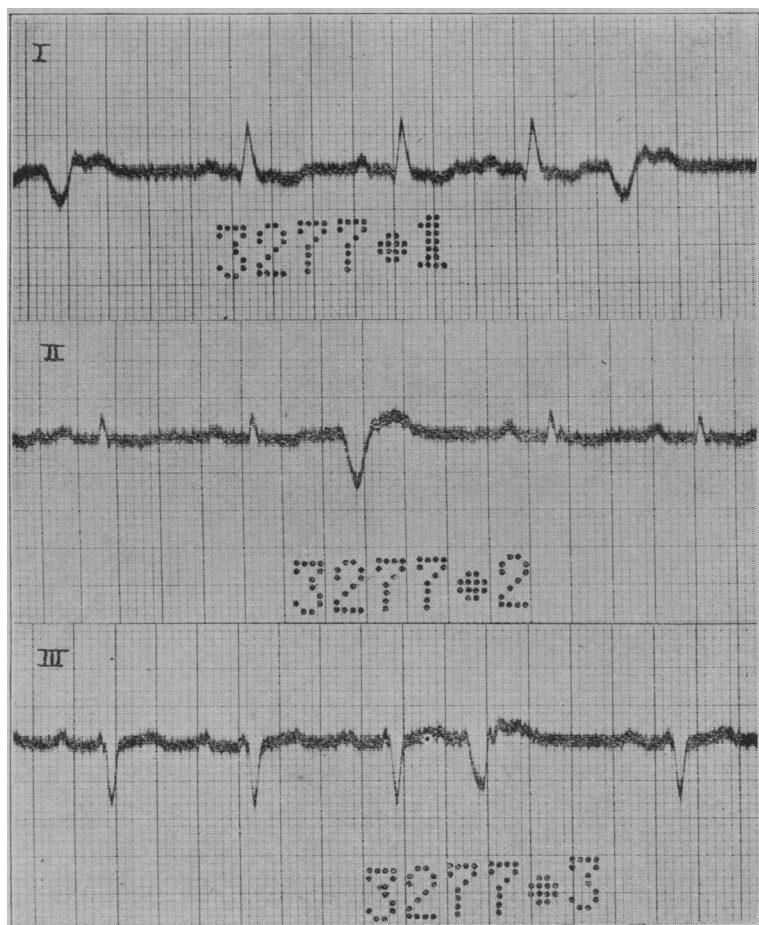


Fig. 6.—Case 2. April 25, 1921. Note the form of the normal and ectopic complexes.

formation either shifted within the sino-auricular node or passed to the auriculo-ventricular node, the conduction time decreased and then all definite evidence of auricular activity ceased. There were two brief periods of ventricular fibrillation interrupted by and followed by fairly regular but gradually slowing complexes due to impulses arising in an undefined part of the heart. The amplitude of the complexes

gradually decreased until it was very small when they finally ceased. The autopsy revealed no changes definitely associated with any of the electrocardiographic findings.

CASE 2.—S. B., male, colored, aged 48, admitted April 23, 1921.

Complaint.—Swelling of legs, shortness of breath and weakness.

Past History.—General health always good up to 1918. Neisserian infection in 1913. In 1918 the patient was admitted to this hospital with cough, shortness of breath and edema, and was found to have arteriosclerosis and hypertension. While in the hospital the patient suffered a right-sided hemiplegia, which cleared up. Between admissions the patient was able to work as usual.

Present Illness.—Began three months before admission with cough, dyspnea and edema which grew worse until the patient became very weak.

