A STUDY WITH THE ELECTROCARDIOGRAPH OF THE MODE OF DEATH OF THE HUMAN HEART.*

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PLATES 34–38.

The changes in the mechanism of the heart-beat which occur at the time of death have been repeatedly observed experimentally in animals, and from these observations it has been generally believed that that part of the heart in which the property of rhythmicity is the most highly developed is the part that ordinarily continues to beat longest as the heart dies. The evidence seems conclusive that it is in the region where the superior vena cava joins the right auricle that rhythmicity is most highly developed, and that it is in this region that the stimulus of the heart-beat originates. Here it is, in other words, that the cardiac pace-making region is situated, and this function belongs apparently to the specialized tissue found along the sulcus terminalis, which constitutes the node of Keith and Flack.

Koch[1] has questioned the belief that this region in the right auricle, the ultimum moriens, is the last part of the heart to die in man, and he bases his objections on observations on four human fetal hearts which returned to activity after the chest was opened soon after birth. He believed that the region of the coronary sinus was the last part of the human heart to die. Hering[2] criticizes his findings and says that the incisions made in the hearts were responsible for the lack of agreement between Koch's observations and his experiments.

Rohmer[3] made electrocardiographic studies of fatal cases of diphtheria and reported that in three complete dissociation of auricles and ventricles occurred. He found an unusual form of ventricular complex which he thought was due to damage to the myocardium. His paper with curves has not as yet been published. Koch and Rohmer have furnished the only observations which deal directly with the changes in the mechanism of the dying human heart.

* Received for publication, May 23, 1912.

[1] Koch, W., *Beitr. z. path. Anat. u. z. allg. Path.*, 1907, xlii, 203.

[2] Hering, H. E., *München. med. Wchnschr.*, 1909, lvi, 845.

[3] Rohmer, *München. med. Wchnschr.*, 1911, lviii, 2358.

The present study is based upon electrocardiographic records obtained from seven patients before and during the actual stoppage of the heart. There were two cases of poliomyelitis, one of pneumococcus meningitis, and four cases of lobar pneumonia. Death in the cases of poliomyelitis occurred from paralysis of the respiratory muscles, the children being otherwise in relatively good physical condition. As wires run throughout the hospital, records could be taken without moving or disturbing the patients in any way. The second lead was used in each case, the German silver electrodes being attached to the right arm and left leg by bandages wet with salt solution.

I sought to obtain records of the cardiac mechanism just before the occurrence of death and to continue taking records at frequent intervals as long as any cardiac activity persisted. There were naturally many failures to obtain records, especially when fatalities occurred suddenly; while success in some instances followed only after hours of careful watching with the electrodes in place and the galvanometer in operation.

Clinical death was considered to have occurred when respiration finally ceased, when no heart sounds could be heard, and when muscular relaxation and the general appearance of the patient indicated to the physician that death had occurred. Not until all these conditions were fulfilled was the patient considered dead. The electrocardiograms have been analyzed in order to determine the changes in the mechanism of the heart-beat in relation to the time of clinical death in each case. The rate of the heart-beat has been calculated; the conduction time from auricles to ventricles (P–R time), and in some cases through the ventricles as indicated by the duration of the QRS complex, and the length of ventricular systoles (R to end of T) have been measured in seconds. The heights of the various waves have been measured in millimeters and the results of these analyses are seen in the tables.

Case 1.—A child, eighteen months old, suffered with poliomyelitis and died of paralysis of the respiratory muscles. The heart sounds could be heard occurring regularly for three minutes after respirations ceased, and clinical death is considered to have occurred when the heart sounds were no longer audible. The rate of the heart-beat gradually declined from 132, eleven minutes before death (figure 1), to 45, ten minutes later. The cessation of respiration produced no

TABLE I.

Case 1. Age 18 months. Poliomyelitis. Curve No. 134.

Number of curve.	Number of figure.	Time.	Rate per minute.		Conduction time in seconds.		Length of ventricular systole in seconds.	Height of waves in mm.		
			Auricular.	Ventricular.	P-Q.	Q to end of R.	Q to end of T.	P.	R.	T.
134.3	1	11 min. a.m.[4]	132.0	132.0	0.10	0.057	0.24–0.26	1.4	11.5	2.0
		8 min. a.m.	113.0	113.0	0.11	0.053	0.25	1.3	11.2	2.0–2.5
		5 min. a.m.	59.0	59.0	0.09–0.10	0.066	0.30–0.31	1.3	10.5	3.2
		4 min. a.m.	54.7	54.7	0.12	0.066	0.31	1.3	10.0	3.5–4.0
		3 min. a.m.	54.0	54.0	0.12	0.066–0.077	0.30–0.315	1.0	10.0	3.0
		Respiration ceased here without affecting electrocardiogram.								
		1 min. a.m.	45.0	45.0	0.14	0.068	0.34	1.0	9.0	2.5
134.11	2	8½ min. p.m.	—	22.6	——	——	0.26	—	11.0	5.0
			Auricles not beating.							
134.12	3	10 min. p.m.	39.0	17.3	——	0.083	0.29	1.0	9.5–10.0	5.5
			Complete block.							
		11 min. p.m.	0	15.8	——	0.093	0.30	—	9.0	5.5–6.0
			Auricles not beating.							
134.14	4	11½ min. p.m.	28.5	15.4	——	——	0.29	—	8.0–8.5	5.5
		To show change in complex.								
		11¾ min. p.m.	Occasional beat.	16.2	——	——	0.27–0.28	—	8.0	5.0–5.5
		12½ min. p.m.	Occasional beat.	18.2	——	——	0.311	—	6.5	—
		13½ min. p.m.	Occasional beat.	22.2	——	——	0.29	—	6.5	—
		14 min. p.m.	Occasional beat.	26.0	——	——	0.29	—	7.5	—
		14½ min. p.m.	Occasional beat.	—	——	——	0.28	—	8.0	—
				Long pause.						
134.22	5	16½ min. p.m.	Occasional beat.	35.0	——	——	0.28	—	8.0	—
		17½ min. p.m.	—	37.0	——	——	0.30	—	8.0	—
		No movement of string after 18 minutes p. m.								

