

**STUDIES OF THE CIRCULATION IN THE PRESENCE OF
ABNORMAL CARDIAC RHYTHMS. OBSERVATIONS RELATING
TO (PART I) RHYTHMS ASSOCIATED WITH RAPID VEN-
TRICULAR RATE AND TO (PART II) RHYTHMS ASSOCIATED
WITH SLOW VENTRICULAR RATE**

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STUDIES OF THE CIRCULATION IN THE PRESENCE OF ABNORMAL CARDIAC RHYTHMS.¹ OBSERVATIONS RELATING TO (PART I) RHYTHMS ASSOCIATED WITH RAPID VENTRICULAR RATE AND TO (PART II) RHYTHMS ASSOCIATED WITH SLOW VENTRICULAR RATE¹

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Since the function of the heart is that of a pump it is of interest both to physiologists as well as to those working in the clinic to know how it manages not only when it is beating regularly in a normal fashion but also how it performs this function when it is the subject of abnormalities in its rhythm.

Stewart and his coworkers (1 to 6 inclusive) have reported the effects of artificially induced regular and irregular tachycardia on the blood flow and cardiac output of the heart of dogs. It appeared from these observations that the heart was less effective as a pump during these abnormal rhythms, since its minute volume output of blood was diminished. Dilatation of the heart was also observed.

There has been no systematic study of the effect of cardiac irregularity on the volume output of blood from the heart in human beings. Observations relating to several of the irregularities are included in cases recorded by Starr and his associates (7, 8). Kerkof and Baumann (9, 10) have made observations on 4 patients suffering from mitral stenosis, first when they exhibited auricular fibrillation and later again after restoration of normal sinus rhythm.

It was to contribute to our knowledge of the effects of abnormal rhythms on the circulation that studies were undertaken in patients exhibiting the irregularities of the heart that are most frequently encountered. Measurements were made

of cardiac output, cardiac size, arm to tongue circulation time, venous blood pressure, vital capacity, heart rate, and arterial blood pressure. Electrocardiograms were taken to record the prevailing rhythm.

METHODS

All observations were made in the morning while the patients were in a basal metabolic state. Measurements of the cardiac output were made by the acetylene method, three samples of gas being taken as first recommended by Grollman (11), and by Grollman, Friedman, Clark, and Harrison (12). During this measurement the patients were sitting in a steamer chair (angle 135 degrees) with the legs extended. They were trained beforehand to carry out the procedures. While the patient was at rest, the cardiac rate was counted at intervals of 5 minutes. At the end of one-half hour the acetylene-air-oxygen mixture was rebreathed. Three samples of gas were taken during each rebreathing period for estimation of the arteriovenous oxygen difference. The first sample was taken after rebreathing 10 to 12 times in 20 seconds, the second after 2 to 3 breaths more, and the third after 2 to 3 additional breaths. All three samples were usually obtained before the end of 30 seconds. Samples were taken during expiration. Two to three periods of rebreathing were carried out. Shortly afterward, the oxygen consumption was measured with a Benedict-Roth spirometer. After a short pause, the vital capacity was measured, and height and weight recorded. Then the patient rested again, lying down. In succession, sufficient time being allowed between each procedure for the patient to return to a basal metabolic state, an electrocardiogram was taken, the arm to tongue circulation time recorded, the venous pressure estimated, and the blood pressure measured; finally, the patient still in a basal state, an x-ray photograph of the heart was made at a distance of two meters.

The arm to tongue circulation time was estimated by the use of decholin: 5 cc. of a 20 per cent solution were injected rapidly (1 to 2 seconds) through an 18-gauge needle into an antecubital vein while the patient was lying quietly in the supine position. This was repeated in

¹ An abstract of certain of these studies was read before the American Heart Association, Atlantic City, June 11, 1935, and also at the Scientific Meeting of the Committee on Cardiac Clinics of the New York Heart Association, at the New York Academy of Medicine, April 23, 1935.

one and one-half minutes after the response to the first test had been elicited. The time was recorded from the beginning of the injection until the patient perceived the bitter taste. The injection time was also recorded, but since the response may come with a minimal amount of the drug, the time which we have used was taken from the start, rather than from the conclusion of the injection.

The venous pressure was measured by the direct method (13), using a large antecubital vein, the arm being placed on a level with the right auricle. The apparatus consisted of an L-tube of glass attached to a three-way stopcock, a syringe, and an 18-gauge needle. The apparatus was filled with a solution of sterile normal saline, a venepuncture performed, and the direct pressure readings recorded. Normal pressures with this apparatus range from 4.0 to 9.0 cm. of saline. The antecubital vein of one arm was reserved for the injection of decholin and of the other arm for the measurement of venous pressure. In subsequent measurements the vein was entered at the site first punctured.

X-ray photographs of the heart were taken with the patient in the standing position, in full inspiration, at a distance of two meters.² Measurements of the cardiac area were carried out by the technique of Levy (14) and estimations of volume were made as recommended by Bardeen (15). The volumes recorded in Table I have not been multiplied by the constant which is in Bardeen's formula. This was done in order to make our observations comparable to those of Starr and his coworkers (7, 8). The patients assumed as nearly as possible exactly the same position for each observation in order to make comparison with subsequent ones of this patient possible and to assure uniformity from this point of view. In addition, each procedure in the observation was carried out by the same investigator.

Observations were made first during the abnormal rhythm and later again after restoration of normal sinus rhythm.³ Most of the patients exhibited no clinical evidence of congestive heart failure (see text), nor were they anemic (16).

PART I. OBSERVATIONS RELATING TO RHYTHMS ASSOCIATED WITH RAPID VENTRICULAR RATE

OBSERVATIONS RELATING TO AURICULAR FIBRILLATION

In Table I are reported seven patients exhibiting auricular fibrillation. In them, observations were made first during auricular fibrillation and

later again after restoration of normal sinus rhythm.

R. D. (*N. Y. H. No. 48714*), a white man, aged 32 years, was admitted to the hospital on December 6, 1933, complaining of sudden onset of irregularity of the heart beat the day before while "cranking an automobile." The first attack occurred 11 years before when he was 21 years old. He had experienced approximately one attack every 6 months since then. Each attack was of 5 to 6 days' duration. Neither digitalis nor quinidine had been effective in terminating an attack. He had never exhibited manifestations of rheumatic fever. When seen 24 hours after the onset of this attack he did not appear acutely ill; there were no signs of congestive heart failure. The heart was not enlarged. The diagnoses were: *A*, unknown; *B*, mitral stenosis; *C*, paroxysmal auricular fibrillation.⁴ On December 8, 1933, when auricular fibrillation was present, measurements of the circulation were made; these were repeated on December 11th, 2 days after restoration of normal sinus mechanism occurred spontaneously (Table I, Figure 1). In October 1936 we had occasion to make observations of this patient during still another attack of paroxysmal auricular fibrillation. Following discharge from the hospital in 1933 he experienced attacks of paroxysmal auricular fibrillation at 6 to 7-month intervals; during the last year, however, the frequency had increased to 3 attacks. This last attack began on October 25, 1936. Because of its persistence the patient was admitted to the hospital on October 27, 1936. The physical signs were essentially the same as on the first admission; as before there were no signs of congestive heart failure, and there were signs of early mitral stenosis. Studies of the circulation were made on October 28, 1936 in the presence of auricular fibrillation and again on October 30, 1936, 24 hours after reversion to normal sinus mechanism under quinidine therapy (Table I, Figure 1). Measurements of the circulation during normal sinus rhythm in 1934 were approximately the same as two years later in 1936 during this rhythm, and no significant change in the size of the heart had occurred during this interval.

