CLINICAL STUDIES ON THE VELOCITY OF BLOOD FLOW

IX. The Pulmonary Circulation Time, the Velocity of Venous Blood Flow to the Heart, and Related Aspects of the Circulation in Patients with Cardiovascular Disease[1]

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Introduction

This paper presents the first study of the pulmonary circulation time in cardiovascular disease, and its relation to the velocity of venous blood flow in the arm, to the vital capacity of the lungs, to the arterial and venous blood pressures, and to the clinical symptoms and signs. The early occurrence of dyspnea and the early reduction of vital capacity are among the first disturbances in cardiac failure and indicate early changes in the dynamics of the circulation of blood through the lungs. The physiological and pathological importance of the pulmonary blood flow has consequently always attracted considerable interest but, until now, the peculiar inaccessibility of the pulmonary vessels has necessitated recourse to animal experimentation for direct observations. Unfortunately, however, such experiments reproduce but imperfectly and crudely conditions comparable to clinical cardiovascular disease. In man, on the other hand, only indirect observations have been possible by measurements such as the pulmonary minute volume flow according to the principle of Ficke. Such observations are unsatisfactory in the presence of dyspnea and demand considerable coöperation on the part of the patient.

In preceding communications (1) (2) (3) the time of the circulation from the right antecubital vein to the left antecubital artery was

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studied in patients with cardiovascular disease. Such an "arm to arm circulation time" is a rather complex expression of the peripheral arm blood flow as well as of the central pulmonary blood flow. By means of the technique described in preceding communications (4) (5), measurement of the time of arrival of the active deposit of radium in the right chambers of the heart has become possible. The time that elapses between the injection of the active deposit into the antecubital vein and the arrival of the active deposit in the right chambers of the heart has been termed "the arm to heart time" for it is a measure of the velocity of the venous blood of the arm to the heart. The time that elapses between the arrival of the active deposit of radium in the right chambers of the heart and its arrival in the arteries about the elbow of the arm may be called "the crude pulmonary circulation time." Although the "crude pulmonary circulation time" includes the time of transit of the active deposit from the heart to the antecubital arteries; the velocity of arterial blood flow, particularly in vessels as large as the aorta, the subclavian, and brachial arteries, is conspicuously rapid and must be relatively short compared to the actual pulmonary circulation time. For practical purposes the crude pulmonary circulation time provides an estimate of the velocity of blood flow through the lungs. Consequently it seemed desirable to study the pulmonary and the arm blood flow in cardiovascular disease and their relation to the other aspects of the circulation above cited. As in a previous communication, the patients have been grouped according to the etiology of their cardiovascular disease in order to learn whether the sequence of events in the development of cardiac failure differs according to the etiology and according to the corresponding types of lesions produced.

METHOD

The procedures employed were those described in preceding studies (4) (5) on the pulmonary circulation time in normal resting individuals. The conditions of the tests were identical except that in some patients with circulatory insufficiency slightly larger amounts of active deposit of radium (8 to 10 millicuries) were injected. In brief, venous pressure was measured directly by the venipuncture method of Moritz and Tabora; the vital capacity, by means of a Collins spirometer. The

velocity of blood flow was measured by injecting small amounts of active deposit of radium into the antecubital veins of the right arm and detecting the time of arrival first, in the right chambers of the heart, and later, in the antecubital arteries about the elbow of the left arm.

As previously pointed out, the advantages of the method are as follows: (1) a quantitative and objective measurement of a fundamental and hitherto unstudied aspect of the circulation is made feasible; (2) no coöperation on the part of the patient is necessary; (3) the substance injected is non-toxic in the amounts utilized; (4) measurements can be repeated after three hours; (5) the velocity of the pulmonary flow can be estimated for the first time in man; (6) the variability of the peripheral capillary circulation is largely obviated, and (7), since with the arrival of the active deposit in the antebrachial arteries, the radiations from the active deposit automatically cause registration of the time of arrival, withdrawal of blood is not necessary. The method also possesses certain limitations. The "circulation time" expresses the time necessary for the transit of the fastest particle through the shortest path and does not directly measure the mean velocity. That such a "circulation time" is closely related, however, to the mean velocity, at least in normal individuals, is borne out by considerations discussed elsewhere.

The interval between the moment of injection and the time of arrival of the active deposit in the right auricle is called "the arm to heart time." The time required for the active deposit to travel from the right auricle to the antecubital arteries is termed "the crude pulmonary circulation time." By applying a standard correction of 4.3 seconds based on other measurements secured by us in the same normal individuals, the actual pulmonary circulation time was estimated (5). In 112 normal persons, the average arm to arm circulation time was 17.5 seconds. In 58 normal persons, the arm to heart time averaged 6.6 seconds; the crude pulmonary circulation time, 10.8 seconds, and the actual pulmonary circulation time, 6.5 seconds. The arm to arm circulation time of some of the patients included in this study has been utilized in preceding communications (3) (4).

I. THE PULMONARY CIRCULATION TIME, THE VELOCITY OF VENOUS FLOW TO THE RIGHT AURICLE, AND THEIR RELATION TO OTHER ASPECTS OF THE CIRCULATION IN PATIENTS WITH RHEUMATIC HEART DISEASE

Rheumatic infection of the heart causes its serious effects in at least three ways: (1) by invasion of the myocardium; (2) by deformation of the valves; (3) by producing conditions favorable for the occurrence of auricular fibrillation. In order to learn the relative importance of these factors in affecting the velocity of blood flow through the lungs and of the venous blood to the right auricle, patients with rheumatic heart disease (table 1) have been grouped in three classes.

A. Patients after acute rheumatic fever but without evidence of valvular damage

The two patients, F. D. (no. 292) and G. (no. 267), represent the immediate and the very late effects of myocardial involvement uncomplicated by any demonstrable effect on the valves or on the rhythm. F. D. (no. 292), who had recovered from acute rheumatic fever but one week previously, showed a normal pulmonary circulation time, a normal velocity of venous blood to the right auricle, a normal vital capacity, normal venous and arterial blood pressures, and a normal electrocardiographic tracing. G. (no. 267), on the other hand, although he showed neither evidence of valvular damage nor disturbance of rhythm, had been troubled by increasing dyspnea for four months and by orthopnea for three weeks. He showed a rapid ventricular rate, squeaking rhonchi over the bases of the lungs, and a lowered vital capacity. The pulmonary circulation time was definitely prolonged (twenty-four seconds) while the velocity of venous blood to the right auricle was within the limits of normal (seven seconds). In this instance, the rheumatic damage was confined to the myocardium while the valves, according to clinical evidence, were uninvolved.

B. Patients with rheumatic valvular heart disease with regular rhythm

Of the eight patients in this group, the first seven showed pulmonary circulation times within normal limits. It should be emphasized that with the exception of patient R. F. (no. 363), who became dyspneic only on walking up one flight of stairs, none of these seven patients

TABLE 1

The pulmonary circulation time, the arm to heart time and their relation to other aspects of the circulation in patients with rheumatic heart disease