[4] In the tables a. m. stands for ante mortem, p. m. for post mortem.

appreciable effect. The conduction (P-R) time gradually lengthened from 0.10 to 0.14 of a second during this time. Records could not be obtained until eight and a half minutes after death on account of an accident to the instrument. Then the P-wave was absent, indicating cessation of auricular activity, the ventricles beating 22.6 times per minute (figure 2). Ten minutes post mortem the auricles were again beating regularly at a rate of 39 per minute, while the R- and T-waves indicated that the ventricles beat 17.3 times a minute, complete dissociation being present (figure 3). The auricles again ceased beating for a short time and then began again, at first at a rate of 28.5 (figure 4). Afterwards they beat only occasionally and irregularly until sixteen and a half minutes post mortem, when they finally ceased. The ventricles continued to beat until eighteen minutes post mortem and gradually increased in rate from 15.4 per minute, at eleven and a half minutes post mortem (figure 3), to 35, at sixteen and a half minutes post mortem (figure 5). This increase in rate seems to depend, however, at least in part, on the rest afforded the heart by the long pauses that occurred. In this case then the ventricles beat after the auricles had ceased. The curves reproduced from those obtained in this case show the characteristic changes in the ventricular complex which occurred in every case. These changes consist in a decrease in the size of the R-wave, an increase in the size of the T-wave, and a gradual fusing of these two waves. There is no striking change in the duration of ventricular systole in this case. The time of conduction of the impulse throughout the ventricles, as measured from the beginning of Q to the end of R, gradually lengthened from 0.057 to 0.093 of a second. On account of the fusion of R and T, it could not be measured later than eleven minutes post mortem (table I).

Case 2.—A child of four years, with poliomyelitis, died of respiratory paralysis. The time when the heart sounds ceased to be heard was not recorded, but on account of definite changes that took place suddenly in the electrocardiograms corresponding to changes that occurred in other cases at the time of clinical death, the time of these changes is inferred to be the time of clinical death.

For several hours before death the patient breathed with great difficulty, one half of the diaphragm alone supporting respiration. Four hours and a half before death, marked changes in rate occurred in a fairly rhythmic manner (figure 6). Diminution in the size of the P- and T-waves occurred with the periods of slow rate, producing an electrical complex resembling that seen when the vagus tone is raised (Rothberger and Winterberg[5]). It seems probable that these changes in rate and in the form of the complex are dependent on changes in vagus tone accompanying respiratory movements.

At the time when clinical death is inferred to have occurred, there was marked slowing of the cardiac rate and the auricular activity apparently ceased, the ventricles contracting alone at a rate of 61 per minute. The auricles were seen (figure 7) to become active once more, and when they set the pace of the ventricles again, the rate increased to 90 per minute. The auricles stopped beat-

[5] Rothberger, J., and Winterberg, H., *Arch. f. d. ges. Physiol.*, 1910, cxxxv, 506.

TABLE II.

Case 2. Age 4 years. Poliomyelitis. Curves 118–122.

Number of curve.	Number of figure.	Time.	Rate per minute.		Conduction time in seconds.		Length of ventricular systole in millimeters.[6] R to end of T.	Height of waves in mm.		
			Auricular.	Ventricular.	P–R.	R–S.		P.	R.	T.
		60 hrs. a.m.	125	125	0.14	0.067	0.29	2.3	9.5	3.0
		10 hrs. a.m.	91	91	0.13	0.065	0.30	1.3	12.5	3.0
119.7	6	4 hrs. and 35 min. a.m.	100	100	0.14	0.061–0.008	0.28–0.29	1.0–1.5	9.0	2.0–3.5
		There is a marked change of rate at this time. Changes in conduction through the bundle and through the ventricles do not go parallel.								
121.4	7	Death probably here.	——	61.8	——	——	11.0 mm.	——	5.0	3.0
			90.0	90.0			13.0 mm.	——	6.0	2.5
		Rate more rapid when auricles set the pace.								
		½ min. p.m.	Not active.	58.5	——	——	13.0 mm.	——	5.5	3.0
		¾ min. p.m.	57.6	57.6 Not delayed.	——	——	11.0–12.0 mm.	——	——	——
		The auricles return and take up the pace in this curve after having ceased beating for 15 seconds. The rate is not altered.								
		5 min. p.m.	56.8	56.8	Delay in P–R time.		10.0 mm.	1.5	4.5	3.5
		12 min. p.m.	49.4	About 33 (irregular).	——	——	10.0 mm.	——	——	——
		There is partial block, 3 to 2 rhythm being present. Conduction much delayed when a beat gets through.								
		17 min. p.m.	——	21.4	——	——	10.6 mm.	——	7.5	2.0
		No evidence of auricular activity; ventricles regular.								
		18 min. p.m.	54.8	18.0	——	——	10.0 mm.	——	7.5	3.0
		19 min. p.m.	39.2	14.7	——	——	9.2 mm.	——	5.0–6.0	3.0–4.0
		Complete block typical.								
		19¼ min. p.m.	40.0	13.6 (irregular)	——	——	9.7 mm.	——	5.0	3.4
122.2	8	20 min. p.m.	40.0	Ventricular fibrillation.	——	——	——	——	——	——
		20¼ min. p.m.	40.0	One last ventricular wave.	——	——	6.5 mm.	——	5.0	——
		20½ min. p.m.	38.4	Not active.	——	——	——	——	——	——
		Auricles still beating presumably 20 minutes after clinical death.								

[6] These measurements were made in millimeters, instead of seconds, as the time-marker was not operating.

ing again for about fifteen seconds, and then the ventricular rate again sank to 58.5 per minute. The auricles began again three quarters of a minute after the time clinical death is inferred to have occurred and took up the pace at about the same rate as that at which the ventricles were already beating. The auricles then continued to beat almost constantly, decreasing slightly in rate, and were still active at a rate of 34 beats a minute in the last curve taken, twenty minutes after the inferred time of clinical death. The ventricles followed the pace of the auricles until twelve minutes after inferred clinical death, when, following a period of delayed conduction, partial block (3 to 2 rhythm) occurred. Complete heart-block occurred six minutes later, the ventricular rate being 18 per minute. This rate gradually decreased and at nineteen minutes after inferred clinical death, the ventricles beat irregularly 13.6 times a minute. One minute later ventricular fibrillation set in (figure 8), after which one ill formed ventricular complex occurred. There was a gradual but not marked shortening of the ventricular systole, as was indicated by the length of the ventricular complex in the electrocardiograms, and the characteristic change in its form occurred (table II).