J. R. (*N. Y. H. No. 52524*), a white man, aged 21 years, was admitted to the hospital on January 17, 1934, complaining of rapid, irregular, forcible beating of his heart of 16 hours' duration. He had never experienced a similar attack. History of rheumatic infection was not elicited. Examination revealed no evidence of congestive heart failure. After exercise a presystolic impurity of the first sound was heard at the apex; this was not sufficient, however, to warrant the diagnosis of valvular disease. The heart was not enlarged. The diagnosis was

² The authors are deeply indebted to the X-ray Department of the New York Hospital for their cooperation in this investigation.

³ Deviation from this routine is indicated in the text and tables.

⁴ The diagnoses in this paper conform to the nomenclature for cardiac diagnosis recommended by the Heart Committee of the New York Tuberculosis and Health Association "Criteria for the Classification and Diagnosis of Heart Disease." New York Tuberculosis and Health Association, New York, 1929, 2d ed.

TABLE I
Data in patients exhibiting auricular fibrillation, auricular flutter, and paroxysmal tachycardia

Case; Hospital number; Age	Date	Body surface area	Oxygen consumption	Basal metabolic rate	Arterio-venous oxygen difference	Cardiac output	Cardiac output per beat	Cardiac area	Cardiac volume*	Blood pressure	Left atrial work	Circulation time	Venous pressure	Vital capacity	Rhythm †	Digitalis ‡	Other drugs	Evidence of congestive heart failure §					Red blood count	Hemoglobin		
																		Dyspnea	Cyanosis	Rales	Liver	Edema				
		sq. m.	cc. per minute	per cent	cc.	liters per minute	cc.	sq. cm.	cc.	mm. Hg	grams per meter per beat	seconds	cm.	cc.		grams	grams	Dyspnea	Cyanosis	Rales	Liver	Edema	milli-grams	per cent		
AURICULAR FIBRILLATION																										
Auricular fibrillation → Normal rhythm without digitalis without quinidine (Figure 1)																										
R. D. 4871A ♂ 32 yrs.	Dec. 8, 1933 Dec. 11, 1933	1.81 1.82	255 237	+3 -5	88.3 61.4	2.89 3.86	1.60 2.12	100 66	28.9 58.5	98/48 ¹ 105/60	28.7 65.6		4,700 4,700		A.F. N.R.	0 0	0 0	0 0	0 0	0 0	0 0	0 0	0 0	6.1	105	
J. R. 4629A ♂ 21 yrs.	Jan. 18, 1934 Jan. 20, 1934 Jan. 30, 1934	2.02 2.01 2.00	282 261 285	0 -5 +4	103.4 63.0 64.7	2.73 4.14 4.40	1.36 2.06 2.20	85 83 85	32.1 49.8 51.7	132/80 120/84 138/76	46.3 69.1 74.5		5,250 5,350 5,270		A.F. N.R. N.R.	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	5.3	100	
Auricular fibrillation → Normal rhythm with quinidine without digitalis (Figure 1)																										
M. R. 4971B ♀ 26 yrs.	April 11, 1934 April 15, 1934 April 16, 1934 April 17, 1934 April 18, 1934 April 20, 1934	1.52 1.50 1.50 1.46 1.50 1.52	191 172 176 166 184 180	-2 -10 -7 -13 -13 -5	96.2 71.1 59.6 69.0 71.3 68.2	1.98 2.42 1.96 1.60 2.30 1.83	1.30 1.60 1.31 1.60 1.53 1.20	86 66 79 72 68 106	23.0 36.6 24.6 33.6 33.8 17.2	98/60 ¹ 106/76 98/60 ¹ 106/84 95/65 98/60 ¹	24.7 53.3 26.6 43.2 36.8 18.5		2,100 2,430 2,410 2,540 2,475 2,310		A.F. N.R. A.F. N.R. N.R. A.F.	0 0 0 0 0 0	0 0 0 0 0 0	0 0 0 0 0 0	0 0 0 0 0 0	0 0 0 0 0 0	0 0 0 0 0 0	0 0 0 0 0 0	4.2	90		
R. D. 4871A ♂ 34 yrs.	Oct. 28, 1936 Oct. 30, 1936	1.80 1.80	249 234	+1 +5	98.6 61.8	2.53 3.79	1.40 2.10	90 70	28.0 54.0	108/70 102/70	33.9 63.2	19.9 12.7	8.3 6.9	4,600 4,600		A.F. N.R.	0 0	0 0	0 0	0 0	0 0	0 0	0 0	0 0	0 0	0 0
Auricular fibrillation with digitalis with quinidine → Normal rhythm (Figure 1)																										
C. C. 76892 ♀ 44 yrs.	Oct. 25, 1934 Oct. 27, 1934 Nov. 8, 1934	1.47 1.46 1.44	218 189 212	+22 +6 +20	125.6 89.3 95.2	1.74 2.12 2.14	1.18 1.46 1.49	142 92 100	12.2 22.0 21.4	145/100 130/70 160/100	20.7 31.3 37.3	17.0 10.0 10.0	1,900 2,100 2,000		A.F. A.F. N.R.	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	5.0	100	
Auricular fibrillation with digitalis → Normal rhythm (Figure 1)																										
F. K. 9026A ♂ 61 yrs.	Feb. 28, 1935 Mar. 2, 1935 Mar. 4, 1935 Mar. 6, 1935	1.99 1.97 1.94 1.94	240 240 232 234	-3 -2 -4 -3	63.1 74.4 60.2	2.89 3.23 3.89	1.45 1.64 2.00	119 80 72	24.3 35.3 54.0	(170) 160/110 (140) 130/76 170/98 160/80 ¹	44.6 52.0 17.1 91.8	20.1 17.9 17.1 16.0	3,200 2,750 3,000 3,000		A.F. A.F. N.R.	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	5.3	100		
Auricular fibrillation with digitalis with quinidine → Normal rhythm (Figure 1)																										
P. B. 57869 ♂ 55 yrs.	April 13, 1934 April 20, 1934	2.05 2.07	243 247	-5 -5	107.6 70.3	2.26 3.51	1.10 1.70	98 66	25.0 53.2	129/80 ¹ 139/80	32.2 78.9		3,900 3,900		A.F. N.R.	+	+	0 0	0 0	0 0	0 0	0 0	0 0	6.6 grams in 6 days	5.7	108