Test number	Date	Name	Age	Temperature	Pulse	Surface area	Venous pressure	Arterial pressure		Vital capacity	Vital capacity per square meter	Circulation time			Circulation time per square meter			Injected
								Systolic	Diastolic			Arm to heart	Pulmonary	Arm to arm	Arm to heart	Pulmonary	Arm to arm	
Group A. Patients convalescent from acute rheumatic fever but without evidence of valvular damage																		
						square meter	*cm. H_2O*	*mm. Hg.*	*mm. Hg.*	*cc.*	*cc.*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*millicuries*
292	November 4, 1926	F. D.	39	98.3	88	1.52	−2.0	138	72	3,300	2,172	5.0	9.0	14.0	3.3	5.9	9.2	4.5
267	October 19, 1926	G.			142	1.67	5.0	120	54	2,000	1,197	7.0	24.0	31.0	4.1	14.4	18.5	5.2
Group B. Patients with rheumatic valvular heart disease with regular rhythm																		
376	February 3, 1927	A. C.	15	99.1	108	1.52	2.5	126	66	3,350	2,205	3.5	8.0	11.5	2.3	5.2	7.5	6.0
287	October 28, 1926	K. N.	23	97.8	88	1.74		180	66	5,000	2,816	7.0	8.0	15.0	4.0	4.6	8.6	4.5
338	January 6, 1927	F. M.	30	98.8	85	1.68	4.0	124	74			4.5	9.0	13.5	2.7	5.3	8.0	7.0
340	January 6, 1927	J. G.	24	98.8	88	1.76	3.5	134	50	4,350	2,470	5.5	10.0	15.5	3.1	5.7	8.8	6.0
363	February 10, 1927	R. F.	22	98.6	66	1.76	9.5	142	78	3,900	2,215	16.0	11.0	27.0	9.1	6.2	15.3	8.0
368	February 16, 1927	M. E.	22	97.8	59	1.59	1.5	108	50	3,600	2,262	12.0	11.5	23.5	7.5	7.2	14.7	7.0
283	October 28, 1926	W. O.	42	99.2	101	1.88	−1.0	142	42	3,200	1,702	4.0	15.0	19.0	2.1	7.9	10.0	5.0
369	February 16, 1927	P. G.	22	98.8	92	1.67	10.5			2,400	1,437	17.0	18.5	35.5	10.2	11.0	21.2	
Group C. Patients with rheumatic valvular heart disease with fibrillation of the auricles																		
258	September 30, 1926	D. S.	40	98.4	64	1.60	4.0	130	85	2,300	1,438	11.0	19.0	30.0	6.8	11.8	18.6	
324	December 7, 1926	S. C.	26	96.6	76	1.57	10.0	115	66	2,575	1,694	9.5	19.0	28.5	6.0	11.7	17.7	9.5
265	October 19, 1926	S. C.	26	97.4	75	1.50	4.2	105	40	2,650	1,766	9.0	20.0	29.0	6.0	13.3	19.3	5.5
406	April 6, 1927	E. G.	37	98.2	52	1.79	14.5			2,600	1,452	14.0	29.0	43.0	7.8	16.2	24.0	9.0
358	February 9, 1927	H. M.	27	97.4	45	1.60	7.5	106	84	3,050	1,906	14.0	38.0	52.0	8.7	23.8	32.5	8.0

had had symptoms or signs of circulatory insufficiency in spite of some having clinical evidence of advanced valvular lesions. Patient P. G. (no. 369), on the other hand, showed symptoms of circulatory insufficiency at the time of test, and his pulmonary circulation time was prolonged. In the first seven patients in whom the circulation was clinically entirely compensated, the venous pressure averaged 8.3 cm. of water (average normal 7.3); the vital capacity of the lungs 2278 cc. per square meter of body surface (average normal 2376 cc.); the arm to heart time, 7.5 seconds (average normal 6.6 seconds); and the crude pulmonary circulation time 10.4 seconds (average normal 10.8 seconds). It should be noted that the measurements on these patients coincide within the limit of error to the values found in a larger group of normal individuals.

The pulmonary circulation times found in the first seven patients demonstrate that in spite of unquestionable evidence of valvular deformity, the myocardium may be capable of maintaining a normal speed of blood flow through the lungs. The absence of previous symptoms and signs of circulatory insufficiency, the practically normal venous pressure, and the normal vital capacities of the lungs are in accord with the normal speed of blood flow.

C. Patients with rheumatic valvular heart disease with fibrillation of the auricles

The venous pressure in the four patients in this group averaged 14.1 cm. of water (average normal 7.3 cm.); the vital capacity, 1088 cc. per square meter of body surface (average normal 2376 cc.); the arm to heart time, 12.5 seconds (average normal 6.6 seconds); and the crude pulmonary circulation time, 21.6 seconds (average normal 10.8 seconds).

The pulmonary circulation times of these patients were, in all cases, more prolonged than in the presence of group B with regular ventricular rhythm, and it should be noted that, likewise, all the patients with auricular fibrillation had suffered from severe circulatory decompensation and showed at the time of test symptoms or signs of congestive failure. Comparison of the measurements with the clinical summaries shows that in each patient the arm to heart time, the pulmonary circulation time, and the vital capacity of the lungs cor-

responded closely to the degree of circulatory compensation whereas the venous pressure varied considerably.

Measurements 265 and 324 are of interest since they were performed on the same patient S. C. almost six weeks apart. His condition, according to clinical signs and symptoms, according to the vital capacity, and to the pulmonary circulation time, was approximately the same at the time of both tests. This is in harmony with our general experience that the velocity of blood flow parallels, in general, the degree of circulatory competence and as such provides an objective index of the degree of circulatory compensation.

Discussion

The patients of groups A, B, and C show, so far as it is possible to differentiate them clinically, (1) the effects of rheumatic fever without evidence of valvular damage or disturbance in rhythm, (2) the effects of rheumatic fever with deformation of the valves, and (3) the effects of rheumatic fever on the velocity of blood flow when, in addition to the valvular deformity, fibrillation of the auricles is present.

A consideration of the foregoing data emphasizes the importance of myocardial damage since the velocity of blood flow through the lungs may be seriously slowed even without clinical evidence of valvular damage. On the other hand, if the myocardium is less affected the velocity of blood flow may be normal though the mitral or aortic valves are seriously damaged.

The two patients of group A typify the cardiovascular effects of rheumatic fever in the absence of valvular damage or disturbance in rhythm. The somewhat increased pulmonary blood velocity in patient F. D. (no. 292) corresponds to our findings in the arm to arm blood velocity soon after the clinical subsidence of the rheumatic infection but before evidence of valvular damage appears. As was then pointed out, (1) the somewhat increased velocity of the blood stream conforms to the other clinical evidences of cardiac hyperactivity such as forcible precordial pulsation, rapid ventricular rate, and flushed skin. Whether such patients always manifest myocardial damage later cannot be stated from our observations, although clinical experience suggests that the heart muscle may frequently escape any evident change. According to the findings in patient G. (no. 267) the myocardium

may be severely damaged without evidence of valvular deformity for, although the peripheral flow from the arm to the right auricle was within the limits of normal, the velocity of blood flow through the lungs was greatly slowed. The vital capacity was likewise reduced while the venous pressure was within normal limits.

The findings in the patients of group B indicate that generally myocardial involvement is less than that in patient G. (no. 267), for in seven of the eight patients studied, in spite of the additional work demanded of the myocardium because of valvular deformity, the heart muscle was able to maintain a velocity of pulmonary blood flow within the limits of normal.

The patients of group C showed fibrillation of the auricles in addition to the valvular damage, and as might have been expected, the reduction of the velocity of blood flow was in general greater. Whether this slowing is due to the disturbance in rhythm or whether such slowing simply tends to occur in the more severely damaged hearts will be discussed in a later communication.

The slowing of the blood flow through the lungs early in circulatory failure in rheumatic valvular heart disease may well be due to increased pressure in the pulmonary vessels. With insufficiency of the mitral value and cardiac insufficiency, Straub (6) has shown that the left intra-auricular pressure rises and, in some experiments, also the pressure in the pulmonary artery and right auricle. Under such circumstances an increased amount of blood may be accommodated in the readily distensible pulmonary vessels. The resulting increase in total cross sectional diameter of the flowing stream would tend to cause a reduction in velocity if unattended by a proportionate increase in the volume flow.

The first seven patients of group B showed the signs of mitral insufficiency, and, with the exceptions of K. N. (no. 287) and F. M. (no. 338), the signs of mitral stenosis. With regurgitation of part of the left ventricular contents at each systole and with narrowing of the mitral orifice, the filling of the ventricle by the left auricle is hindered. According to Kornfeld (7), the total quantity of the blood in the lungs is increased and the minute volume flow and blood velocity are diminished under such circumstances. Straub (8) working with a heart-lung preparation also observed a rise in the left intra-auricular pressure

but found no increase in the right auricular or intra-ventricular pressure. On the contrary, the maximal right ventricular pressure appeared to decrease. Similar results were obtained by Gerhardt (9), who believed that in compensated valvular lesions excess accumulation of blood is accommodated in the pulmonary veins.