Case 3.—A child of nine months, who died of pneumococcus meningitis. No tracings were taken until four minutes after clinical death, at which time the heart was beating at a rate of 40 per minute. The electrical complex was at this time abnormal, there being a diphasic P-wave representing the auricular activity, while the T-wave was much exaggerated and almost fused with the R-wave. The conduction (P-R) time gradually lengthened in the record made at this time, from 0.22 to 0.28 of a second. At five minutes post mortem the rate was 28. The P-wave had become negative and very small, while the conduction time had shortened, varying from 0.145 to 0.16. This seems to indicate that the point of origin of stimulus formation had moved to a point in the auricles nearer the atrioventricular junction. The R- and T-waves approached each other. At the end of the record a long pause of over six seconds occurred. At six minutes post mortem two beats occurred at the same rate as before. Both auricular and ventricular complexes were seen. In the latter the R- and T-waves were partly fused. No evidence of cardiac activity was seen in records made during the next few minutes, so in this case cardiac activity ceased six minutes after clinical death, the auricles and ventricles stopping synchronously (table III).

TABLE III.

Case 3. Age 9 months. Pneumococcus Meningitis. Curve 138.

Time.	Rate per minute.	Conduction time in seconds. P-R.	Length of systole in seconds. R-T.	Height of waves in mm.		
				P.	R.	T.
4 min. p.m.	40	0.22–0.28	0.24–0.28	1.3	6.0	5.0
	Diphasic P-wave.					
5 min. p.m.	28	0.145–0.20	0.20–0.27	—	6.0–7.5	3.5–4.0
	P-wave is negative and nearer the ventricular complex than before.					
6 min. p.m.	27	0.16	0.25	—	4.5	—
	Only two beats.					

Case 4.—A man of thirty-seven, who died of lobar pneumonia. The heart was beating at a rate of 136 per minute sixteen and a half hours before death and the electrocardiogram shows that the rhythm was regular except for an occasional auricular extrasystole. The first post-mortem record was not made until four minutes after clinical death, when the heart was beating irregularly and the electrocardiogram showed a very unusual form of arhythmia (figure 9). In the second group a positive P-wave was followed by a small R-wave, the P-R time being 0.15 of a second. Then a negative P-wave occurred, followed by a small R-wave, the P-R time being 0.24 of a second. Then after a long diastole, a large R-wave, which was not preceded by any evidence of auricular activity, occurred. This group was repeated several times. One large R-wave alternated with two small R-waves, the small R-waves alone being preceded by P-waves. Later these were always negative. From six to fourteen minutes post mortem the cardiac rate varied from 43 to 64 a minute in an irregular manner. At first there was no evidence of auricular activity, but at eight minutes post mortem there was a small negative P-wave preceding the R-wave by 0.123 of a second. The P-wave gradually increased in prominence, but remained negative, while the conduction (P-R) time gradually lengthened until it was 0.31 of a second (figure 10). After fourteen minutes post mortem there was no evidence of auricular activity, while the ventricles continued to beat for thirty-five minutes after clinical death. Their rate became gradually slower and

TABLE IV.

Case 4. Age 37 years. Pneumonia. Curve 179.

Number of curve.	Number of figure.	Time.	Rate per minute.	Conduction time in seconds.		Length of ventricular systole in seconds. R to end of T.	Height of waves in mm.		
				P-R.	R-S.		P.	R.	T.
		16 hrs. and 37 min. a.m.	136.4	0.13–0.14	0.075	0.315	2.2	9.5	2.0
179.2	9	4 min. p.m.	78.5	0.15–0.24	0.065-0.08	——	——	9.0–12.5	——
			A very unusual type of arhythmia seen.						
		6 min. p.m.	64.0	Auricles not active.	0.077	0.285	——	7.5	4.0
		8 min. p.m.	43.0	0.123	——	0.250	——	5.5	4.0
			Auricles return but P-wave is very small.						
		9 min. p.m.	46.0	0.24	——	0.230	Negative P-wave.	6.5	3.5
		10 min. p.m.	60.0	0.26	——	0.260	——	6.0	3.5
179.7	10	14 min. p.m.	54.0	0.31	——	0.240	——	6.0	3.0
			Last of auricular activity.			Very long conduction time.			
		15 min. p.m.	40.6	——	——	0.220	——	6.5	3.0
		21 min. p.m.	36.0	——	——	0.240	——	7.5	2.5
		23 min. p.m.	30.0	——	——	0.240	——	8.0	—
		28½ min. p.m.	26.0	——	——	0.240	——	7.0	—
			Ventricles beating irregularly.						
		31½ min. p.m.	——	——	——	0.240	——	8.0	—
		33 min. p.m.	13.8	——	——	0.240	——	7.5	—
		33½ min. p.m.	6.3 seconds between the two beats.						
179.23	11	35 min. p.m.	1 beat only.		——	0.350	——	6.0	—

their activity was irregular after twenty-eight and a half minutes post mortem. Only a single but well defined ventricular complex was seen thirty-five minutes post mortem (figure 11). The characteristic fusion of the R- and T-waves took place gradually, while except for the final ventricular complex, which was abnormally prolonged, no marked change took place in the length of the ventricular systoles. In this case was seen the longest post-mortem cardiac activity that was observed (table IV).

Case 5.—A woman of twenty-five years, who died of pneumonia. No records were made until twelve minutes after clinical death, when the heart was beating 40 times a minute. The electrical complex was at this time abnormal, consisting of a fairly well defined P-wave, a deep Q-wave, and a small R-wave already fused with the T-wave. At thirteen minutes post mortem the rate was 35.6 and the conduction (P-Q) time was delayed (0.225 of a second, figure 12). The rate slowed gradually and the conduction time became slightly more delayed until at sixteen minutes post mortem the rate was 24.4 per minute, while the conduction time was 0.24 of a second. At seventeen minutes a long pause occurred, 13.6 seconds of which were recorded. This pause was followed by a very unusual ventricular complex which followed a notched P-wave after a conduction time of 0.28 of a second. The complex consists of a typical left ventricular, or apical complex followed by a right ventricular, or basal complex (figure 13). That these forms of complexes result from left and right ventricular contractions has been demonstrated by Nicolai[7] and others. This unusual form of complex suggests that first the left and then the right ventricle contracted separately. It was followed by a pause, eight seconds of which were recorded. The cardiac activity was again resumed and the heart continued

TABLE V.

Case 5. Age 25 years. Pneumonia. Curve 149.