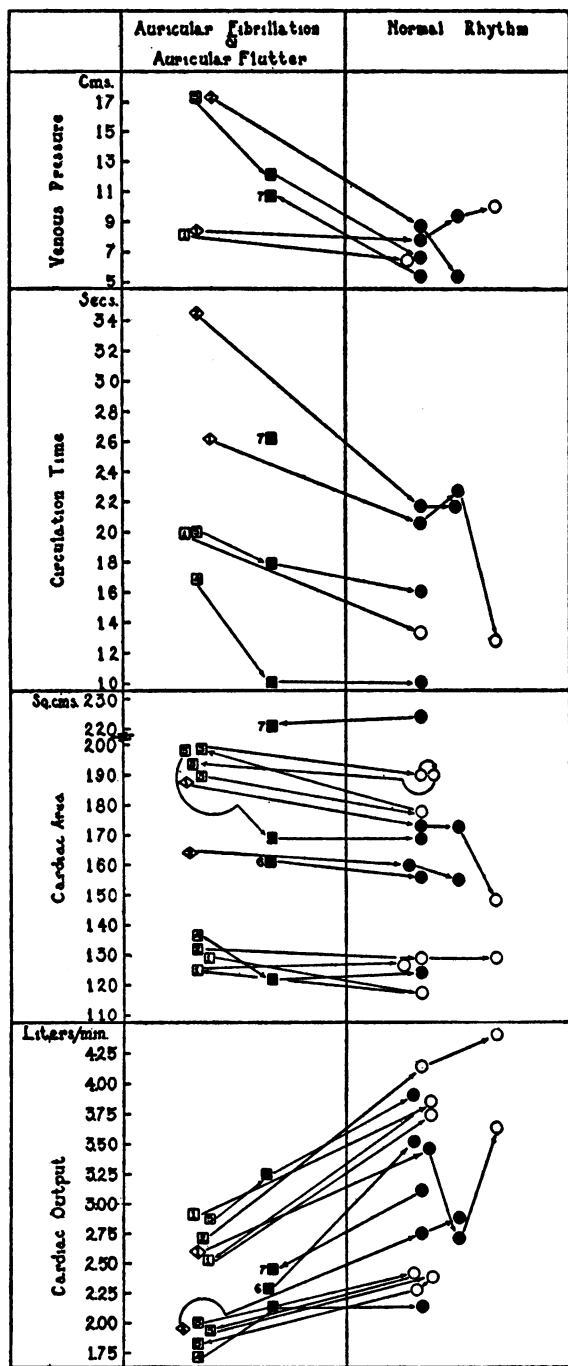


FIG. 1. DATA RELATING TO STUDIES OF THE CIRCULATION IN PATIENTS EXHIBITING AURICULAR FIBRILLATION AND AURICULAR FLUTTER

Observations were made during the irregularity and after restoration of the normal sinus mechanism. In this figure as well as in Figure 3 square and diamond symbols refer to patients exhibiting auricular fibrillation and auricular flutter respectively. The numerals 1 to 7

paroxysmal auricular fibrillation, etiology unknown. Observations were made on January 18, 1934, while auricular fibrillation was present (Table I, Figure 1) and again on January 20, 1934, 18 hours after spontaneous reversion (3 p.m., January 19) to normal sinus rhythm had occurred. Ten days later, normal rhythm still persisting, observations were again recorded (January 30, 1934).

M. R. (N. Y. H. No. 19718), a white female, aged 25 years, was admitted to the hospital on April 10, 1934, because of the onset 2 days before of irregularity of her heart which was associated with dyspnea and followed by weakness, anorexia, and nausea. She suffered rheumatic polyarthritis at 7 years of age with cardiac involvement, and one year later an attack of chorea. She experienced a normal pregnancy when 23 years of age without cardiac distress. On admission to the hospital, when auricular fibrillation had been present 2 days, she revealed at rest no evidence of circulatory insufficiency except enlargement of the liver. The diagnoses were: A, rheumatic heart disease; B, mitral stenosis and insufficiency, cardiac enlargement; C, paroxysmal auricular fibrillation. Observations were made on April 11, 1934, when auricular fibrillation was present (Table I, Figure 1). On April 15, 1934 when normal rhythm had been restored following the administration of quinidine (total amount 3.0 grams) observations were repeated. On reversion to auricular fibrillation on April 16, 1934, studies were made again. There was occasion to make several observations of this patient when reversion from auricular fibrillation to normal sinus rhythm occurred and each time the cardiac output was found to be less and the heart larger during auricular fibrillation than during normal sinus rhythm (Table I, Figure 1).

C. C. (N. Y. H. No. 75892), a white female, aged 44 years was admitted to the hospital October 3, 1934. There was no history of rheumatic fever. The onset of auricular fibrillation had probably occurred on September 17th and was associated with nausea, fatigue, and breathlessness. One week later she consulted a physician because of increase in these symptoms, at which time the presence of valvular heart disease was discovered. Examination at the time of admission revealed that auricular fibrillation was present and that there were signs of mitral stenosis and insufficiency. There was cyanosis and slight enlargement of the liver. The diagnoses were: A, unknown; B, mitral stenosis and insufficiency, cardiac enlargement; C, paroxysmal auricular fibrillation. On October 25, when the patient was not under the influence of digitalis and the ventricular rate was 142 per minute, studies of the circulation were made (Table I, Figure 1). On October 26th, the patient was given digitalis 1.8

inclusive in the square symbols refer to Cases R. D., J. R., M. R., C. C., F. K., P. B., and W. H., respectively, and numerals 1 to 4 inclusive in the diamond symbols refer to Cases H. B., G. G., A. G., and A. G1., respectively in Table I. Circles indicate normal sinus mechanism. Closed and open symbols designate that the patient had and had not been given digitalis respectively. Arrows indicate the change in rhythm.

grams⁵ in 24 hours. On October 27th when the heart rate had decreased to 92 per minute, studies were repeated. Maintenance doses of digitalis, 0.2 gram, were now given daily and the use of quinidine was instituted. On the 10th day of its administration (total given 9.7 grams), reversion to normal sinus rhythm occurred. At this time, November 8, 1934, measurements of the circulation were repeated, the patient still being under the influence of digitalis. When auricular fibrillation recurred one week later, no further effort was made to restore the rhythm to normal sinus mechanism.

F. K. (*N. Y. H. No. 90264*), a white male, aged 61 years, was admitted to the hospital February 27, 1935. A history of rheumatic fever was not secured. Syphilitic infection was denied. He had been well until the onset of the present illness. Five days before admission he began to expectorate frothy white sputum associated with hoarseness and wheezing respirations. Three days before admission, he experienced a sense of suffocation for 5 minutes following which he became dyspneic on exertion. On admission dyspnea, cyanosis, basal pulmonary râles, and slight edema were evidence of congestive heart failure. Auricular fibrillation with rapid ventricular rate was present. The heart was enlarged and x-ray examination revealed aneurysm of the ascending aorta. The Wassermann reaction of the blood was positive. The diagnoses were: *A*, lues; *B*, aneurysm of the ascending aorta, cardiac enlargement; *C*, paroxysmal auricular fibrillation. On February 28, 1935, when the ventricular rate was 119 per minute, observations were made (Table I, Figure 1). The next day digitalis 2.0 grams was given. The day following (March 2, 1935), when the ventricular rate was 90 per minute, observations were repeated. On March 4, 1935, spontaneous reversion to normal sinus rhythm occurred and on March 6, 1935, the rhythm being normal and the patient still under the influence of digitalis, studies were carried out again. On March 7, auricular fibrillation recurred and persisted.

P. B. (*N. Y. H. No. 57869*), a white male, aged 55 years, was admitted to the hospital on April 10, 1934, because of urethral stricture. History of rheumatic infection was not obtained. For 10 years he had experienced attacks of irregularity of the heart associated with forceful heart beat and sensation of palpitation. The attacks were sudden in onset and in offset. During passage of a urethral sound the patient complained of "fluttering" of the heart. Auricular fibrillation was found to be present. The heart was slightly enlarged; there was no valvular defect. The diagnoses were: *A*, arteriosclerotic heart disease; *B*, cardiac enlargement; *C*, paroxysmal auricular fibrillation. He was given digitalis. On April 13, 1934, when the ventricular rate was

98 per minute, and there were no signs of congestive heart failure, observations were made (Table I, Figure 1). On giving quinidine, reversion to normal sinus rhythm occurred, and on April 20th, 24 hours afterward, studies of the circulation were repeated.

W. H. (*N. Y. H. No. 64020*), a white male, aged 24 years, was admitted to the hospital September 16, 1935, because of mild recurrence of rheumatic infection. He was discharged December 23, 1935. He had suffered from chorea since he was 8 or 10 years of age. At 10 years of age rheumatic heart disease was discovered. Mitral stenosis and insufficiency had been present since 12 years of age. The heart was enlarged. Normal sinus rhythm was present. The diagnoses were: *A*, rheumatic fever; *B*, mitral stenosis and insufficiency, cardiac enlargement; *C*, paroxysmal auricular fibrillation. On October 7, 1935, when the patient was under the influence of digitalis and there were no signs of congestive heart failure, observations of the circulation were made (Table I, Figure 1). On November 8, 1935, the rhythm changed to auricular fibrillation. On November 20, 1935, when auricular fibrillation was present, the patient still being under digitalis, studies were made again (Table I, Figure 1).