The finding of normal pulmonary circulation times and arm to heart times in two patients, K. N. (no. 287) and F. M. (no. 338), who showed the clinical signs of mitral insufficiency and in five patients (nos. 376, 340, 363, 368, 283), who showed, in addition to mitral insufficiency, the signs of mitral stenosis, favors the hypothesis of Straub and Gerhardt that in man dilatation and hypertrophy of the left auricle accommodates the regurgitated blood and forces the increased amount through the narrowed mitral orifice without slowing the time of blood

TABLE 2

Averages of findings in patients of groups B and C with rheumatic valvular disease

	Regular rhythm	Auricular fibrillation
Arm to heart time	9 seconds	12 seconds
Pulmonary circulation time (crude)	12 seconds	26 seconds
Venous pressure	9 cm. water	14 cm. water
Vital capacity per square meter	2,068 cc.	1,704 cc.

flow. Only when the auricle becomes incompetent does engorgement of the pulmonary vessels occur with retardation in the pulmonary blood flow.

The general decrease in velocity of blood flow through the lungs in patients with auricular fibrillation (table 2), is probably due to several causes. The occurrence of auricular fibrillation may well be an expression of more profound myocardial damage. Although in arteriosclerotic patients with auricular fibrillation we have noted occasionally but slight reduction in the velocity of blood flow, blood can flow far more easily from auricle to ventricle in such patients than in those whose mitral valve is stenosed. In the latter, auricular systole may assume a rôle of greater importance than it does normally. With paralysis of the auricle, however, blood flow from auricle to ventricle depends solely on the difference in pressure. Under these circumstances and especially when the mitral orifice is narrowed and the

length of diastole is curtailed by the rapid and irregular ventricuar rate, adequate ventricular filling becomes difficult. The importance of the rôle of the auricle in mitral stenosis is evidenced by hypertrophy of the auricular muscle and of the right ventricle as well as by the accentuation of the pulmonic second sound.

II. THE PULMONARY CIRCULATION TIME, THE VELOCITY OF VENOUS BLOOD FLOW AND RELATED ASPECTS OF THE CIRCULATION IN PATIENTS WITH SYPHILITIC HEART DISEASE

Eleven measurements of the pulmonary circulation time and related aspects of the circulation were made in eight patients. The venous pressure of all the subjects in this group averaged 10.5 cm. of water (average normal, 7.3 cm.); the vital capacity, 1412 cc. per square meter of body surface (average normal, 2376 cc.); the crude pulmonary circulation time, 19.9 seconds (average normal, 10.8 seconds); and the arm to heart time, 10.0 seconds (average normal 6.6 seconds).

Practically all the patients complained of paroxysmal dyspnea and substernal pain, and it should be noted, that although the pulmonary circulation times were within the limits of normal in patients J. P. (no. 240), J. C. (nos. 276, 293, 325), P. T. (no. 326) and T. P. (no. 318), the blood flow through the lungs was slower than the normal average in all except J. P. (no. 240). The other four patients of this group all showed a more conspicuous slowing of the blood flow through the lungs and it should be observed that all had recently suffered congestive failure, or, at the time of test, showed definite physical signs of circulatory insufficiency. M. B. (no. 316), for example, complained of dyspnea on the slightest exertion, while patients A. S. (no. 271) and A. J. (no. 330) showed physical signs of passive congestion. The crude pulmonary circulation times of twenty-six and thirty-three seconds parallel the other clinical evidences of circulatory insufficiency. The findings in patient W. H. are of interest for the pulmonary circulation time was measured on two occasions when, according to physical examination, his condition was unchanged. At the time of the second test, September 22, 1926, he stated that he felt distinctly better than at the time of the first test, and his pulmonary circulation time was

TABLE 3

The pulmonary circulation time, the arm to heart time and their relation to other aspects of the circulation in patients with syphilitic heart disease

Test number	Date	Name	Age	Temperature	Pulse	Surface area	Venous pressure	Arterial pressure		Vital capacity	Vital capacity per square meter	Circulation time			Circulation time per square meter			Injected
								Systolic	Diastolic			Arm to heart	Pulmonary	Arm to arm	Arm to heart	Pulmonary	Arm to arm	
	1926					*square meters*	*cm. H_2O*	*mm. Hg.*	*mm. Hg.*	*cc.*	*cc.*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*millicuries*
240	September 2	J. P.			84	1.72	7.5	140	75	2,500	1,453	7.0	10.0	17.0	4.0	5.8	9.8	10.0
276	October 26	J. C.	52	98.8	70	1.92		136	0	2,700	1,406	8.0	14.0	22.0	4.1	7.2	11.3	5.0
293	November 4	J. C.	52	98.2	82	1.92		162	0	2,750	1,434	4.0	16.0	20.0	2.1	8.3	10.4	4.0
326	December 7	P. T.	64	98.8	74	1.58	2.5	162	64	2,400	1,519	7.5	16.0	23.5	4.7	10.1	14.8	6.0
318	November 23	T. P.	46	99.4	84	1.59	3.5			2,500	1,572	6.0	17.0	23.0	3.7	10.7	14.4	5.0
325	December 7	J. C.	52	98.7	72	1.92	1.5			1,925	1,005	8.0	18.0	26.0	4.2	9.4	13.6	
316	November 23	M. B.	55	98.0	98	1.77	9.5	172	0			5.5	19.5	25.0	3.1	11.0	14.1	9.0
243	September 22	W. H.	54	98.0	88	1.62	−4.0	110	40	3,000	1,852	14.0	22.0	36.0	8.6	13.5	22.1	5.4
271	October 21	A. S.	57	95.0	89	1.58	5.0			2,300	1,455	6.0	26.0	32.0	3.7	16.4	20.1	5.0
239	September 2	W. H.	54		88	1.66						16.0	27.0	43.0	9.6	16.2	25.8	
330	December 14	A. J.	56	95.8	86	1.78	15.5	165	65	1,800	1,011	30.0	33.0	63.0	16.8	18.5	35.3	8.5

twenty-two seconds, that it to say, five seconds shorter. Tests were performed on three different occasions on patient J. C. (no. 276, 293, 325). At no time did he show clinical signs of passive congestion although he stated that he felt somewhat more dyspneic at the time of the third test, when his crude pulmonary time was eighteen seconds (four seconds longer than the first) and his vital capacity was 1005 cc. per square meter of body surface against 1406 cc. and 1434 cc. at the earlier tests.

Discussion

The decrease in the velocity of blood flow through the lungs in patients suffering from syphilitic heart disease parallels the clinical evidences of circulatory failure but the slowing of the blood stream may not be as great as that observed in patients with rheumatic heart disease and an apparently similar degree of cardiac failure. The vital capacity in practically all subjects was considerably reduced, averaging 1378 cc. per square meter of body surface. Whether this reduction in vital capacity is due to engorgement or whether it is due in part to reflex spasm, such as in bronchial asthma, cannot be stated from the available data.

The venous pressure averaged 9.9 cm. of water in the six of the eight patients measured. Although this average is higher than the normal of 7.3 cm., such a small difference is probably not of any significance because of the much greater variability of the venous pressure as compared to the other measurements.

It is worthy of note that even with slowing of the blood flow through the lungs and with reduction in the vital capacity, the peripheral venous blood flow may be well within the limits of normal according to the measurement of the venous pressure and the velocity of the venous blood from the right antecubital vein to the right auricle. Excepting W. H. (no. 243, 239) who had suffered congestive failure shortly previous to his tests, and patient A. J., who had slight pitting edema on the day of the test, the arm to heart times averaged 6.5 seconds (average normal, 6.6. seconds), while the crude pulmonary circulation times averaged 17.0 seconds (average normal, 10.8 seconds). The absence of peripheral edema associated with normal arm to heart times suggests that the difference between so-called "dry heart failure" and "congestive heart failure" may be due to differences in the velocity of the

peripheral blood stream. This relation between the arm to heart time and the appearance of peripheral edema is not clear in every instance nor is a close correspondence to be expected since the arm to heart time is an index of the speed of the venous blood flow of the arm while the signs of peripheral edema characteristically appear elsewhere.