Number of curve.	Number of figure.	Time.	Rate per minute.	Conduction time in seconds. P–Q.	Length of ventricular systole in seconds.	Height of waves[8] in mm. T.
		12 min. p.m.	40.0	—	—	—
149.2	12	13 min. p.m.	35.6	0.225	0.31	5.5
		14 min. p.m.	34.0	0.25	0.31	6.0
		15 min. p.m.	30.4	0.24	0.31	6.0
		16 min. p.m.	24.4	0.24	0.33	6.0
149.6	13	17 min. p.m.	Unusual type of complex suggesting hemisystole.			
					0.87	6.5
		18 min. p.m.	30.8	0.24	0.34	5.2
		19 min. p.m.	30.0	0.24	0.33	5.7
		20 min. p.m.	31.0	0.26	0.36	5.0
		21 min. p.m.	24.0	0.28	0.34	5.5
		22 min. p.m.	32.7	0.35	0.36	5.0
			A pause of 22 seconds preceded the foregoing curve.			
		23 min. p.m.	20.0	0.375	0.35	6.0
149.15	14	25½ min. p.m.	After a stoppage of 2½ minutes.			
			33.0	0.42–0.54	0.40	0.35

[7] Nicolai, G. F., *Med. Klin.*, 1912, viii, 322.

[8] P and R not measurable.

to beat slowly but with marked changes of rate and long periods of complete cardiac standstill. The heart finally ceased beating twenty-five and a half minutes after clinical death. Just before the final record was obtained, a pause for two and a half minutes occurred. After this pause both auricles and ventricles again became active at a rate of 33 beats a minute. The conduction time in this record (figure 14) gradually lengthened until the ventricles failed, the auricles continuing for a short time at the same rate. The conduction time, which was already distinctly delayed at nineteen minutes post mortem, further lengthened and at twenty-three minutes post mortem it was 0.375 of a second. In the final record it increased beat by beat from 0.42 to 0.54 of a second. The form of the ventricular complex changed very little in this case, except that the duration of ventricular systole gradually lengthened (table V).

Case 6.—A woman of thirty-seven years, with pneumonia. Before death, electrocardiograms showed a heart rate of 156, a conduction time of 0.12, and ventricular systole of 0.25 of a second duration (figure 15). The first record obtained one minute after clinical death showed that the heart was beating regularly at a rate of 72 per minute with practically no change in conduction time (figure 16). At two minutes post mortem there was complete dissociation of auricles and ventricles, and groups of ventricular contractions seperated by long

TABLE VI.

Case 6. Age 37 years. Pneumonia. Curve 195.

Number of curve.	Number of figure.	Time.	Rate per minute.		Conduction time in seconds.		Length of ventricular systole in seconds. R to end of T.	Height of waves in mm.		
			Auricular.	Ventricular.	P-R.	R-S.		P.	R.	T.
195.2	15	52 min. a.m.	156.0	156.0	0.120	0.09	0.25	1.5–2.5	11.5–12.3	2–3
		14 min. a.m.	155.6	155.6	——	——	——	—	——	——
195.5	16	1 min. p.m.	72.0	72.0	0.128	0.08	0.265	1.5	11.0	3.0
		1½ min. p.m.	65.4	65.4	0.115	0.08	0.30	1.0	11.0	3.5
195.7	17	2 min. p.m.	Irregular. Occasional auricular activity. Last seen in this curve. Ventricles beating irregularly. Long pauses seen.							
195.8	18	2½ min. p.m.	——	168.0	——	0.08–0.094	0.30	1.5	9.5–10.3	2.5
			Ventricular tachycardia.							
		3½ min. p.m.	——	138.0	——	——	——	—	8.0– 8.5	2–3
			in small group. Ventricles beating in groups and isolated beats without auricles. Long pauses of several seconds occurred.							
		4 min. p.m.	——	47.4	——	0.10	——	—	7.0	2.0
			Ventricular rhythm established. Slight arhythmia.							
195.12	19	4½ min. p.m.	——							
			Ventricular fibrillation.							
			31.5							
		5 min. p.m.	Ventricles at first irregular and then rhythmic. Marked change in complex.						0.33–0.22	5.5
195.14	20	5½ min. p.m.		31.9					0.23	6.0
		6 min. p.m.	No beats.							
		6½ min. p.m.		27.0					0.23	6.0
		7 min. p.m.	One beat.							
				24.0						
		7½ min. p.m.	Two beats only.						0.20	5.3

pauses were seen. Auricular activity ceased altogether at this time (figure 17). Two and a half minutes post mortem a group of ventricular complexes occurred at a rate of 168 per minute, showing apparently true ventricular tachycardia (figure 18), while four minutes post mortem the ventricular rhythm established itself at a rate of 47.4 with but slight arhythmia. Four and a half minutes post mortem ventricular fibrillation of short duration set in (figure 19), and was followed for about one minute by a period of distinct arhythmia, but at a fairly slow rate. Five and a half minutes post mortem the ventricular rhythm again became regularly established at a rate of 32 per minute (figure 20). The ventricular complexes were markedly altered and showed the characteristic form seen in nearly all the cases observed just before cessation of ventricular activity. The rate then became slower and long periods without cardiac activity occurred. Two last beats, occurring at a rate of 24 per minute, were seen seven and a half minutes after clinical death. This case showed at first a lengthening and then finally a distinct shortening of the length of ventricular systole, which reached 0.33 of a second at five minutes post mortem and then shortened to 0.20 of a second in the last record (table VI).

Case 7.—A woman of thirty-four years, who died also of pneumonia. The electrocardiograms from this case indicated that the auricles had ceased to beat one minute before clinical death, while the ventricles continued for seventeen minutes after the patient ceased to breathe. The ventricles showed marked irregularities in rate, but two minutes post mortem they were beating regularly at 45.8 per minute. They beat at a rate of 107 times a minute thirteen minutes post mortem, showing a bigeminal type of arhythmia. They beat regularly at a rate of 47, sixteen minutes post mortem, but no evidence of cardiac activity was seen seventeen minutes post mortem. Because the records were not satisfactory, a more detailed analysis is not possible (table VII).

TABLE VII.

Case 7. Age 34 years. Pneumonia. Curve 228.