Summary of effects of auricular fibrillation

1. In 7 patients, the cardiac output in all except one (C. C.) was less during auricular fibrillation than after the reversion to normal sinus rhythm, whether the patient was or was not under the influence of digitalis at the time reversion to normal sinus rhythm occurred. In one patient (M. R.) on each reversion from normal rhythm to auricular fibrillation, the cardiac output decreased, and increased again when the normal rhythm supervened. In two instances (C. C. and F. K.) when digitalis was given during auricular fibrillation there resulted increase in cardiac output and decrease in cardiac size;⁶ in one of these (F. K.) reversion to normal mechanism was accompanied by still further increase in output to normal limits.

2. The heart was in some instances larger in the presence of auricular fibrillation than after restoration of normal sinus rhythm, and in other instances it was unchanged. When the use of digitalis was resorted to, decrease in size of the heart was observed. Results similar to this are reported by Stewart and his coworkers (18, 19, 20).

3. In those instances in which the arm to tongue circulation time was measured, it was prolonged

⁵ Experience with this particular "batch" showed that this amount was required regardless of body weight to slow the rapid ventricular rate in the presence of auricular fibrillation to around 70 per minute, when given within 24 hours; it was considered the digitalizing amount (17).

⁶ These observations are being reported at greater length (18, 19).

during auricular fibrillation and became shorter on restoration of normal sinus rhythm.

4. The venous pressure was elevated in those instances in which it was measured and restoration of normal sinus rhythm witnessed a fall to normal levels.

OBSERVATIONS RELATING TO AURICULAR FLUTTER.

There are observations of 4 patients exhibiting auricular flutter (Table I). In two of these (Cases H. B. and G. G.) comparison could be made between those made during auricular flutter and during normal sinus rhythm.

H. B. (N. Y. H. No. 113637), a white male, aged 21 years, was admitted to the hospital on November 25, 1935. Eleven days before admission he experienced shortness of breath, weakness, fatigue, and a tight feeling under the sternum on running up elevated railway stairs. Three days before admission he vomited and on examination by his physician paroxysmal tachycardia was discovered. The past history was not important. On examination there was slight cyanosis of the lips and the liver was palpable. The electrocardiogram showed 2:1 auricular flutter. There was no evidence of valvular disease. The diagnoses were: *A*, unknown; *B*, cardiac dilatation; *C*, paroxysmal auricular flutter.

Measurements were made on November 26, 1935 when auricular flutter was present (Table I, Figure 1). Digitalis, 2.2 grams, was given in 28 hours, and measurements were repeated on November 29th, reversion to normal sinus rhythm having occurred the day before. More digitalis was not given, and observations were made again on December 2d, as well as on December 18th, 21 days after digitalis had been discontinued. To make a valid comparison of the effect of auricular flutter on the circulation in this patient, the first observations made during auricular flutter (November 26th) and the last ones (December 18th) made after excreting digitalis should be compared (Table I, Figure 1). In this patient the presence of auricular flutter was not associated with rise in venous pressure.

G. G. (N. Y. H. No. 108714), a white male, aged 56 years, was admitted to the hospital on September 13, 1935. The patient gave a history of attacks of paroxysmal tachycardia during the last 14 years. The recent attack began 5 days before admission and was associated with headache, nervousness, constriction of the chest, weakness, palpitation, and vomiting. Dyspnea was present, as well as cyanosis, pulmonary râles, and enlargement of the liver. The electrocardiogram showed 2:1 auricular flutter, auricular rate 216 per minute. There was no evidence of valvular disease. The diagnoses were: *A*, arteriosclerosis; *B*, coronary artery disease; cardiac enlargement; coronary occlusion; [†] *C*, paroxysmal auric-

ular flutter. On September 14, 1935, studies of the circulation were made (Table I, Figure 1). After administration of digitalis, 0.8 gram, reversion to normal sinus rhythm occurred; he received, however, in all a total of 1.2 grams. On September 16, 1935, normal rhythm being present, observations were repeated, as well as on September 20, 1935. The abnormal rhythm in this patient was associated with rise in venous pressure (see also observations relating to ventricular paroxysmal tachycardia). It was not until the digitalis effect on the electrocardiogram had worn off that it was suspected that this attack had been associated with occlusion of a coronary artery (see observations relating to ventricular paroxysmal tachycardia).

A. G. (N. Y. H. No. 94972), a white male, aged 34 years, was admitted to the hospital on April 19, 1935, because of tachycardia of one day's duration. A similar attack had occurred 3 months before. Valvular heart disease had been discovered one year earlier. Eight months before admission dyspnea appeared; since it progressed rapidly it required him to remain in bed. The heart was tremendously enlarged. There was cyanosis of the lips, and the liver was palpable. The diagnoses were: *A*, unknown; *B*, mitral stenosis and insufficiency; aortic stenosis and insufficiency, cardiac enlargement; *C*, paroxysmal auricular flutter. On April 20, 1935, studies of the circulation were made (Table I, Figure 1). The patient was given digitalis and in 6 days received 4.5 grams, when normal rhythm was restored. Three hours later pulmonary edema developed, and the patient died shortly afterward.

A. Gl. (N. Y. H. No. 108422), a white female, aged 49 years, was admitted to the hospital on September 25, 1935, for measurements of the circulation. She was discharged the next day. Auricular flutter with a slow ventricular rate was discovered one and a half years ago when the patient began to have dyspnea, nervousness, and loss of weight. A diagnosis of hyperthyroidism was made. After thyroidectomy had been performed, auricular flutter persisted although she was given quinidine, as well as digitalis. She had experienced no signs of congestive heart failure and did not exhibit any at this time. The heart was perhaps slightly enlarged. In this patient the functions of the circulation which were measured were all in normal range while the patient was at rest and when the ventricular rate was slow (Table I, Figure 1).

Summary of effects of auricular flutter

It appears that in auricular flutter with rapid ventricular rate there is marked decrease in cardiac output per minute and very marked decrease per beat, with prolongation of circulation time and dilatation of the heart. Increase in venous pressure occurred in certain ones and not in others. With restoration of normal sinus rhythm, changes in all these functions in the reverse di-

[†] This diagnosis was not made until after the patient had been under observation for some time.

rection toward a normal level occur. In one patient who exhibited auricular flutter with a moderately slow ventricular rate approximately normal values were observed.

OBSERVATIONS RELATING TO SUPRAVENTRICULAR PAROXYSMAL TACHYCARDIA

There are observations of three patients exhibiting paroxysmal tachycardia of the supraventricular type (Table I).

M. H. (N. Y. H. No. 85620) exhibited paroxysmal tachycardia of nodal origin. She was a white female, aged 65 years, who had been subject to attacks of rapid, regular forcible beating of the heart associated with dyspnea and weakness since she was 37 years of age. They occurred every one to two months, lasting 5 minutes to 12 hours. There were no known precipitating factors. The attacks terminated spontaneously. She was admitted to the hospital on January 16, 1935, during an attack of paroxysmal tachycardia, which was auriculoventricular in origin. Reversion to normal sinus rhythm occurred before studies of the circulation could be made. The heart was not enlarged. The radial vessels were moderately thickened. The diagnoses were: A, arteriosclerosis; B, no cardiac enlargement; C, auriculoventricular paroxysmal tachycardia. On the morning of January 17th, the patient had only begun to eat breakfast when paroxysmal tachycardia recurred. She remained quiet for several hours after which studies of the circulation were made (Table I, Figure 2). Shortly after finishing these observations normal rhythm recurred spontaneously, and observations were repeated. Between this time and discharge on January 19, 1935, the patient experienced two more paroxysms.