The occurrence of normal arm to heart times in aortic insufficiency is likewise evidence of the relatively late appearance of failure of the right chambers of the heart in this form of valvular defect. It is of interest to observe (table 1) that although the pulmonary circulation time in patients with syphilitic heart disease and regular rhythm averaged five seconds more (17.0 seconds) than that of patients with rheumatic heart disease and regular rhythm, the arm to heart time averaged two seconds less. This average relative shortening of seven seconds in the arm to heart time is in harmony with general clinical experience. The left auricle and ventricle labor under a great handicap in aortic insufficiency and seem to give way sooner than the right chambers, which in the earlier stages, are still capable of receiving all the venous blood from the periphery and transferring it into the pulmonary vessels. This situation contrasts with the early strain of the right ventricle in mitral stenosis and insufficiency. Consequently peripheral stasis may occur in patients with mitral stenosis while the left ventricle is still functionally capable, whereas in patients with aortic insufficiency, peripheral stasis is a sign of failure of all the chambers of the heart. It is hardly surprising that when in syphilis the right chambers of the heart fail and peripheral edema appears, the slowing of the venous blood from the arm to the heart may be fully as great as that observed in patients with rheumatic valvular disease (patients W. H. (nos. 239, 243), A. J. (no. 330)). This consideration of events explains why pulmonary congestion and peripheral stasis occur so late in aortic insufficiency and why their occurrence is of such grave prognostic import.

III. THE PULMONARY CIRCULATION TIME, THE VELOCITY OF VENOUS BLOOD AND THEIR RELATION TO OTHER ASPECTS OF THE CIRCULATION IN PATIENTS WITH ARTERIOSCLEROSIS AND MYOCARDIAL DEGENERATION

The pulmonary circulation time, the velocity of venous blood from the antecubital vein to the right auricle, the venous and arterial pres-

TABLE 4

The pulmonary circulation time, the arm to heart time, and their relation to other aspects of the circulation in patients who, with evidence of arteriosclerosis and myocardial degeneration, showed regular rhythm

Test number	Date	Name	Age	Temperature	Pulse	Surface area	Venous pressure	Arterial pressure		Vital capacity	Vital capacity per square meter	Circulation time			Circulation time per square meter			Injected
								Systolic	Diastolic			Arm to heart	Pulmonary	Arm to arm	Arm to heart	Pulmonary	Arm to arm	
						square meters	*cm. H_2O*	*mm. Hg.*	*mm. Hg.*	*cc.*	*cc.*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*millicuries*
371	February 16, 1927	J. C.	58	99.2	84	1.87	4.0	142	90	3,800	2,021	5.5	10.0	15.5	2.9	5.4	8.3	6.5
332	December 14, 1926	N. B.	68	98.5	92	1.57	5.5	162	86	3,300	2,090	4.5	13.0	17.5	2.9	8.3	11.1	5.0
361	February 9, 1927	A. F.	60	98.2	72	1.68	−3.5	130	85	3,650	2,171	13.0	14.0	27.0	7.7	8.3	16.0	7.0
370	February 16, 1927	H. S.	65	98.6	74	1.32	4.0	130	70	2,100	1,591	4.0	14.5	18.5	3.0	11.0	14.0	8.0
372	February 16, 1927	J. H.	70	97.6	43	1.67	7.5	140	70	3,150	1,886	15.0	17.0	32.0	8.9	10.1	19.0	9.0
319	November 23, 1926	N. B.	68	97.8	82	1.57	6.0	176	82	3,000	1,910	5.0	18.0	23.0	3.2	11.5	14.7	
290	October 28, 1926	W. L.	60	99.0	72					3,000		6.0	19.0	25.0				
389	March 14, 1927	J. H.	70	97.4	38	1.67	2.0	122	68	3,100	1,856	11.0	21.0	32.0	6.6	12.6	19.2	8.0
242	September 22, 1926	J. B.	57	96.6	64	1.77	−7.0	118	80	3,750	2,118	14.0	25.0	39.0	7.9	14.1	22.0	4.6
295	November 4, 1926	M.	72	98.4	62	1.46	3.5			1,550	1,062	12.0	27.0	39.0	8.2	18.5	26.7	
241	September 22, 1926	D. M.	78	95.2	72	1.57	1.5	114	50	3,900	2,038	13.0	32.0	45.0	8.3	20.3	28.6	4.8
284	October 28, 1926	J. G.	60	97.2	100	1.57	3.5	125	90	2,000	1,273	18.0	33.0	51.0	11.4	21.0	32.4	4.5
360	February 9, 1927	M. H.	48	97.2	77	1.78	17.0	138	110	1,800	1,011	25.5	42.0	67.5	14.3	23.6	37.9	9.0
341	January 6, 1927	T. R.	47	96.8	82	1.91	17.5		110	1,000	523	22.5	45.5	68.0	11.8	23.8	35.6	8.0
312	November 19, 1926	J. G.	60	96.5	80	1.57	15.0	152	112	1,000	637	31.0	67.0	98.0	19.7	42.7	62.4	7.9

sures, the vital capacity of the lungs, and the clinical signs and symptoms were studied in sixteen patients with generalized arteriosclerosis and with myocardial degeneration (tables 4 and 8). The venous pressure averaged 10.0 cm. of water (average normal, 7.3 cm.); the vital capacity 1506 cc. per square meter of body surface (average normal, 2376 cc.); the arm to heart time, 13.0 seconds (average normal, 6.6 seconds); and the crude pulmonary circulation time, 26 seconds (average normal, 10.8 seconds). In two patients the circulatory tests were repeated on three different occasions; and in two patients, on two different occasions. The subjects are divided into

TABLE 5

Patients with signs of arteriosclerosis without history of cardiac failure and without signs of congestive failure

Test number	Vital capacity	Vital capacity per square meter	Pulmonary circulation time (crude)	Arm to heart time	Venous pressure
	cc.	*cc.*	*seconds*	*seconds*	*cm. H_2O*
371	3,800	2,021	10.0	5.5	4.0
332	3,300	2,090	13.0	4.5	5.5
370	2,100	1,591	14.5	4.0	4.0
319	3,000	1,910	18.0	5.0	6.0
242	3,750	2,118	25.0	14.0	−7.0
Average.....	3,190	1,946	16.1	6.6	2.4

two groups; one, showing regular ventricular rhythm, the other, totally irregular rhythm.

A. Patients with regular ventricular rhythm (table 4)

As has been pointed out in a previous communcation, the study of patients who show circulatory insufficiency in the absence of valvular damage and arrhythmia provides an excellent opportunity to observe the practically uncomplicated effect on the circulation of but one factor, myocardial weakness. Circulatory tests were made on fifteen patients who showed regular ventricular rhythm. In two, the measurements were repeated once (table 4). These patients fall into three groups.

Group 1 (table 5). Of five tests on patients with arteriosclerosis,

but without history of cardiac failure in whom the circulation was compensated on physical examination; two of the tests (nos. 332 and 319), were on the same individual, several weeks apart. In general, this group showed a vital capacity 15 per cent less, and a crude pulmonary circulation time 49 per cent greater than the average normal value found by us in normal individuals, while the venous pressure and arm to heart time were normal.