TABLE 2.—MEASUREMENTS OF ELECTROCARDIOGRAMS OF CASE 2 (LEAD III)

Film No.	Fig. No.	Date	Rate		Rhythm	Duration, Sec.			Amplitude, Mm.			Notes
			A.	V.		P-R	QRS	Q-T	P	S	T	
3277-1	6	4/25/21	77	77	Reg.	0.19	0.10	0.31	0.75	7.0	0.5	Ventricular extra-systoles Complete dissociation
3277-2	7	4/26/21	65	65	Irreg.	0.31 to 0.12	0.10	0.38	-1.0	11.0	1.5	
3277-3	..	4/26/21	58	58	Reg.	0.10	0.36	-1.0	7.0	1.0	
3277-4	..	4/26/21	56	57	Reg.	0.10	0.36	-1.0	7.5	1.0	Series of V. E. S.
3277-5	..	4/26/21	58	59	Irreg.	0.10	0.36	-1.0	7.0	1.0	
3277-6	8	4/26/21	56	60	Irreg.	0.10	0.37	-1.0	7.5	1.0	
3277-7	..	4/26/21	57	73	Irreg.	0.11	0.38	-1.0	7.0	1.0	Auricular fibrillation; series V. E. S. Series V. E. S.
3277-8	9	4/26/21	56	58	Irreg.	0.11	0.38	-1.0	7.0	1.0	
3277-9	..	4/26/21	55	61	Irreg.	0.11	0.38	-1.0	7.5	1.0	
3277-10	..	4/26/21	56	64	Irreg.	0.11	0.38	-1.0	7.5	1.0	Series of 46 V. E. S.
3277-11	10	4/26/21	50	48	Irreg.	0.11	0.40	-1.0	7.0	0.5	
3277-12	..	4/26/21	50	32	Irreg.	0.11	0.44	-0.5	7.0	0.5	
3277-13	..	4/26/21	44	32	Irreg.	0.11	0.42	-0.5	6.0	0.5	Auricular fibrillation Auricular fibrillation; series of 34 V. E. S. Auricular fibrillation
3277-14	..	4/26/21	?	55	Irreg.	0.11	0.42	?	6.5	0.25	
3277-15	..	4/26/21	38	75-10	Irreg.	0.11	0.41	?	5.0	0.25	

Physical Examination.—Emaciation. Pallor. Edema of dependent parts. Râles at bases of lungs. The relative cardiac dulness extended 12 cm. to the left and 4.5 cm. to the right of the midline. The pulse showed numerous extrasystoles with a deficit of 10 per minute. The liver edge was 4 cm. below the costal margin.

Laboratory Findings.—The blood showed a marked secondary anemia and a slight neutrophilic polymorphonuclear leukocytosis. Phenolsulphonephthalein, 59 per cent. in two hours. Wassermann reaction negative. The urine showed albumin and a few hyaline and granular casts.

Course.—The patient sank gradually and died April 26, three days after admission.

Necropsy Findings.—Marked emaciation. Extreme anasarca. The heart weighed 720 gm. The coronary arteries were tortuous and sclerotic. The left ventricle was much hypertrophied and dilated, and contained a large mural thrombus measuring 7 by 5 by 5 cm. Mural thrombi were also present in both auricles. The myocardium showed numerous opaque grayish areas of scarring. Chronic passive congestion of the lungs. Multiple cysts containing concretions in the left kidney. Slight chronic diffuse nephritis. Generalized arteriosclerosis.

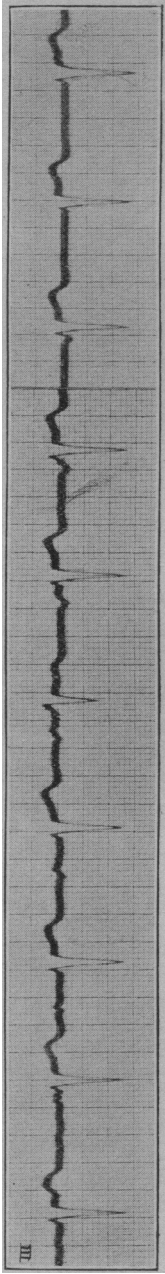


Fig. 7.—Case 2. Lead III. April 26. Twenty minutes before patient died. Note the changes in the P wave.