C. F. (N. Y. H. No. 80473), a white female, aged 53 years, gave a history of having occasional attacks of rapid, regular beating of the heart, sudden in onset and offset for 15 years. Dyspnea and weakness accompanied the attacks which were likely to be brought on by emotional disturbances. She was admitted to the surgical service of the hospital on November 29, 1934, because of carcinoma of the rectum. On December 5, 1934, an abdominal exploration and transverse colostomy was performed in preparation for removal of the carcinoma by the perineal route. There was no evidence of valvular disease on examination. The diagnoses were: A, arteriosclerosis; B, no enlargement of the heart; C, auriculoventricular paroxysmal tachycardia. On December 20, 1934, at 3 p.m., paroxysmal tachycardia arising above the ventricles occurred when the patient was told a second operation was to be performed. Neither ocular nor vagal pressure was successful in ending the attack. At 4:30 p.m., morphine, 10 mgm., was given. In the evening (11 p.m.), when the heart rate was 187 per minute, observations of the circulation were made (Table I, Figure 2). Following the injection of mecholin, 25 mgm., subcutaneously, reversion to normal sinus rhythm occurred

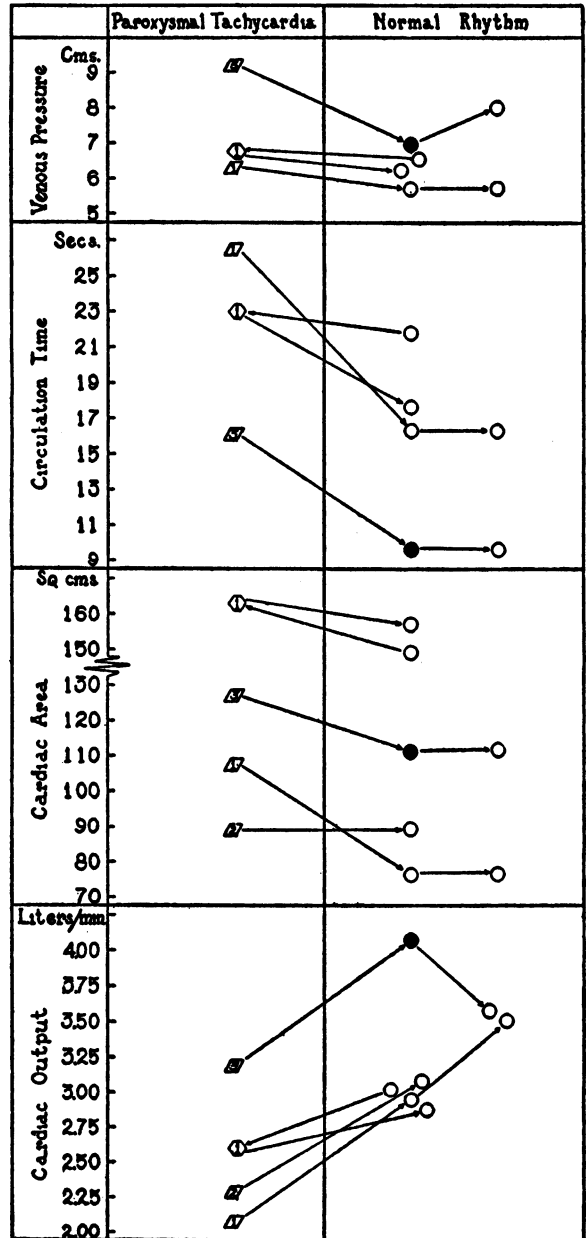


FIG. 2. DATA RELATING TO STUDIES OF THE CIRCULATION IN PATIENTS EXHIBITING PAROXYSMAL TACHYCARDIA

In this figure as well as in Figure 3 parallelograms and hexagons and circles indicate that the patient exhibited supraventricular and ventricular paroxysmal tachycardia, and normal rhythm respectively. The numerals 1, 2 and 3 in the parallelograms refer to Cases M. H., C. F., and R. T., respectively, and number 1 in the hexagon to Case G. G., in Table I. Closed and open symbols indicate that the patient was or was not under the influence of digitalis respectively.

in four and a half minutes. Forty-five minutes later when normal sinus rhythm was present, studies of the circulation were repeated (Table I, Figure 2).

The case of *R. T.* (*N. Y. H. No. 124470*), a white female, aged 29 years, illustrates the effect of auricular paroxysmal tachycardia. An attack started 4 days before admission, continuing for half a day. Tachycardia recurred the day before admission. It was accompanied by dyspnea and anorexia. The patient suffered rheumatic fever at 11 years of age and was told she had valvular heart disease. She had indulged in ordinary activities since 11 years of age without discomfort. Since 15 years of age she had experienced attacks of paroxysmal tachycardia lasting from a few minutes to several hours. Spontaneous reversion to normal sinus rhythm had occurred until this occasion. The patient exhibited no signs of congestive heart failure. The diagnoses were: *A*, rheumatic fever; *B*, mitral stenosis and insufficiency; *C*, auricular paroxysmal tachycardia. Studies of the circulation were made on February 26, 1936, during auricular paroxysmal tachycardia (Table I, Figure 2). The use of mecholol, 25 mgm., subcutaneously on two occasions resulted in reversion to normal sinus rhythm for a few minutes only. Digitalis was then given and reversion to normal sinus rhythm occurred after administration of 1.6 grams. On February 28, 1936, when the rhythm was normal, studies were repeated, as well as on March 14, 1936 (2 weeks later) after excretion of digitalis. The precise effect of the paroxysmal tachycardia in this patient is revealed by comparison of the data made during the paroxysm (February 26, 1936) with those made during the normal sinus rhythm on March 14, 1936, after excretion of digitalis.

Summary of data relating to supraventricular paroxysmal tachycardia

It appears from studies of three patients exhibiting paroxysmal tachycardia of supraventricular origin that this rhythm was associated with decrease in cardiac output per minute and per beat and slowing of the circulation time. Rise in venous pressure did not occur.

VENTRICULAR PAROXYSMAL TACHYCARDIA

There are studies of one patient suffering from ventricular paroxysmal tachycardia (Table I).

The history of *G. G.* (*N. Y. H. No. 108714*) has already been recorded (*G. G.*, auricular flutter, Table I). While the patient was still resting in bed, he suffered a second attack of paroxysmal tachycardia on September 26, 1935, which was ventricular in origin at a rate of 179 per minute. This stopped spontaneously in 24 hours. The electrocardiogram after reversion to normal sinus rhythm suggested that this attack was associated with coronary occlusion. On November 6, 1935, before allowing the patient to sit up, studies of the circulation

were made. At this time, when normal sinus rhythm was present, measurements of the circulation were made (Table I, Figure 2). On November 8th, ventricular paroxysmal tachycardia recurred; it was still present on November 10th, 36 hours later, at a ventricular rate of 200 per minute, and observations were repeated. The patient received 5.2 grams of quinidine in 3 days and reversion to normal sinus rhythm occurred at 2 a.m. on November 13th, and later in the morning when normal sinus rhythm was still present observations were repeated. The patient experienced another attack of ventricular paroxysmal tachycardia on December 7th. He was discharged December 21, 1935.

Summary of effects of ventricular paroxysmal tachycardia

Observations made of this patient before the onset of ventricular paroxysmal tachycardia, during paroxysmal tachycardia, and again after restoration of normal sinus rhythm revealed decrease in cardiac output per minute and per beat, prolongation of the circulation time, and dilatation of the heart during this rhythm.

PART II. OBSERVATIONS RELATING TO RHYTHMS ASSOCIATED WITH SLOW VENTRICULAR RATE

There are eight patients exhibiting certain abnormalities of the rhythm in which the ventricular rate was slow: in 4 (*Cases H. B., G. N., A. R., and C. K.*) complete heart block was present, in 2 (*Cases T. C. and J. L.*) 2:1 heart block, in 1 (*Case H. J.*) sinus bradycardia, and in 1 (*Case J. G.*) coupled rhythm due to auricular premature contractions (Table II).