Time.	Rate.		
1 hr., 6 min. a.m.	120.0	Auricles active.	Ventricles regular.
1 min. a.m.	57.1	Auricles not active.	Ventricles regular.
½ min. a.m.	51.7		Ventricles regular.
2 min. p.m.	45.8		Ventricles regular.
4 min. p.m.	60.0		Ventricles nearly regular.
The type of ventricular complex changed here.			
11 min. p.m.	60.0	Ventricles beating irregularly.	
13 min. p.m.	107.0	Ventricles beating bigeminally.	
14 min. p.m.	71.0	Ventricles beating irregularly.	
15 min. p.m.	53.6	Ventricles beating regularly.	
16 min. p.m.	47.0	Ventricles beating regularly.	
No movement of string after 17 minutes post mortem.			

SUMMARY.

In four of the seven cases the ventricles remained active from one and a half to eighteen minutes after the electrocardiograms

failed to show evidence of auricular activity. In two cases the auricles outlasted the ventricles and in one case only did the auricles and ventricles stop apparently at the same time. Complete dissociation occurred three times. Some delay in the conduction time was seen in five of the seven cases. In two cases the auricles ceased to beat before evidence of impaired conduction appeared. There was always marked slowing; the slowest independent ventricular rates varied from 13.6 to 47.0. The slowest rates at which the auricles beat regularly varied from 20 to 65 per minute. There was never evidence of auricular fibrillation, although in two cases the electrocardiograms give fairly conclusive evidence that ventricular fibrillation occurred. The ventricles reëstablished a regular rhythm after a short period of ventricular fibrillation in one case, while in the other but one ventricular contraction occurred after the appearance of fibrillation.

Characteristic changes in the ventricular complex of the electrocardiograms occurred in all the records. They consisted of a gradual fusion of the R- and T-waves, forming, when the fusion was complete, a large rounded or peaked wave. In some cases the identity of the two waves was not entirely lost. In spite of the marked change in shape of the ventricular complexes, there was often but little change in their duration. In some cases the ventricular systole was shortened at the end, while in others it was prolonged. The change in the form of the ventricular electrical complex indicates that the course of the stimulus and the manner of the contraction of the muscle were abnormal. The fact that the R-wave became gradually prolonged suggests that the conduction of the stimulus through the ventricular walls became delayed as the heart died. The fact that after death there is a continuation of cardiac muscular activity sufficient to cause a difference in electrical potential between the two sides of the body does not necessarily mean that a ventricular systole in the sense of muscular shortening takes place. It has been observed experimentally that well defined electrical complexes may be caused by cardiac activity which cannot be seen or recorded graphically. As the duration of the ventricular complexes characteristic of the dying heart usually does not differ markedly from the duration of the complexes before clinical death, it seems probable

that the entire musculature of the ventricles participates in the contraction; as definite shortening, or at least a marked change in duration, would be expected if only a part of the ventricular musculature participated in the activity which produced the complex.

CONCLUSIONS.

In acute infectious diseases cardiac activity sufficient to give a definite record with the electrocardiograph may continue in the human heart for some minutes after clinical death has occurred. In the seven cases described the cardiac activity continued from six to thirty-five minutes after all the usual clinical signs of death had occurred. In four cases the ventricular outlasted the auricular activity; in two cases this was reversed; and in one case the two parts of the heart seemed to cease synchronously. Marked slowing of the rate of cardiac activity always occurred and there was usually distinct delay in the conduction time between auricles and ventricles. Complete dissociation was seen in three cases. Ventricular fibrillation occurred in two cases, in one of which the ventricles again established a regular rhythm. Evidence of auricular fibrillation was never seen. Characteristic changes in the ventricular electrical complex occurred in all cases. They consisted of a decrease in the size of the R-wave and an increase in the size of the T-wave, and a tendency to a fusion of these waves. There was usually but little change in the duration of the ventricular complexes as the cardiac activity gradually ceased. The foregoing observations indicate that when death occurs from an acute infectious disease there is no one point in the human heart which may be considered as the "ultimum moriens."

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EXPLANATION OF PLATES.

PLATE 34.

FIG. 1. Curve 134.3. Case 1.
FIG. 2. Curve 134.11. Case 1.
FIG. 3. Curve 134.12. Case 1.
FIG. 4. Curve 134.14. Case 1.

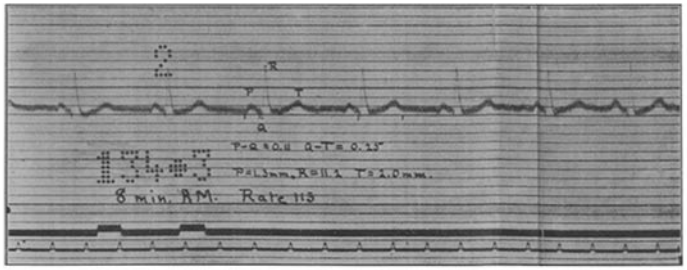

FIG. 1.

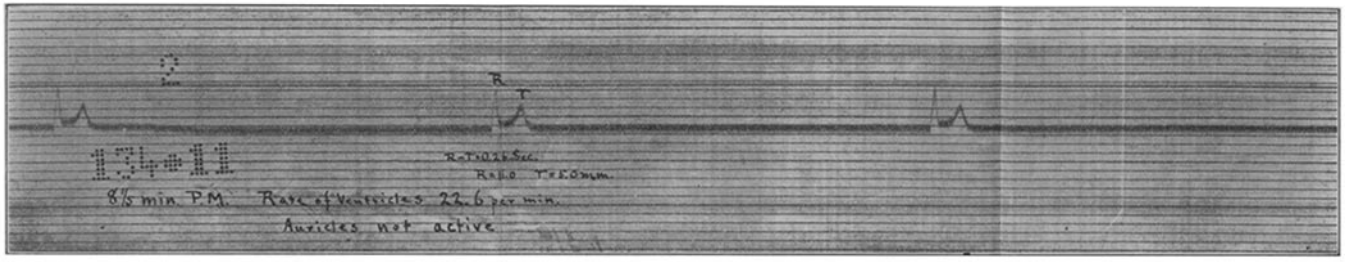

FIG. 2.