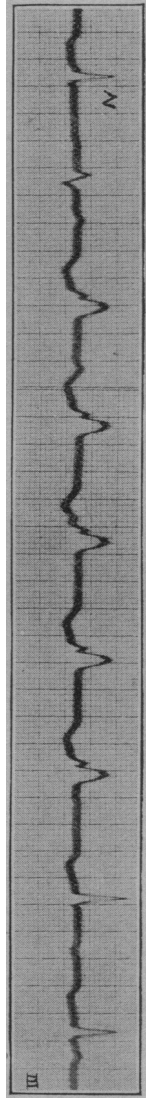


Fig. 8.—Case 2. Lead III. Five minutes later. Series of five ventricular extrasystoles with retrograde P waves.

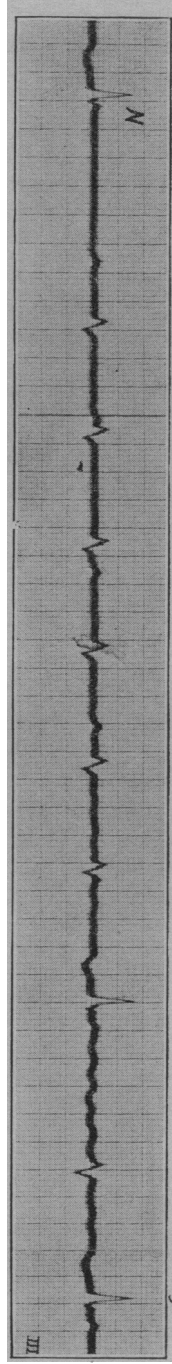


Fig. 9.—Case 2. Lead III. A few minutes later than Figure 8. Transient auricular fibrillation and series of six ventricular extrasystoles of a second type. N = normal complex.

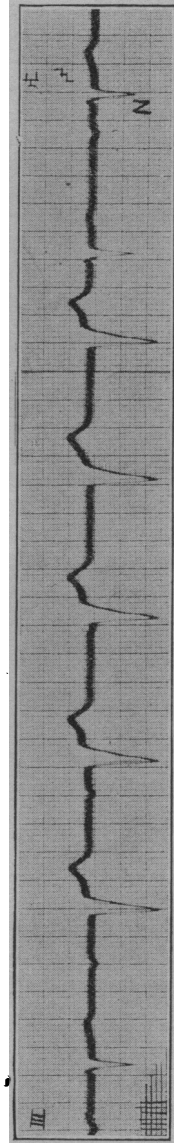


Fig. 10.—Case 2. Lead III. Five minutes after Figure 9. Series of five ventricular extrasystoles of third type.

Electrocardiographic Records.—Electrocardiographic records of this patient were made April 25, two days after his admission, and again April 26, at short intervals during the thirty minutes just before and during the patient's death. Table 2 gives the measurements of these records (the figures are for Lead III throughout). The first record (Fig. 6) shows a normal mechanism with left ventricular preponderance and occasional ventricular extrasystoles of a form which frequently recurred in the records of exitus. During the taking of Lead III of the second record (Fig. 7) the P waves changed their form from positive to negative and a complete dissociation was apparently established. Occasional ventricular extrasystoles, mainly of three types, occurred throughout the remainder of the records. It is interesting that paroxysms or series of varying lengths of each form are found. A brief series of five ventricular extrasystoles of the first type with apparently retrograde P waves is shown in Figure 8, and records 14 and 15 show a series of fifty-six extrasystoles of the same form. Figure 9 is an illustration of the second type of extrasystole which is not retrograde but is accompanied by independent P waves and a brief period of auricular fibrillation. A short paroxysm of ventricular extrasystoles of a third form appears in Figure 10. This record just precedes the onset of a series of forty-six extrasystoles of the same type. This form also is accompanied by independent P waves at a slower rate. These series seem to meet the criteria suggested by Robinson and Herrmann¹³ for ventricular tachycardia, although in this instance the rate is only relatively rapid. There are four periods of temporary auricular fibrillation, one of which is mentioned above. Ventricular fibrillation did not occur in this case. The rate of the auricular contractions was established at from 56 to 58 at the onset of complete dissociation, and gradually fell to 38. The ventricular rate varied greatly from 70 to intervals in the last record corresponding to a rate of 10. The amplitude of R and T gradually decreased and the Q R S and Q-T intervals increased slightly. No tendency for R and T to fuse is shown. It would seem that the ventricles of this patient had several irritable foci which gave rise to the various abnormal types of ventricular complexes. It is interesting that notwithstanding this fact, ventricular fibrillation did not supervene.