OBSERVATIONS IN COMPLETE HEART BLOCK

There are observations of 4 patients exhibiting complete heart block (*H. B., G. N., A. R., and C. K., Table II*).

H. B. (*N. Y. H. No. 26415*), a white male, aged 56 years, was admitted to the hospital on March 22, 1934. He suffered an attack of acute rheumatic fever when 36 years of age. At 46 years of age, insurance was refused because of a cardiac "condition." In September, 1932, he first observed dyspnea. On April 28, 1933, the electrocardiogram showed normal sinus rhythm with right intraventricular heart block. On July 31, 1933, the electrocardiogram showed 3:1 heart block. In December, 1933, complete heart block occurred and persisted. He experienced Stokes-Adams attacks occasionally. The heart was enlarged. He exhibited no signs of congestive heart failure at the time these observations were made. The diagnoses were: *A*, arteriosclerosis; *B*, cardiac en-

TABLE II *

Data on patients exhibiting heart block and other rhythms associated with slow ventricular rate (Figure 4)

Case; Hospital number; Age	Date	Body surface	Oxygen consumption	Basal metabolic rate	Arterio-venous oxygen difference	Cardiac output	Cardiac output	Heart rate	Cardiac output	Cardiac area	Cardiac volume	Arterial pressure	Left ventricular work	Circulation time†	Venous pressure	Vital capacity	Rhythm	Red blood count	Hemoglobin
		sq. m.	cc. per minute	per cent	cc.	liters per minute	liters per sq. m. per minute	per minute	cc. per beat	sq. cm.	cc.	mm. Hg	gram-meters per beat	seconds	cm. saline	cc.		millions	per cent
COMPLETE HEART BLOCK																			
H. B. No. 20415 ♂ 56 years	Mar. 23, 1934	1.75	204	-10	79.3	2.58	1.47	35	73.7	134.9	1430	128/60	94.2			2710	C.-H.B.	5.0	104
	Mar. 25, 1934	1.75	209	-8	76.0	2.75	1.57	35	83.3	134.9	1430	120/58	100.9			2670	C.-H.B.		
G. N. No. 46694 ♀ 52 years	April 24, 1934	1.53	148	-20	83.7	1.81	1.18	44	41.1	118.6	1186	120/80	55.9			1620	C.-H.B.	5.1	101
	May 1, 1934	1.53	149	-20	71.0	2.09	1.37	40	52.2	118.2	1168	108/68	62.5			1750	C.-H.B.		
A. R. No. 4025 ♂ 49 years	May 16, 1935	1.64	224	+4	67.3	3.33	2.03	37	90.0	145.2	1587	160/58	133.4	12.4	9.5	3600	C.-H.B.		
C. K. No. 189625 ♂ 54 years	Jan. 10, 1938	1.74	197	-11	64.8	3.04	1.75	32	95.0	141.6	1540	155/95	161.5	25.8 s 35.6 d	2.5	3650	C.-H.B.	4.7	98
2:1 HEART BLOCK																			
T. C. No. 111203 ♀ 73 years	Nov. 19, 1935	1.60	159	-12	95.2	1.67	1.04	30	55.7	192.2	2428	150/68	82.6	32.2	5.8	2250	2:1 H.B.	5.1	100
J. L. No. 41361 ♂ 71 years	April 27, 1936	1.87	228	+2	83.3	2.74	1.47	48	57.1	137.2	1466	164/80	94.7	23.1	5.7	3200	2:1 H.B.	3.9	70
SINUS BRADYCARDIA																			
H. J. No. 42048 ♂ 41 years	April 26, 1934	1.80	245	0	77.4	3.16	1.76	47	67.3	147.2	1620	118/86	93.4			3810	N.R.	5.8	102
COUPLED RHYTHM DUE TO AURICULAR PREMATURE CONTRACTIONS																			
J. G. † No. 68753 ♀ 68 years	June 6, 1934	2.00	218	-5	73.2	2.98	1.49	30	99.0	185.0	2295	190/140	217.6			2750	Idiovent. and A.P.C. giving coupled rhythm	5.5	95
	June 9, 1934	1.99	224	0	82.8	2.71	1.37	27	100.0	190.1	2394	190/130	222.2			3125			

* See Table I for abbreviations.

† "s" and "d" indicate arm to tongue circulation time recorded during systole and the prolonged diastolic period respectively.

‡ Figure 4 was not extended to include this patient.

largement; C, complete heart block, right intraventricular heart block. Observations were made on March 23, 1934, as well as 2 days later (Table II).

G. N. (N. Y. H. No. 46694), a white female, aged 52 years, suffered from complete heart block which had its onset during an attack of diphtheria at 9 years of age. She was admitted to the hospital on April 21, 1934. At 12 years of age, she suffered an attack of acute rheumatic fever with recurrences at 24 and 35 years of age. She suffered cardiac symptoms first in November 1933. There was precordial pain accompanied by palpitation and dyspnea and constriction of the chest, lasting one to two minutes. These attacks increased to 6 to 8 every day but after digitalization in the outpatient department the at-

tacks decreased to one or two. After resting two weeks, the attacks increased again in frequency when she attempted greater activity. She was admitted to the hospital April 21, 1934, to be given larger amounts of digitalis. The diagnoses were: A, diphtheria; B, cardiac enlargement; C, complete heart block. She received digitalis, 1.0 gram, on April 22 and 23, 1934. At rest there were no signs of congestive heart failure. Studies of the circulation were made on April 24, 1934 (Table II) and were repeated on May 1, 1934. She had received digitalis, 0.2 gram, daily except on April 26th when the amount was 0.7 gram.

A. R. (N. Y. H. No. 4025), was a white male aged 49 years. At 26 years of age, a routine physical examina-

tion showed a "slow pulse and a heart murmur." Life insurance was refused at 31 years because of these. At 41 years he became easily fatigued. He suffered an attack of rheumatic fever at 42 years followed by a second attack at 45 years. The electrocardiogram showed incomplete auriculoventricular heart block in February, 1932; complete heart block had been present, however, since September, 1932. At the time our observations were made, he experienced slight precordial pain, and dyspnea on exertion. Examination revealed no signs of congestive heart failure. The diagnoses were: *A*, rheumatic fever, hypertension; *B*, mitral insufficiency; *C*, complete heart block. On May 16, 1935, studies of the circulation were made (Table II).

C. K. (*N. Y. H. No. 189625*), a white male, aged 54 years, was admitted to the hospital on December 31, 1937, complaining of "dizzy spells." He had never suffered from rheumatic fever nor chorea. He had enjoyed excellent health until 10 weeks before admission when he began to experience cramps in the calves of both legs on walking. Eight weeks before admission he began to suffer from moderate dyspnea on exertion, and five weeks before admission he experienced an attack characterized by the successive appearance of whistling in the ears, whirling vertigo, and brief loss of consciousness. The entire succession of events lasted about 60 seconds. Attacks of this nature recurred daily in the 3 weeks preceding admission. Examination on December 31, 1937, revealed no evidence of heart failure of the congestive type. Observation of the venous pulsations in the neck together with auscultation at the apex of the heart indicated that the rhythm was complete heart block with a ventricular rate of 30 per minute. The peripheral arteries were moderately thickened and tortuous, but the arterial pulses at both wrists appeared to be of good volume. The diagnoses were: *A*, arteriosclerosis; *B*, enlargement of the heart, fibrosis of the myocardium; *C*, complete heart block alternating with incomplete heart block, right intraventricular heart block. The patient was kept at rest in bed, and did not experience any further attacks. On January 6, 1938, he was given ephedrine sulphate, 0.025 gram, 4 times daily. The drug did not appear to induce any significant change in the ventricular rate. Studies of the circulation were made on January 10, 1938, when complete heart block was present.