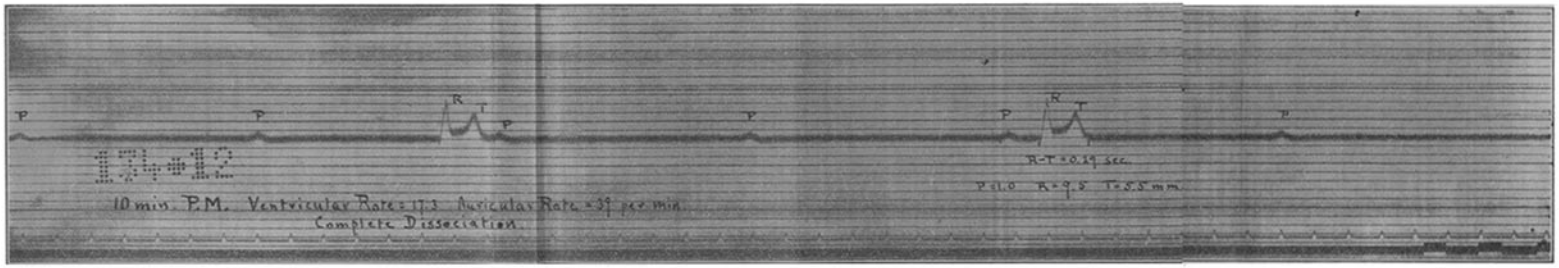

FIG. 3.

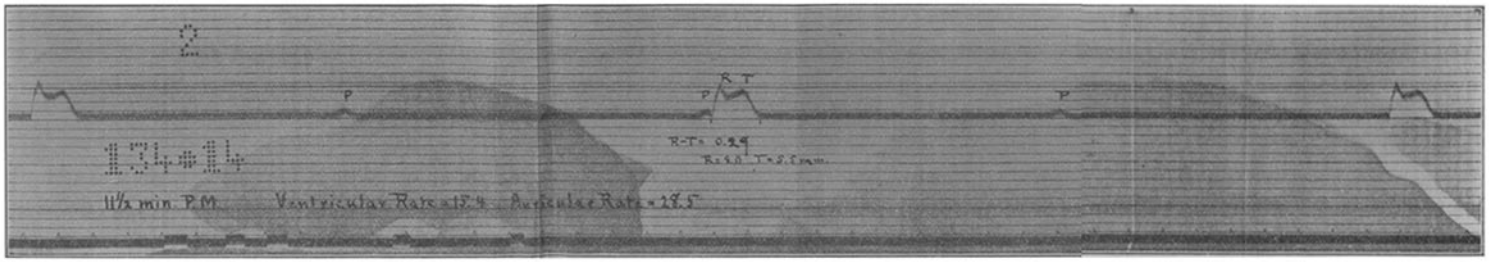

FIG. 4.

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PLATE 35.

FIG. 5. Curve 134.22. Case 1.
FIG. 6. Curve 119.7. Case 2.
FIG. 7. Curve 121.4. Case 2

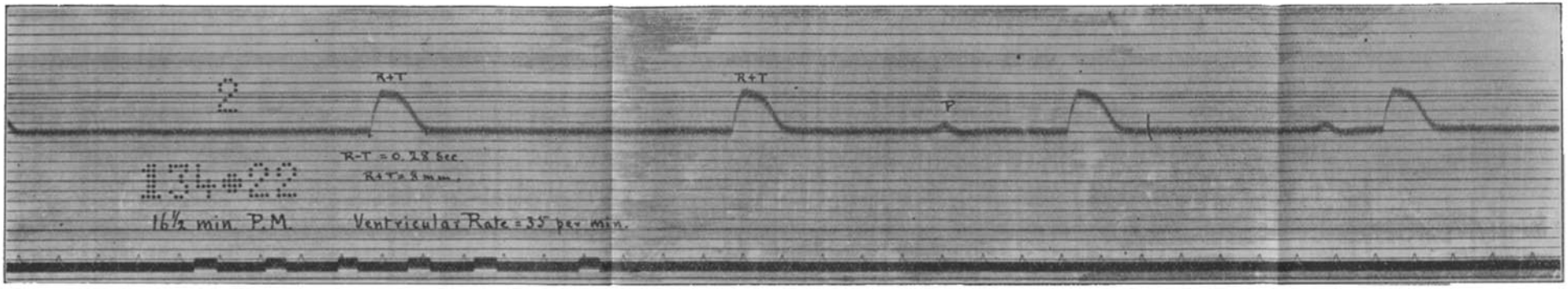

FIG. 5.

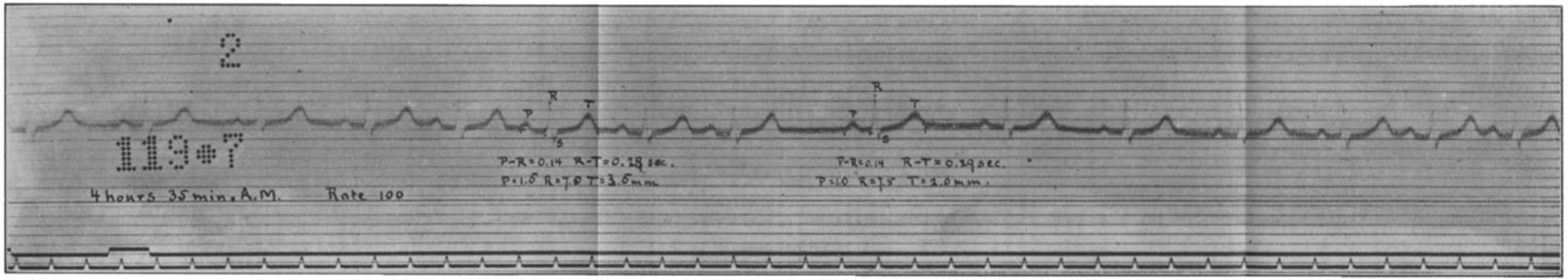

FIG. 6.

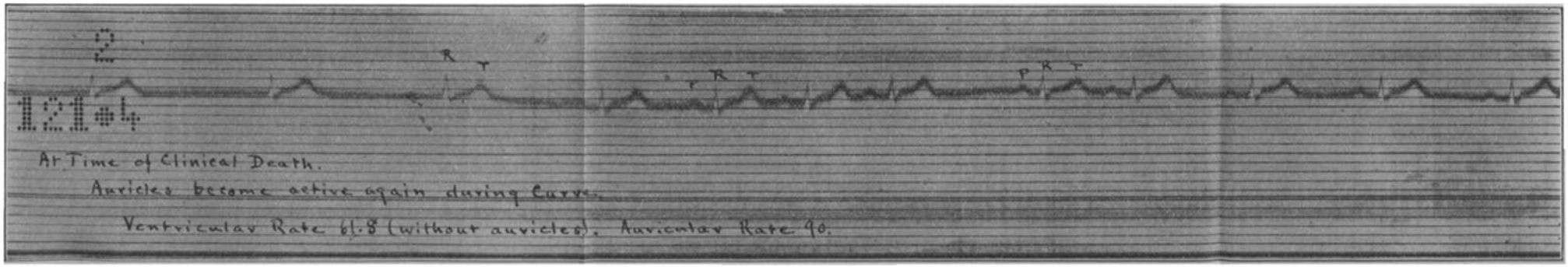

FIG. 7.