The necropsy of this case revealed marked myocardial changes and abnormal left ventricular preponderance, thus confirming in a general way the important electrocardiographic findings.

CASE 3.—M. M., female, white, aged 23, admitted April 27, 1921.

Complaint.—Chills and fever; shortness of breath and palpitation.

13. Robinson, G. C., and Hermann, G. R.: Paroxysmal Tachycardia of Ventricular Origin, *Heart* 8:59, 1921.

Past History.—Acute rheumatic fever at 12. Admitted to this hospital in 1911 with mitral stenosis and insufficiency, aortic insufficiency, bronchopneumonia and pleurisy. After an interval, tonsillectomy was performed in the same year. In 1919, the patient was on the obstetric service suffering from toxemia of pregnancy. The patient had frequent sore throats, more or less constant slight dyspnea and transient slight edema of the legs.

Present Illness.—Onset two weeks before admission with sore throat, chills and fever. The patient developed acute rheumatism. She became very weak.

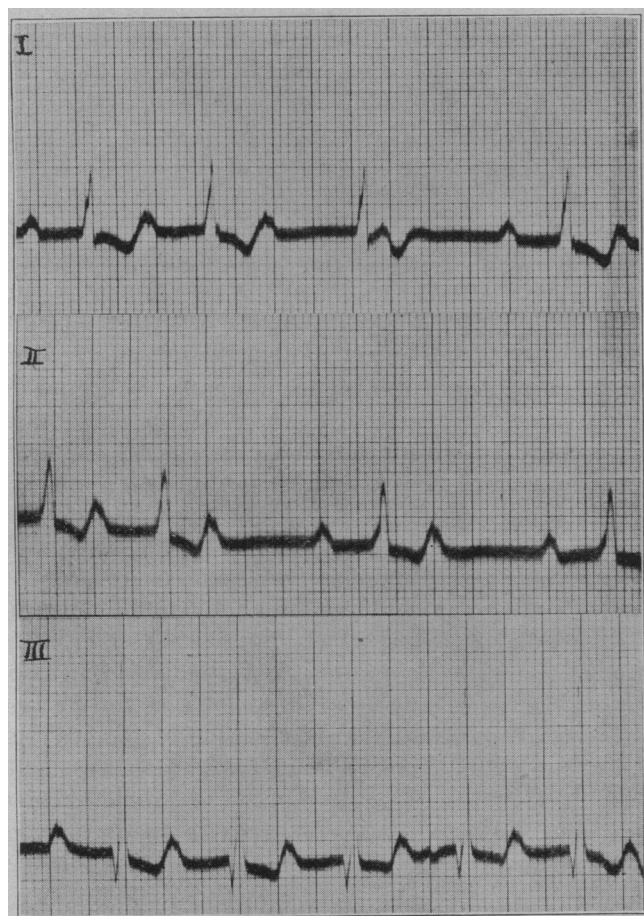


Fig. 11.—Case 3. April 29, 1921. Second degree heart block. Note the form of the Q R S complex.