Summary of data relating to complete heart block

Studies were made of 4 patients exhibiting complete heart block without congestive heart failure. The cardiac output per minute was decreased in three and normal in one, and the output per beat was increased or only slightly decreased. The basal metabolic rate was decreased in three subjects.

OBSERVATIONS IN 2:1 HEART BLOCK

There are studies of 2 patients exhibiting 2:1 heart block (*T. C.* and *J. L.*, Table II).

T. C. (*N. Y. H. No. 111203*), a white female, aged 73 years, was admitted to the hospital on October 30, 1935, and discharged November 24, 1935. She exhibited signs of congestive heart failure (ascites, dyspnea, slight edema) beginning 16 months before admission. The heart was enlarged. There was marked arteriosclerosis. The electrocardiogram showed auricular flutter (auricular rate 272 per minute) with complete heart block, ventricular rate 30 per minute. On November 19, 1935, however, normal sinus rhythm was present, with 2:1 heart block; the ventricular rate was 30 per minute. The patient now being free of signs of congestive heart failure, the cardiac output was measured (Table II). The diagnoses were: *A*, hypertension; *B*, cardiac enlargement; *C*, 2:1 heart block; right intraventricular heart block.

J. L. (*N. Y. H. No. 41361*), a white male, aged 71 years, was admitted to the hospital on April 23, 1936, because of symptoms of early prostatic obstruction. Hypertension had been present for many years and the patient had suffered right hemiplegia 9 years before. Heart block, 2:1, was discovered in 1934. The radial vessels were thickened. At rest, there was no evidence of congestive heart failure. The diagnoses were: *A*, hypertension; *B*, enlarged heart; *C*, 2:1 heart block. Studies of the circulation were made on April 27, 1936 (Table II).

Summary of data relating to 2:1 heart block

The total cardiac output per minute was decreased in the presence of 2:1 heart block as a consequence of which the circulation time was prolonged. The output per beat, due to the slow cardiac rate, was within normal limits.

SINUS BRADYCARDIA

H. J. (*N. Y. H. No. 42048*), a white male, aged 41 years, was admitted to the hospital on April 13, 1934, suffering from dry pleurisy of 10 days' duration. There was no rise in temperature. Examination of the sputum did not reveal acid fast organisms. The friction rub and pleural pain disappeared within a few days and convalescence was uneventful. The x-ray photograph of the chest revealed no evidence of tuberculosis. There was no history of rheumatic infection. Examination of the heart and circulation revealed no abnormality. Sinus bradycardia was present. There were no signs of congestive heart failure. On April 26, 1934, when the heart rate was 47 per minute, the cardiac output was measured (Table II).

Coupled rhythm due to auricular premature contractions

J. G. (*N. Y. H. No. 66753*), a white male, aged 68 years, was admitted to the hospital on June 1, 1934, because of the presence of dyspnea and moderate edema of several weeks' duration. He had been given small amounts of digitalis for 5 days but none had been given

for 6 days before admission to the hospital. He had suffered one attack of polyarthritis when 49 years old. On admission there was no dyspnea and no cyanosis at rest. A few râles were heard at the lung bases posteriorly. The liver was not large. There was very slight edema of the ankles. The heart was enlarged to the left. Peripheral arteriosclerosis was marked. The Wassermann reaction of the blood was negative. On rest in bed and limitation of the salt (2.0 grams daily) and fluid intake, signs of congestive heart failure disappeared. Because of the abnormal rhythm, studies of the circulation were made on June 6, 1934, and again on June 9, 1934 (Table II). The diagnoses were: *A*, hypertension, arteriosclerosis; *B*, enlarged heart; aortic insufficiency; *C*, idioventricular rhythm; auricular premature contractions giving rise to coupled rhythm.

Summary

Coupled rhythm due to auricular premature contractions in the presence of idioventricular rhythm was associated with decrease in cardiac output per minute, but with marked increase in the output per beat.

DISCUSSION

It appears that auricular fibrillation and flutter, as well as auricular, nodal, and ventricular paroxysmal tachycardia, are associated with marked decrease in the functional capacity of the heart. At rest, the heart pumps a decreased amount of blood per minute, and as a consequence, a decreased output per beat since the cardiac rate is rapid. This change is reflected in increase in the arm to tongue circulation time, in short, in slowing of the velocity of blood flow; in one instance of auricular fibrillation and one of auricular flutter, the venous pressure was elevated. In most instances, the abnormal rhythms were associated with dilatation of the heart, for the size of the heart became smaller after reversion to normal sinus rhythm. The most extreme decrease in cardiac output per beat was observed in the cases exhibiting auricular flutter and paroxysmal tachycardia; in certain ones it fell to 9 cc. per beat only. Our data regarding cardiac output in auricular fibrillation are in agreement with those recorded by Kerkof and Baumann (9, 10), and Stewart (21, 22).

The data relating to the work of the heart is of special interest: the work of the heart per beat was markedly decreased during these abnormal rhythms. The work of the heart has been cal-

culated from the formula (23):

$$W = QR + \frac{wV^2}{2g},$$

where W equals work done per minute or per beat; Q equals volume of blood expelled per minute or per beat; R equals mean arterial blood pressure in mm. of Hg $\times 13.6$; V equals velocity of blood at the aorta; w equals weight of blood; g equals acceleration due to gravity. The last part of the formula ($wV^2/2g$) has been omitted to make our results comparable to those reported by Starr and his associates (7, 8). By substituting values in this formula, we have calculated the amount of work done by the left ventricle per beat. The cardiac volume has been plotted against grammeters of work of the left ventricle (Figure 3) in a fashion similar to that used by Starr and his coworkers (7, 8). It appears to be a fact that the work of the left ventricle, which is maintaining an adequate circulation, bears a linear relation to the size of the heart, and from their data, they have defined a zone of normal circulatory function.

Correlation of the work per beat with the size of the heart in patients exhibiting auricular fibrillation, auricular flutter, and paroxysmal tachycardia places most of them outside the zone of normal circulatory function during the abnormal rhythm, below the line CD , but they move up closer or into the zone of normal circulatory function, between lines CD and EF , or close to the best line, AB (Figure 3), with reversion to normal sinus rhythm. In two cases reported by Starr, changes in cardiac output and work similar to ours are reported.

In one patient exhibiting auricular flutter with slow ventricular rate (A. Gl., Table I) the circulatory function was normal.

When digitalis was given to those exhibiting auricular fibrillation, there resulted an increase in cardiac output, decrease in cardiac size, and shortening of the circulation time, observations which are reported in more detail elsewhere (18, 19). These results are in agreement with those recorded by Stewart and Cohn (20). When reversion to normal sinus rhythm occurred after digitalization, the cardiac output increased further and the size of the heart became smaller still.