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PLATE 36.

FIG. 8. Curve 122.2. Case 2.
FIG. 9. Curve 179.2. Case 4.
FIG. 10. Curve 179.7. Case 4.
FIG. 11. Curve 179.23. Case 4.

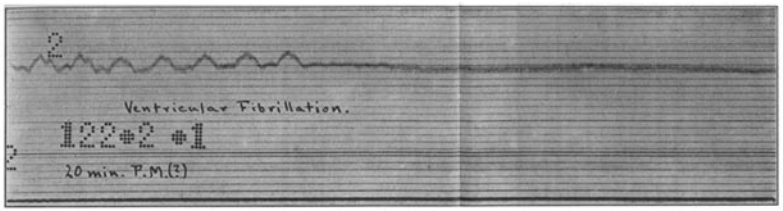

FIG. 8

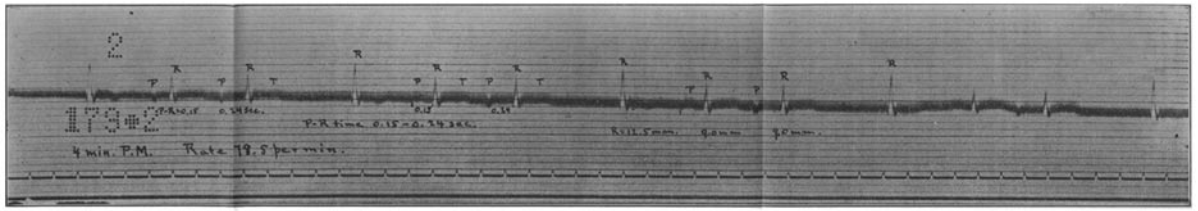

FIG. 9.

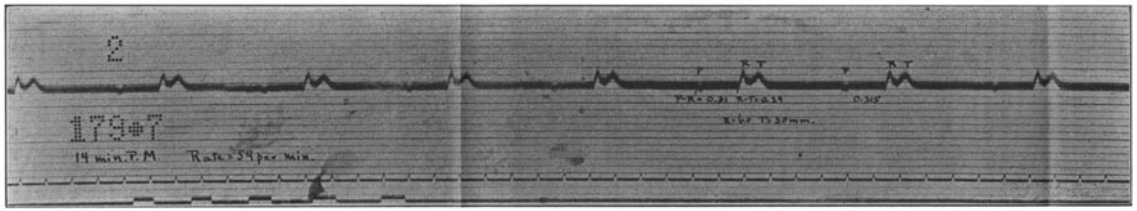

FIG. 10.

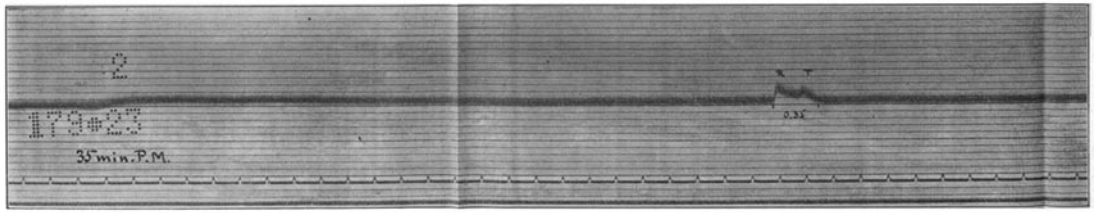

FIG. 11.

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PLATE 37.

FIG. 12. Curve 149.2. Case 5.
FIG. 13. Curve 149.6. Case 5.
FIG. 14. Curve 149.15. Case 5.
FIG. 15. Curve 195.2. Case 6.
FIG. 16. Curve 195.5. Case 6.
FIG. 17. Curve 195.7. Case 6.

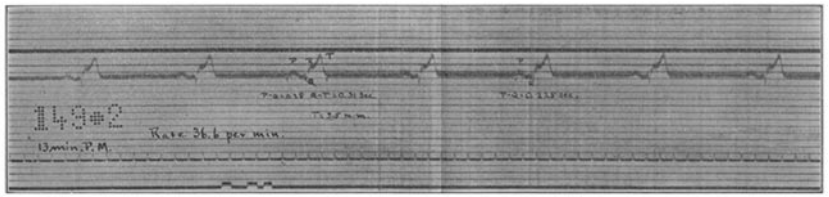

FIG. 12.

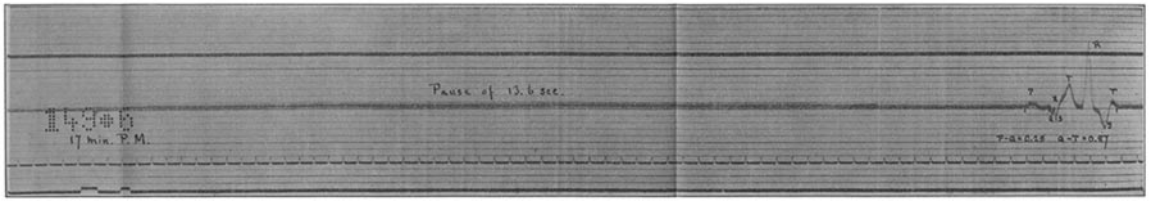

FIG. 13.

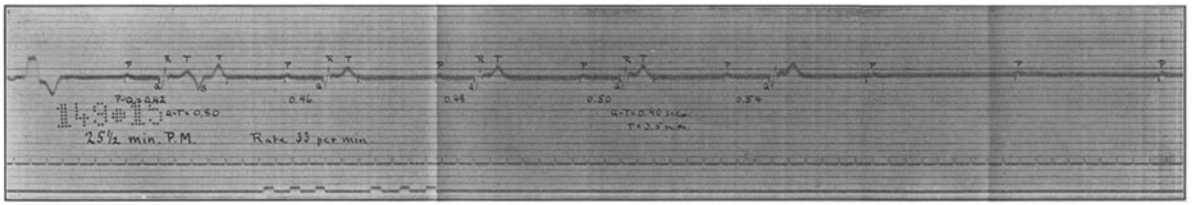

FIG. 14.