TABLE 6

Patients complaining of dyspnea on exertion but having no signs of congestive failure on physical examination at time of test

Test number	Vital capacity	Vital capacity per square meter	Pulmonary circulation time (crude)	Arm to heart time	Venous pressure
	cc.	*cc.*	*seconds*	*seconds*	*cm. H_2O*
361	3,650	2,171	14.0	13.0	−3.5
272	3,150	1,886	17.0	15.0	7.5
389	3,100	1,856	21.0	11.0	2.0
295	1,550	1,062	27.0	12.0	3.5
241	3,900	2,038	32.0	13.0	1.5
Average.....	3,070	1,803	22.2	12.2	2.2

TABLE 7

Patients who showed at time of test signs of congestive failure

Test number	Vital capacity	Vital capacity per square meter	Pulmonary circulation time (crude)	Arm to heart time	Venous pressure
	cc.	*cc.*	*seconds*	*seconds*	*cm. H_2O*
284	2,000	1,273	33.0	18.0	3.5
360	1,800	1,011	42.0	25.5	17.0
341	1,000	523	45.0	22.5	17.5
312	1,000	637	67.0	31.0	15.0
Average.....	1,450	861	46.7	24.2	13.2

Group 2 (table 6). This group showed no signs of decompensation at the time of test but had previously experienced symptoms of circulatory insufficiency. The vital capacity was 24 per cent less than the normal; the crude pulmonary circulation time was 105 per cent, and the arm to heart time 84 per cent greater than the normal, while the venous pressure coincided with the normal average.

Group 3 (*table 7*). The group with signs of congestive failure at the time of the test had a vital capacity 36 per cent of the normal, while the crude pulmonary circulation time and the arm to heart time indicated a slowing of the blood flow to approximately one-fourth normal speed. The subjects of these tests were of advanced years and so the low vital capacity measurements may reflect the presence of pulmonary emphysema as well as of circulatory failure.

In all patients with arteriosclerosis, pulmonary circulation and vital capacity measurements showed departures from normal values that generally paralleled the clinical symptoms and signs, although this relation did not necessarily hold in each instance. In test 241, for example, the vital capacity was 2036 cc. per square meter of body surface, while the pulmonary crude circulation time was 32.0 seconds.

The relation between the appearance of edema and the slowing of the peripheral blood stream indicated by the arm to heart time is interesting. The patients of group 2 without edema had an arm to heart time of fifteen seconds or less, while the patients of group 3 with edema showed arm to heart times of eighteen seconds or more. This suggests, as has been stated, that the appearance of edema is associated with slowing of the peripheral blood stream and that the difference between so-called "dry" and "congestive" heart failure may be due to differences in the speed of the peripheral blood flow. The moderately prolonged times found in group 2, associated with dyspnea but without physical signs of congestive failure, may denote a greater collection of blood in, and dilatation of, the pulmonary vessels, just as occurs in the peripheral veins before the pressure begins to rise. The normal venous pressure associated with abnormally slow velocity of venous blood flow and the decrease in the vital capacity conform to this possibility. The appearance of edema probably does not coincide with a definite degree of slowing of the blood stream for it is also dependent on physicochemical changes which themselves are secondary to diminished blood flow and which are influenced by many other factors.

B. Patients with fibrillation of the auricles (*table 8*)

Of the patients who, in addition to signs of generalized arteriosclerosis and myocardial degeneration, showed complete ventricular arrhythmia, all had previously suffered congestive failure or showed such signs

TABLE 8

The pulmonary circulation time, the arm to heart time, and their relation to other aspects of the circulation in patients who with evidence of arteriosclerosis and myocardial degeneration, showed fibrillation of the auricles

Test number	Date	Name	Age	Temperature	Pulse	Surface area	Venous pressure	Arterial pressure		Vital capacity	Vital capacity per square meter	Circulation time			Circulation time per square meter			Injected
								Systolic	Diastolic			Arm to heart	Pulmonary	Arm to arm	Arm to heart	Pulmonary	Arm to arm	
						square meters	*cm. H_2O*	*mm. Hg.*	*mm. Hg.*	*cc.*	*cc.*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*millicuries*
362	February 9, 1927	C.	66	98.6	77	1.94	5.0	132	88			5.0	15.5	20.5	2.6	7.9	10.5	8.0
411	April 20, 1927	M. M.	49	97.2	53	1.92	3.5	144	82	4,200	2,189	11.0	16.0	27.0	5.7	8.3	14.0	8.0
261	September 30, 1926	W. D.	55	97.4	74	1.65	10.0					15.0	27.0	42.0	9.1	16.3	25.4	
246	September 24, 1926	W. D.	55	98.0	42	1.65	3.0	94	65	2,100	1,272	21.0	34.0	55.0	12.7	20.6	33.3	7.8
247	September 24, 1926	F. B.	65	97.6	64	1.60	−3.0	126–114	64–60	1,100	687	7.0	35.0	42.0	4.3	21.8	26.1	3.4

at the time of test. The average vital capacity was 2230 cc. which when reduced to cubic centimeters per square meter of body surface was 1580 cc. (average normal, 2376 cc.). The average crude pulmonary circulation time was 25.6 seconds (average normal, 10.8); the average arm to heart time was 12.1 seconds (average normal, 6.6); the average venous pressure 9.0 cm. (average normal, 7.3 cm.). These four patients did not show as severe circulatory insufficiency as those patients with regular rhythm that we happened to study. This general finding as well as the results of test numbers 362 and 411 demonstrate that the velocity of blood flow may be maintained at a speed within the upper limits of normal in spite of the abnormal mechanism of auricular fibrillation.

IV. THE PULMONARY CIRCULATION TIME, THE VELOCITY OF VENOUS BLOOD TO THE HEART, AND RELATED ASPECTS OF THE CIRCULATION IN PATIENTS WITH ARTERIAL HYPERTENSION

In these patients (table 9), the venous pressure averaged 14 cm. of water or 92 per cent greater than the normal average; the vital capacity 1784 cc. per square meter of body surface or 25 per cent less than the normal; the arm to heart time 9.7 seconds or 47 per cent greater than the normal; and the crude pulmonary circulation time 15.3 seconds or 42 per cent greater than the normal. The patients have been divided into three groups.

Group A. These patients exhibited no evidence of circulatory failure. The velocity of blood flow through the lungs was within the limits of normal although the general average, 12.0 seconds, was slightly higher than that shown by a larger series of normal individuals (10.8 seconds). It may not be without significance that patient T. L. who showed the most rapid velocity of blood flow was likewise the person whose blood pressure was the lowest.

Group B. The distinct retardation in pulmonary blood velocity shown by patients in this group in the absence of signs or symptoms of circulatory insufficiency at rest or on exertion, as well as the tendency toward slowing in the subjects in group A, may be related to back pressure effects of arterial hypertension on the pressure within the pulmonary vessels. There is experimental evidence for this idea. Cloetta and Staubli (11) found that compression of the thoracic aorta always

TABLE 9

The pulmonary circulation time, the arm to heart time and their relation to other aspects of the circulation in patients with arterial hypertension