Physical Examination.—Temperature, 101.6 F.; pulse, 112; respiration, 28. Orthopnea and cyanosis were marked. There were no râles in the lungs. There was a marked precordial heave. The apical impulse was 17 cm. to the left of the midline. The relative cardiac dulness measured 20.5 by 4.5 cm. There was retromanubrial dulness, 3 cm. to each side of the midline. There was a loud systolic murmur at the apex and a “to and fro” murmur at the aortic area. “Pistol shot” sounds were audible over the femoral vessels. The pulse

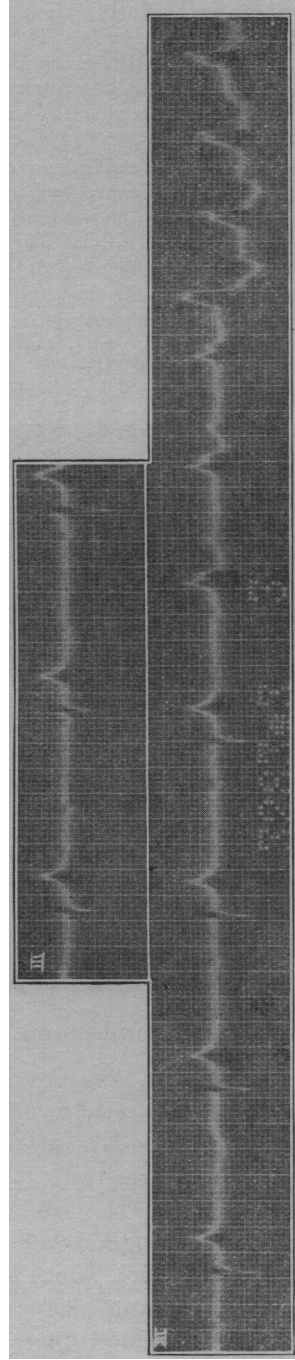


Fig. 12.—Case 3. Lead III. May 3. Five hours before patient's death. Complete heart block (see upper record) with onset of ventricular tachycardia.

was regular and was of the "water hammer" type. There was a distinct capillary pulse. The blood pressure was 146/44. The liver edge was 5 cm. below the costal margin and was tender. There was slight edema of the legs.

Laboratory Findings.—The blood showed a secondary anemia and a slight neutrophilic polymorphonuclear leukocytosis; culture negative. The Wassermann reaction was negative.

Course.—The patient received 18 c.c. of tincture of digitalis which was discontinued because of the occurrence of dropped beats. Three days later the pulse rate suddenly dropped to 38 and a Stokes-Adams attack occurred. This was repeated several times. One-fortieth grain of atropin was given with no effect on the pulse rate. Electrocardiographic records showed the presence of complete heart block with periods of ventricular tachycardia. The patient became progressively worse, with increasing dyspnea, cyanosis, pulmonary congestion and insufficient cerebral circulation. She was given 0.15 mg. ouabain but grew weaker and died the same evening (May 2). There was no necropsy.



Fig. 13.—Case 3. A few minutes later than Figure 12. Ventricular tachycardia with retrograde P wave.

Electrocardiographic Records.—An electrocardiographic record of this patient was made April 29, two days after her admission. It shows a long P-R interval which progressively increases until a beat is dropped and negative T waves (Fig. 11). In view of the patient's history and of the small amount of digitalis administered and of the failure of atropin to relieve the block which later occurred, it is thought that these abnormalities indicate definite myocardial disease and that they were not the result of digitalis therapy. A second record was made May 3, about three hours before the patient died. This shows complete heart block interrupted by paroxysms of ventricular

tachycardia (Fig. 12). The three leads of this tachycardia are shown in Figure 13. The form of the ventricular complexes in the paroxysms is entirely different from that which occurred during the complete dissociation. In Lead I the ventricular complexes are followed by positive P waves (note that the P waves during the idio-ventricular rhythm are negative). Records were not obtained later.