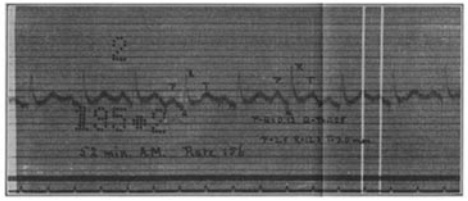

FIG. 15.

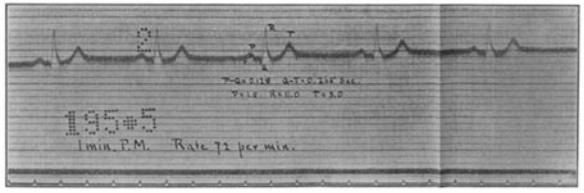

FIG. 16.

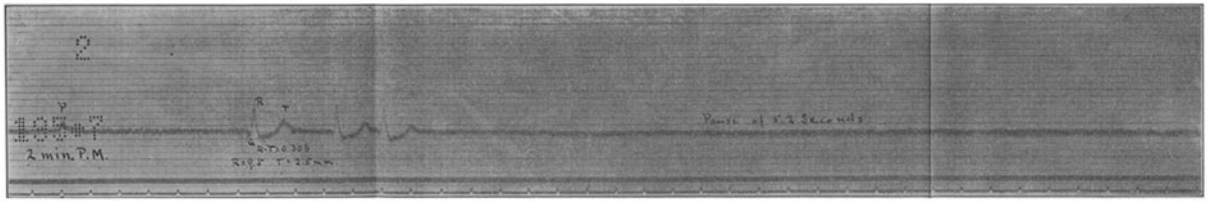

FIG. 17.

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PLATE 38.

FIG. 18. Curve 195.8. Case 6.
FIG. 19. Curve 195.12. Case 6.
FIG. 20. Curve 195.14. Case 6.

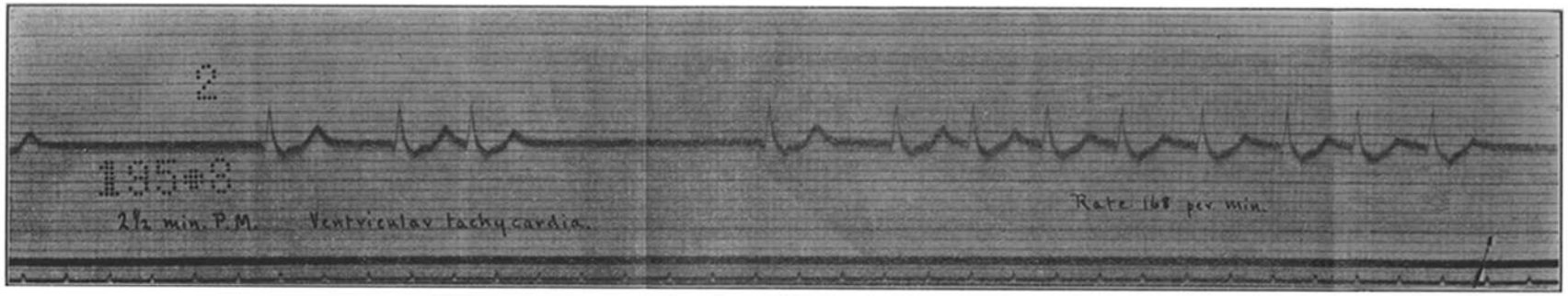

FIG. 18.

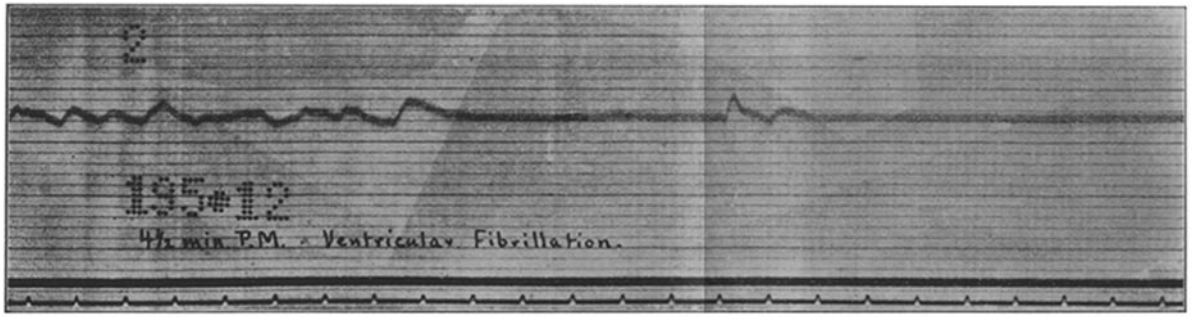

FIG. 19.

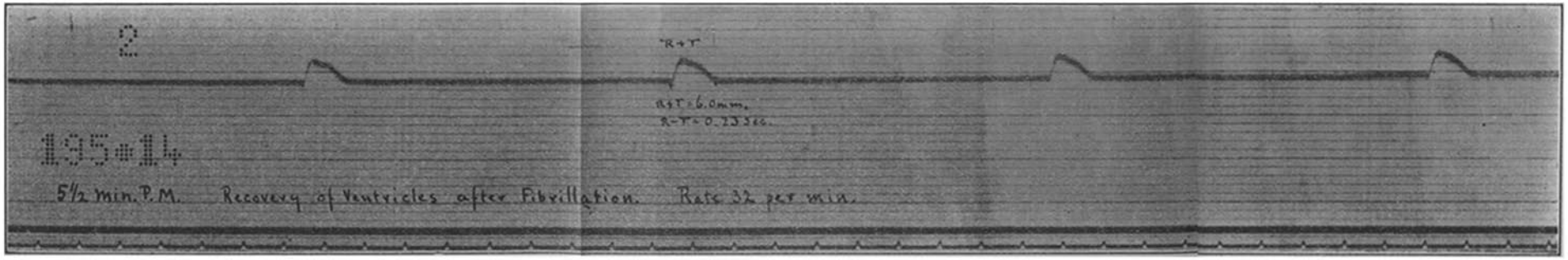

FIG. 20.