Test number	Date	Name	Age	Temperature	Pulse	Surface area	Venous pressure	Arterial pressure		Vital capacity	Vital capacity per square meter	Circulation time			Circulation time per square meter			Injected
								Systolic	Diastolic			Arm to heart	Pulmonary	Arm to arm	Arm to heart	Pulmonary	Arm to arm	
Group A. Patients with compensated circulation at time of test, in whom the pulmonary circulation time was within normal limits																		
						square meters	*cm. H_2O*	*mm. Hg.*	*mm. Hg.*	*cc.*	*cc.*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*seconds*	*millicuries*
377	February 23, 1927	T. L.	47	97.0	76	1.81	5.0	155	100	4,000	2,210	7.0	7.5	14.5	3.9	4.1	8.0	7.0
272	October 21, 1926	E. M.	46	98.2	82	1.81	8.0	184	110	3,800	2,099	11.0	11.0	22.0	6.0	6.0	12.0	5.0
307	November 6, 1926	B. N.	57	98.1	80	1.77	10.5	180	102	3,500	1,980	3.0	12.0	15.0	1.7	6.7	8.4	4.5
331	December 14, 1926	F. S.	41	98.4	82	1.78	7.0	200	110	3,700	2,021	6.0	12.0	18.0	3.4	6.7	10.1	5.0
357	February 9, 1927	G. M.	52	98.0	60	1.78	5.5	248	128	3,400	1,910	6.0	12.0	18.0	3.4	6.7	10.1	8.0
336	January 6, 1927	A. O.	68	98.6	78	1.81	7.0	210	115	3,250	1,785	10.0	13.0	23.0	5.5	7.2	12.7	6.0
309	November 6, 1926	J. M.		98.6	58	1.79	8.5	205	104	3,650	2,039	12.0	14.0	26.0	6.7	7.9	14.6	4.0
305	November 6, 1926	H. M.	70	98.1	90	1.74	20.0	194	104	2,700	1,552	4.0	14.5	18.5	2.3	8.3	10.6	7.0
Group B. Patients with compensated circulation in whom the pulmonary circulation time was prolonged																		
300	November 5, 1926	M. P.	50	99.2	92	1.60	12.5	180	95	4,000	2,500	15.0	18.0	33.0	9.4	11.2	20.6	4.0
308	November 6, 1926	M. C.	57	97.2	66	1.86	8.5	192	116	4,400	2,368	12.0	19.0	31.0	6.4	10.2	16.6	4.0
304	November 6, 1926	M. S.	61	98.2	76	1.76	6.5	190	112	2,900	1,648	7.0	23.0	30.0	4.0	12.2	16.2	
296	November 4, 1926	M. P.	50	98.4	94	1.60	13.5	214	110	4,100	2,560	6.0	23.5	29.5	3.7	14.7	18.4	6.5
Group C. Patients with decompensated circulation in whom the velocity of blood flow was prolonged																		
429	May 21, 1927	C. B.	41	98.4	82	1.73	6.5	200	140	1,200	694	6.5	16.0	22.5	3.7	9.3	13.0	9.5
423	May 12, 1927	P. F.	63	98.8	81	1.96	4.5	210	120	3,500	1,786	8.0	21.0	29.0	4.1	10.7	14.8	7.0
427	May 18, 1927	C. B.	41	98.6	118	1.82	21.0	195	160			22.0	22.0	44.0	12.0	12.0	24.0	9.0
278	October 26, 1926	J. G.	53	97.2	130	1.82	9.0	165	110	2,800	1,538	10.0	24.0	34.0	5.4	13.1	18.5	3.5
334	December 14, 1926	E. A.	58	98.2	72	1.95	4.5	185	105	2,600	1,335	16.0	25.0	41.0	8.2	12.8	21.0	9.0

caused an increased lung volume, and Straub (8) and also Gerhardt, (9), likewise observed that increased arterial pressure in the greater circulation produced an increase in the volume of the lesser circulation. Such an increase in the amount of blood in the lungs would lead to increased cross sectional diameter of the stream of blood flowing through the lungs. Slowing in blood flow with prolongation of the pulmonary circulation time must then occur since the velocity of flow is inversely proportional to the cross sectional diameter of a stream. Observations by Wearn, Barr and German (12) are in accord with this hypothesis, for they observed in animals that slight compression of the abdominal aorta caused considerable dilatation of alveolar capillaries. With such an increase in the amount of blood in the lungs a decrease in the vital capacity might also be expected because of diminished lung elasticity. Although the vital capacities of the patients in groups A and B averaged 2055 cc. per square meter or 86 per cent of the normal, interpretation is ambiguous because of the advanced years of most of the patients.

We emphasize these findings because they constitute the only instance in which the circulation was functionally competent in the presence of retardation in the pulmonary blood flow velocity and because the mechanism by which this slowing is produced is of considerable physiological interest.

As in the preceding study of the arm to arm velocity of blood flow, we have found that the velocity of blood flow was never greater than the normal. This indicates that increased blood pressure, which in itself would tend to increase the speed of flow, is opposed by such factors as increased peripheral resistance. If cardiac hyperactivity were primary, one would expect to find a stage in which the peripheral resistance had not as yet increased and in which the blood flow was abnormally rapid.

Group C. consists of patients in whom the slowing of the blood flow was associated with the symptoms or signs of circulatory failure. This finding is in accord with our experience with other patients suffering from circulatory failure with a normal blood pressure. The degree of slowing in blood flow was approximately that observed in patients with cardiovascular failure of other etiology.

The venous pressures of patients with hypertension showed extreme variability with no evident relationship to the degree of passive con-

gestion. In some patients such as B. N. (no. 307), H. M. (no. 305), M. P. (nos. 296, 300), the elevation of the venous pressure was of a degree usually associated with chronic passive congestion, whereas these patients were free from such signs or symptoms. There was no relation between the venous blood velocity and the venous pressure. In H. M. (no. 305), for example, the venous pressure was equivalent to 25 cm. of water, the arm to heart time was 4 seconds; whereas in M. P. (no. 300) the venous pressure was equivalent to 17.5 cm. of water while the arm to heart time was 15 seconds. These variations in venous pressure, unrelated as they are to changes in the velocity of blood flow, may be an expression of the vasomotor instability and hyperirritability recognized by clinicians. That an increased venous pressure may be associated with arterial hypertension in the absence of circulatory failure has been observed by others (13) (14) (15).

It may be thought that because of vasomotor instability patients in group A might at times show the more prolonged circulation times of those in group B. It is of interest that M. P., in whom the velocity of blood flow was measured twice, showed on both occasions prolonged circulation times.

Summary and Conclusions

1. Sixty-three measurements of the pulmonary circulation time, of the arm to heart time, of the venous and arterial blood pressures, and of the vital capacity of the lungs have been made in fifty-four male patients with cardiovascular disease (rheumatic, syphilitic, arteriosclerotic, arterial hypertension) and their relation to the clinical findings have been studied.

2. The clinical and physiological significance of these observations is discussed.

3. The methods as described in a preceding communication have been found adequate for the study of the clinical aspects of cardiovascular disease.

I. Patients with rheumatic heart disease

A. Without valvular damage

1. Measurements were made on two patients, who showed the immediate and the very late effects of myocardial involvement subsequent to acute rheumatic fever.

2. In one patient, a young adult, the symptoms and signs of severe rheumatic myocardial damage were associated with moderate slowing of the pulmonary blood flow while the peripheral blood flow from the arm to the heart was within normal limits.

3. The other patient showed a slightly increased velocity of blood flow with clinical evidences of exaggerated cardiac activity following acute rheumatic fever.

B. With valvular damage and regular rhythm

1. Normal speed of blood flow through the lungs and from the arm to the heart demonstrates that in spite of valvular deformation, the myocardium may be capable of maintaining a normal velocity of blood flow.

2. In the one patient who was dyspneic at the time of test, but who showed no signs of congestive failure, the pulmonary blood flow was slightly slower than the normal.

3. These observations indicate that the slowing of blood flow in rheumatic heart disease reflects the dysfunction due to myocardial damage.

4. There is no close relationship between the degree of valvular involvement and the degree of circulatory competence as reflected by the velocity of blood flow.

C. With valvular damage and fibrillation of the auricles

1. The velocity of pulmonary blood flow was slowed and bore a definite relationship to the degree of circulatory insufficiency. The peripheral blood flow, as judged by the arm to heart time, while in general retarded, was not so closely related to the clinical findings.

II. PATIENTS WITH SYPHILITIC HEART DISEASE

1. The decrease in the velocity of blood flow through the lungs paralleled the clinical evidences of circulatory failure except that paroxysmal dyspnea and precordial pain did not seem to be associated with quite as much slowing of the blood stream as that observed in patients with rheumatic heart disease. This suggests that paroxysmal breathlessness and pain in patients with syphilitic aortitis may be due in part to a reflex mechanism.

2. Even with slowing of the blood flow through the lungs and with reduction in the vital capacity of the lungs, the peripheral blood flow was found within the limits of normal according to the measurement of the venous pressure and of the arm to heart time. This is in harmony with the late appearance of edema in aortic insufficiency.

3. Our findings suggest that the differences between so-called dry heart failure and congestive heart failure may well be due to differences in the velocity of the peripheral blood stream.