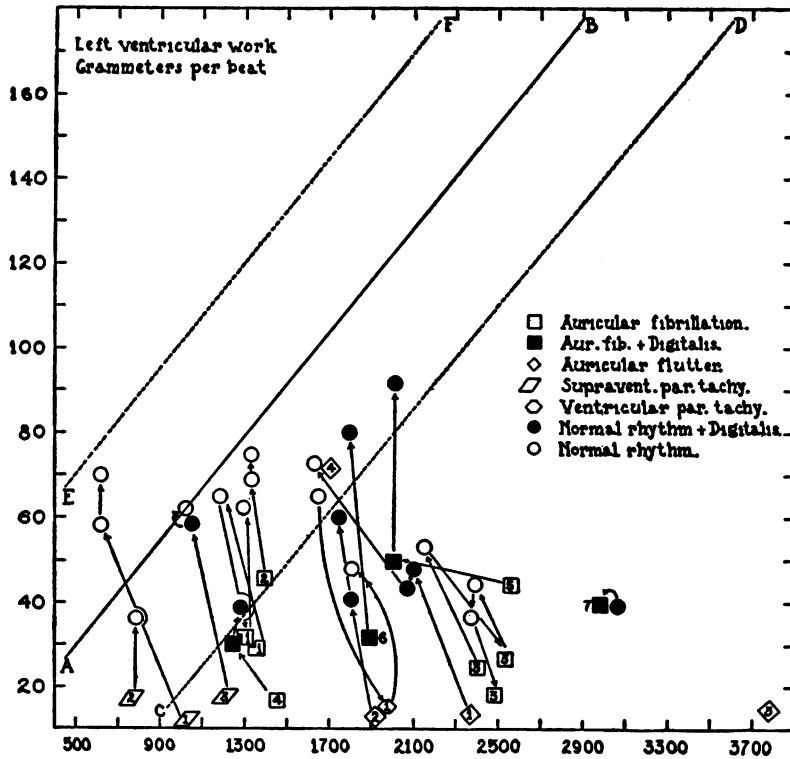


FIG. 3. LEFT VENTRICULAR WORK PER BEAT AND CARDIAC VOLUME

The data from Table I relative to work of the left ventricle per beat in the presence of auricular fibrillation, auricular flutter, and paroxysmal tachycardia are plotted against corresponding cardiac volumes. Line *AB* represents the best line defined by Starr, Collins, and Wood (7). Lines *CD* and *EF* are placed by these authors at a distance of twice the standard deviation from *AB*. A patient falling within the zone *CD-EF* has a normal circulatory function, or the work of the heart is commensurate with its size. The values of patients exhibiting auricular fibrillation, auricular flutter, and paroxysmal tachycardia, now being reported, fell outside the zone *CD-EF* in all except 3 instances. On reversion to normal sinus rhythm, all patients moved up closer toward or even into the zone of normal circulatory function or, if already in this zone, nearer the best line, *AB*. The symbols in this chart are the same as those used in Figures 1 and 2.

There has been demonstrated marked decreased functional capacity of the hearts exhibiting these rhythms while the subjects are resting. It is likely that this becomes even more marked when greater demands are placed on the heart by exertion and exercise.

Observations relating to the cardiac rhythms in which the ventricular rate is slow revealed diminution in cardiac output per minute in all but one patient (A. R., Table II). Nevertheless, because of the slow ventricular rate the output per beat was equal to or greater than normal, or at most not markedly decreased. The hearts were

not large. The net outcome of this was that at rest, these patients fall into the zone of normal circulatory function, area *CD-EF*, in which the work performed by the heart is commensurate with its size (Figure 4). One patient (T. C., Table II) exhibiting 2:1 heart block is of especial interest since she fell below line *CD*, that is to say, outside the zone of normal circulatory function. She had only recently recovered from congestive heart failure. Starr (7, 8) observed that patients who have experienced congestive heart failure but do not exhibit evidence of it at the time of observation may fall outside this zone.

Our own observations relating to congestive heart failure give evidence which points to the same conclusion (19, 24). The graphic representation of patients exhibiting complete heart block in the zone of normal circulatory function is in agreement with the clinical observation that patients suffering from this rhythm carry on for long periods without experiencing congestive heart failure. The decrease in cardiac output which was present in 4 of our cases and 2 of Starr's (8) calls for comment since other observers have reported normal values (Grollman (11), p. 221). The cases we are now reporting were in the middle decades, yet the range from decrease to normal was recorded. We are unable to express an opinion whether arteriosclerotic changes may account for the difference in functional capacity. It is recalled again, however, that all fell into the zone of normal circulatory function (Figure 4).

Our data show that the basal metabolic rate may be low in the presence of complete heart block and 2:1 heart block, a phenomenon to which others have already directed attention (8). This is probably a compensatory mechanism on the part of the human organism, for, by decreasing the total oxygen requirements of the tissues, the cardiac output, though diminished, may be utilized to its fullest extent. This compensatory mechanism is equivalent to presenting this smaller minute volume output to an individual with a smaller body surface, for whose requirements it would be ample.

The two patients exhibiting sinus bradycardia and coupled rhythm (H. J. and J. G., Table II, Figure 4) require no special comment, since the situation in them is essentially the same as in those having heart block.

SUMMARY

Auricular fibrillation, auricular flutter, and paroxysmal tachycardia, of the supraventricular as well as the ventricular type were associated in the instances observed with decreased cardiac output per minute, per beat, decrease in velocity of blood flow, and dilation of the heart, and decrease in the work of the heart per beat. As a consequence, these patients fell in the heart failure zone when the abnormal rhythm was present. The cardiac output per minute was likewise usually decreased in heart block, but, in contrast to

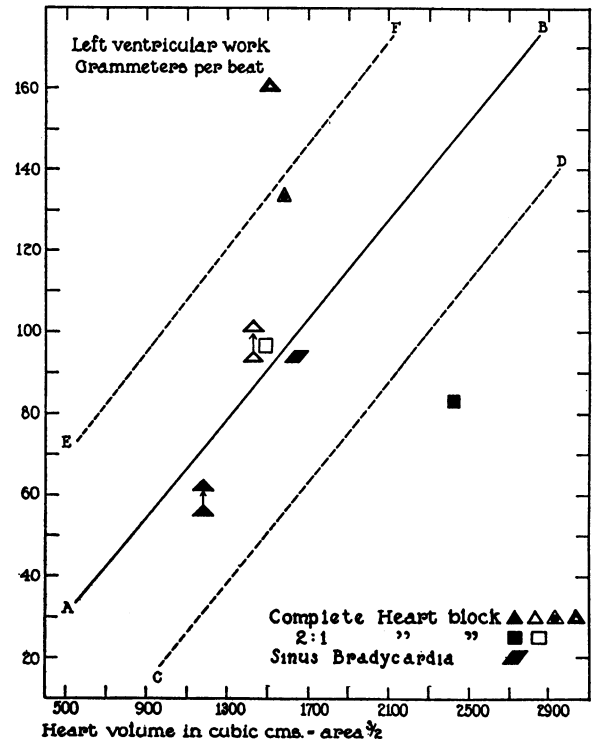


FIG. 4. LEFT VENTRICULAR WORK PER BEAT AND CARDIAC VOLUME

The data from Table II relating to work of the left ventricle per beat in the rhythms associated with a slow ventricular rate are plotted against the corresponding cardiac volumes, in a manner similar to Figure 3. The open and the closed triangles, the triangle with a dot, the triangle within a triangle, the open and the closed squares, and the closed parallelogram refer respectively to the cases in the sequence they are recorded in Table II. All patients fell in the zone of normal circulatory function except T. C. (solid square) who had recently recovered from congestive heart failure.

the rapid rhythms, the output per beat was increased, and the work per beat commensurate with the size of the heart. The work of the heart was normal except in the instance in which the patient had recently suffered congestive heart failure. A patient exhibiting sinus bradycardia, as well as one with coupled rhythm, at rest, resembled those with heart block and fell in the zone of normal circulatory function.

CONCLUSION

The rapid regular and irregular rhythms in human beings at rest are associated with marked decrease in functional capacity of the heart, as measured by cardiac output per minute and per beat

and the work per beat. They were associated with the dilatation of the heart. They are very inefficient rhythms, the work of the left ventricle per beat not being commensurate with the size of the heart. As a consequence, in most instances they fall outside the zone of normal circulatory function. On the other hand, rhythms associated with slow ventricular rate such as those illustrated by complete heart block, are not incompatible with a normal circulatory function when the subject is at rest. Patients suffering from these rhythms may exhibit lowering of the basal metabolic rate as a compensatory mechanism.

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