DISCUSSION

The electrocardiographic findings in these cases are to a certain extent fundamentally similar, in spite of the fact that it seems at first as if we were dealing with a heterogeneous group of records. This is perhaps to be expected since in any terminal record there are not only the direct effects of the special cardiac lesions which may be present but also the results of the general pathological conditions which accompany extreme cardiac failure. Marked impairment of the respiratory function with rapidly increasing insufficiency of oxygen and accumulation of carbon dioxide is the outstanding pathological condition from the functional point of view.

The following features are common to the three cases described: gradual slowing of rate, accompanied by lengthening of the P-R, Q R S and Q-T intervals and diminution in the amplitude of the R and T waves. The normal pacemaker lost control of the rhythm in the first and second cases, probably due to failure both of stimulus production and of impulse conduction. In these instances auriculoventricular nodal rhythm was established. The P waves show that the auricles were contracting irregularly but independently, so that the block must have been retrograde as well as forward. The independent auricular rate was slower than the ventricular and the auricular activity ceased before the nodal rhythm failed. In the second case brief periods of auricular fibrillation occurred. The sino-auricular node early ceased to be the pacemaker in the third case through the failure of the conduction system which was undoubtedly diseased in this instance. This patient's clinical condition showed extreme anoxemia, the myocardium at no time being equal to the task of maintaining a proper circulation.

The more individual abnormalities are, in the first case, interruption of the nodal rhythm by two periods of ventricular fibrillation. In the second case a similar interruption by series of varying lengths of ventricular extrasystoles arising in at least three different foci. Definite paroxysms of ventricular tachycardia occurred in the third case. The underlying pathological physiology of these arrhythmias is probably fundamentally the same.

The later records of the first case show to some extent a tendency for the R and T waves to coalesce, a change which as was stated above the records of Robinson's⁸ cases show consistently. This phenomenon

is said by Samojloff¹⁴ to be associated with a lesion of the apical portion of the myocardium.

In a recent contribution dealing with circulatory responses to oxygen want under experimental conditions Greene and Gilbert¹⁵ present data which are strikingly similar to the early general changes in the cases under discussion. There seems to be no doubt that the sino-auricular node is peculiarly susceptible to oxygen want and loses the function of impulse production early. The function of conduction is also greatly depressed. Whether this is due to vagospasm or direct myocardial asphyxiation is discussed by Greene and Gilbert without reaching any conclusion. No further light can be added on this question at present. Experimental work has not yet been carried to the point of ventricular arrhythmias such as occurred later in the cases presented. It would seem that it might be predicted that these arrhythmias will be found to develop when experimental data are gathered on the further effect of oxygen want after interference with impulse production in the auriculoventricular node. It should be pointed out that the present cases are complicated in comparison with those of Greene and Gilbert by the accumulation of carbon dioxid.

SUMMARY

Terminal electrocardiographic records of three cases are presented and briefly discussed. The general changes which occurred were a gradual slowing of the cardiac rate with coincident lengthening of the P-R, Q R S and Q-T intervals and diminution in the amplitude of the R and T waves; and loss of control of the rhythm by the normal pacemaker, apparently with the inception of auriculoventricular nodal rhythm. Further, as a result of the functioning of various abnormally irritable foci, the following arrhythmias were observed in addition to heart block and nodal rhythm; auricular and ventricular extrasystoles; ventricular tachycardia and auricular and ventricular extrasystoles; ventricular tachycardia and auricular and ventricular fibrillation. It is probable that oxygen want, and perhaps carbon dioxid accumulation, following cardiac failure, underlie these abnormalities in such cases as those presented.

14. Samojloff, A.: Weitere Beitrage zur Elektrophysiologie des Herzens, Arch. f. d. ges. Physiol. **135**:417, 1910.

15. Greene, C. W., and Gilbert, A. C.: Responses of Circulation to Low Oxygen Tension, Arch. Int. Med. **27**:517 (April) 1921.