III. PATIENTS WITH ARTERIOSCLEROSIS AND WITH EVIDENCES OF MYOCARDIAL DEGENERATION

A. With regular rhythm.

1. Of the patients in this group in whom the arm to heart time was fifteen seconds or less, none showed evidence of peripheral edema, while all patients who showed arm to heart times above this, showed signs of peripheral edema.

2. The velocity of blood flow through the lungs paralleled in general the degree of circulatory incompetence and as such provided an objective and quantitative index of the circulation.

B. With fibrillation of the auricles

In two patients the velocity of blood flow through the lungs was within the upper limits of normal in spite of the abnormal mechanism of auricular fibrillation, whereas the velocity of pulmonary blood flow was moderately or greatly reduced in the two others according to the degree of circulatory failure.

IV. PATIENTS WITH ARTERIAL HYPERTENSION

1. Patients with arterial hypertension who show no evidence of circulatory disability may be divided into two groups: in one, the pulmonary velocity of blood flow is within the limits of normal, whereas in the other it is retarded.

2. The arm to heart times in these subjects bore no constant relation to the pulmonary circulation time.

3. In no patients with hypertension was an abnormally rapid velocity of blood flow observed. This suggests that the fundamental

disturbance in arterial hypertension is increased peripheral resistance rather than cardiac hyperactivity.

4. As in a previous study, we observed in some patients an abnormally high venous pressure in the absence of congestive failure.

5. Patients with arterial hypertension and with congestive failure show a retardation in the pulmonary and peripheral blood flow similar to that in patients with a corresponding degree of circulatory failure but with a normal rhythm.

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ABSTRACTS OF HISTORIES AND PHYSICAL EXAMINATIONS

I. Patients with Rheumatic Heart Disease

292. F. D. Two months previously there had been swelling of the right wrist, spontaneously subsiding. He had had a similar attack seven years previously. One week previous to entry red, tender, swelling of right wrist and right elbow appeared. Physical Examination was entirely negative at time of test. The diagnosis was acute rheumatic fever.

267. G. had had numerous attacks of tonsilitis in the past but not rheumatic fever. Ten months previously he noticed gradually increasing dyspnea and finally orthopnea, forcing him to enter the hospital. Physical examination at that time showed cyanosis, orthopnea, a moderately enlarged heart with a soft systolic but no diastolic murmur. The ventricular rate was about forty and electrocardiographic tracings showed the presence of partial heart block. The consensus of opinion was that the patient was suffering from rheumatic myocarditis. He improved and left the hospital but returned because of recurrence of the previous orthopnea. Physical examination showed the apex impulse in the fifth interspace with the left border of dulness 12 cm. from the midsternal line. The rhythm was regular, the lungs were clear and the liver was not palpable. Squeaking rhonchi were heard over both chests posteriorly. There was no edema of the ankles. The diagnosis was rheumatic myocarditis.

376. A. C. entered the hospital for tonsillectomy. He had had an attack of acute rheumatic fever one year previously but had never suffered from circulatory insufficiency. Physical examination was normal save for a slightly enlarged heart with a systolic and rough presystolic murmur over the mitral area. The diagnosis was rheumatic heart disease; mitral stenosis and insufficiency.

287. K. N. entered the hospital because of pain and swelling of both knees.

During the previous five weeks he had had acute rheumatic fever which responded to treatment by salicylates. At time of test there were no joint signs or symptoms. Physical examination: Heart: Left border of dulness 11.5 cm. from midsternal line in fifth space. First sound was accentuated. Soft systolic murmur at the apex was transmitted to the axilla. There was no diastolic murmur. The diagnosis was rheumatic heart disease and mitral insufficiency.

338. F. MacD. No reliable history was obtainable. There was a loud booming first sound over the apex followed by a rough systolic murmur transmitted to axilla. The heart was slightly enlarged. There were no signs of congestive failure. The diagnosis was rheumatic heart disease and mitral insufficiency.

340. J. G. entered the hospital complaining of weakness and palpitation three months in duration. He had had acute rheumatic fever in childhood but had never experienced shortness of breath on exertion or other signs or symptoms of congestive circulatory failure. Physical examination was negative save for slight cardiac enlargement and the signs of mitral stenosis and insufficiency. The diagnosis was mitral stenosis and insufficiency.

363. R. F. entered the hospital complaining of painful joints. He had had several attacks of acute rheumatic fever but had never experienced dyspnea except on walking up one flight of stairs. Physical examination was negative save for a moderately enlarged heart, accentuation of the second pulmonic sound, a loud blowing systolic and a rough presystolic murmur over the mitral area and a loud first sound. There was no past or present evidence of congestive failure. The diagnosis was mitral stenosis and insufficiency.

368. M. E. entered the hospital complaining of painful swollen joints, fever and palpitation. He had never experienced symptoms of circulatory decompensation. At time of test, fourteen days later, the heart was found slightly enlarged and a distinct diastolic and systolic murmur was heard over the apex. The joints were normal. There were no signs of circulatory insufficiency. The diagnosis was acute rheumatic fever, mitral stenosis and insufficiency.

283. W. O. had had rheumatic fever 28 years and 18 years previously. At time of insurance examination 18 years previously he was told he had aortic valvular disease. He never experienced any symptoms referable to the circulatory system and entered the hospital because of acute alcoholic intoxication. He was able to lead a vigorous, normal life. Physical examination showed a well developed man with conspicuous arterial pulsations in neck vessels. Heart was moderately enlarged, with the apex 14 cm. to the left of the midsternal line in the fifth space. Apex impulse was heaving. There were no thrills. At apex, first sound was rough and loud, second sound accentuated. Short, rough presystolic murmur and soft systolic and diastolic murmur were heard. Loud, long diastolic murmur heard along the left border of sternum and faint diastolic murmur, over the aortic area. Corrigan pulse and Duroziez's sign were present. The diagnosis was mitral stenosis and regurgitation, and aortic regurgitation.

369. P. G. entered hospital complaining of substernal pain. He had had rheumatic heart disease for eighteen years. Day before entry, a to and fro friction

rub had been heard to left of sternum. He had occasionally experienced shortness of breath on exertion but never noted swelling of legs. Physical examination showed the heart moderately enlarged, the rhythm regular, and the physical signs of mitral stenosis and insufficiency. There were no signs of congestive circulatory failure. The diagnosis was probable acute pericarditis, mitral stenosis and insufficiency.

258. D. S. had had shortness of breath, attacks of sharp lancinating, non-radiating pain over the heart for several years, which followed exertion, and which lasted a minute or two. He recently had experienced marked orthopnea. One week before entry he coughed up blood-streaked sputum. Physical examination showed left border of cardiac dulness 12 cm. The apex impulse was felt in the 5th and 6th interspaces. The cardiac rhythm was totally irregular. Double murmurs were present over the apex and over the aortic area. Râles were heard over the lungs. There was edema of both ankles. Hemoglobin was 75 per cent. The diagnosis was auricular fibrillation, mitral stenosis and insufficiency.

324, 265. S. C. complained of shortness of breath. He had had rheumatic fever in childhood but had been well until 9 years previously, when, after pneumonia, he developed moderate shortness of breath for 8 months. During the 10 months before entry he experienced slight precordial pain on exertion with shortness of breath and palpitation, which gradually increased in severity forcing him to enter the hospital. Three days before admission, pitting edema was observed over lower legs. After admission to the hospital, on rest in bed and digitalis, he showed moderate improvement. At time of test, 265, he was still slightly orthopneic. Physical examination showed blowing systolic and diastolic murmurs over aortic and mitral areas. The liver was not palpable. There was no edema of the legs. A few moist râles were heard over the left base. Following rest in the hospital patient returned home but was compelled to re-enter hospital because of exacerbation of symptoms. At time of test no. 324 patient felt well and was up and about the ward. There was no dyspnea or orthopnea. Physical examination showed no râles over chest and no edema. The liver was not palpable, and the heart was as noted above. The diagnosis was rheumatic pericarditis; auricular fibrillation, aortic stenosis and insufficiency, mitral stenosis and insufficiency.

406. E. G. entered hospital two weeks previous to test severely decompensated, but on rest in bed and digitalis he improved so that he was but slightly dyspneic at time of test, and was troubled only by a slight cough. Physical examination showed moderately enlarged heart, with the signs of mitral stenosis and insufficiency, total irregularity of the rhythm, signs of fluid over the base of the right lung and slight but definite pitting edema over the ankles. The diagnosis was mitral stenosis and insufficiency and auricular fibrillation.

358. H. M. entered hospital complaining of shortness of breath on the slightest exertion, six weeks in duration. He had no history of rheumatic fever but had had frequent sore throats. He noticed irregularity of heart action two years previously

which had since persisted. Dyspnea had appeared three months previously and had gradually become worse. Physical examination showed moderate cardiac enlargement and the signs of mitral stenosis and insufficiency with total irregularity of the cardiac rhythm. Percussion note was dull over the right chest posteriorly. There were no râles. Tender liver edge was palpable three fingers breadth below the right costal margin. The diagnosis was circulatory insufficiency, auricular fibrillation, mitral stenosis and insufficiency.

II. Patients with Syphilitic Heart Disease

240. J. P. had had occasional slight pain below manubrium with attacks of shortness of breath. He felt weak and was unable to do hard labor. There was no history of congestive failure. Physical examination was negative except for a systolic murmur over the base. By x-rays, aneurysm of the aortic arch was observed. The diagnosis was aneurysm of the aortic arch.

325, 293, 276. J. C. had had shortness of breath, tired feeling, and nocturnal attacks of dyspnea and wheezing. There was no history of congestive failure. Dyspnea was unusually severe on the slightest exertion. Physical examination at time of first test showed marked arterial pulsations visible in the neck, a heaving apex impulse over the fifth space, left border of cardiac dulness 12.5 cm. from the midsternal line, systolic and diastolic murmurs over the apex and over the aortic area. The systolic murmur was transmitted into the vessels of the neck. Corrigan pulse was present. The lungs were clear, the liver was not palpable. Wassermann was positive at time of second test, no. 293. Patient improved subjectively, and was able to walk about without shortness of breath. Physical examination was as noted before. At time of the third test, he had dyspnea on but slight exertion. He believed himself definitely worse than at time of previous tests. Physical examination was as before and showed no signs of congestive failure. The diagnosis was aortic insufficiency and syphilis.

326. P. T. entered the hospital complaining of paroxysmal dyspnea one year in duration. The attacks came mostly at night, were very severe, were accompanied by a choking sensation, lasted several hours and were not attended by pain. He had no swelling of the legs at any time. He had been working and when attacks were absent he felt normal. Physical examination showed left border of cardiac dulness 15 cm. from the midsternal line in the sixth interspace, double murmur over aortic area, Corrigan pulse. X-ray showed dilatation of the arch of aorta. The vessels were sclerosed and tortuous. Kahn test was negative. The diagnosis was syphilis, aortic insufficiency, and aneurysm of aorta.

318. T. P. entered the hospital complaining of paroxysmal attacks of dyspnea, the attacks lasting for about a half an hour and at times accompanied by sensation of tightness over the chest. Attacks occur especially on excitement and on exercise. Physical examination showed a moderately enlarged heart with the signs of aortic insufficiency, and no signs of congestive failure. The diagnosis was syphilis, and aortic insufficiency.

316. M. B. entered the hospital complaining of shortness of breath. Although

he had suffered congestive failure in the past, and at the time of the test he was able to be up and about, he still complained of dyspnea on slight exertion. Physical examination showed a greatly enlarged heart with the signs of aortic insufficiency. The peripheral signs of aortic insufficiency were also present. No signs of congestive failure were present at the time of the test. The diagnosis was syphilis, and aortic insufficiency. Subsequent history: The patient died three and a half months later. He had shown signs of subacute bacterial endocarditis in the interval, as well as more conspicuous attacks of nocturnal paroxysmal dyspnea.

239, 243. W. H. had had for one year progressively increasing dyspnea, marked at night, and increasing weakness and cough for one month, and orthopnea for 2 weeks. Physical examination at the time of admission, one month before test no. 239, showed orthopnea, the apex of the heart in 6th space, left border of cardiac dulness 13 cm. from midsternal line, and double murmurs over the aortic area. At the time of test, no. 239, there was orthopnea, no congestive failure and he was able to walk slowly on the level without becoming dyspneic. At the time of test, no. 243, his circulation was compensated fairly well at rest and on slight exertion. He felt definitely stronger. The diagnosis was aortic insufficency and syphilis.

271. A. S. entered the hospital because of dyspnea and sharp, non-radiating pain in the right upper quadrant of five weeks duration. He noted swelling of the legs, and was troubled by cough. He was forced to use two or three pillows at night, but rapidly improved under rest and digitalis. Physical examination showed the sclerae slightly jaundiced and the heart in fifth space, 13 cm. from midsternal line. Double murmur was heard over the aortic area and the tender edge of the liver was felt three fingers below the costal margin. There was no edema of the legs. The lungs were normal. He could walk on the level without stopping. Kahn test was positive. Fluoroscopy showed aneurysm of the ascending aorta. The diagnosis was aneurysm of ascending portion of the arch of aorta.

330. A. J. entered the hospital complaining of nocturnal dyspnea of fourteen months duration, and of dyspnea on the slightest exertion of four months duration. He had never suffered congestive failure. Physical examination at time of entry to hospital showed no signs of chronic passive congestion. The heart was moderately enlarged and there was a double murmur heard over the aortic area. The pulse was of the water-hammer type and Duroziez's sign was present. One week after entry, on the day of the test, he showed, for the first time, slight pitting edema of feet and ankles. Three days later, coincident with increasing congestive failure, he developed attacks of excruciating pain in the right upper quadrant. He became rapidly worse and died three days later. Post-mortem examination showed a heart weighing 700 grams, several old grayish scars in the myocardium and openings of the coronary arteries nearly obliterated by atheromatous changes in the aorta, which showed the characteristic signs of syphilitic involvement. The diagnosis was syphilis and aortic insufficiency.

III. Patients with Arteriosclerosis and Evidences of Myocardial Degeneration

A. With regular rhythm

371. J. C. entered the hospital on the surgical service where prostatectomy was performed. At time of test he complained of slight weakness. Physical examination was negative except for moderate tortuosity and sclerosis of peripheral vessels. The diagnosis was arteriosclerosis.

319, 332. N. B. entered the hospital because of osteomyelitis of the right first metatarsal bone. There was no history of circulatory embarrassment. Physical examination showed no signs of circulatory insufficiency, heart was normal in size, and tortuous and sclerosed brachial and temporal arteries. Urine was normal. The diagnosis was generalized arteriosclerosis.

361. A. F. entered hospital complaining of a painful left ankle which gradually became swollen, especially in morning after getting up. For several months he experienced some dyspnea on exertion. Physical examination showed the heart normal in size, sounds of fair quality, no murmurs, conspicuous sclerosis of the radial and tibial arteries, but no signs of circulatory insufficiency. The diagnosis was generalized arteriosclerosis.

370. H. S. entered the hospital because of a fractured femur. A diagnosis of Paget's disease was made. He complained of slight dyspnea on exertion. The heart was slightly enlarged, the radial and other superficial arteries conspicuously sclerosed and tortuous. There were no signs of circulatory incompetence. The diagnosis was Paget's disease; generalized arteriosclerosis.

372, 389. J. H. collapsed while walking on street and entered hospital comatose. He gradually improved. He had been troubled with shortness of breath on exertion for several years. Physical examination showed the heart enlarged to the left, sounds fair, and a loud systolic murmur over the mitral area, no signs of circulatory insufficiency. Electrocardiographic tracings showed simple bradycardia. The diagnosis was bradycardia and generalized arteriosclerosis.

290. W. L. gave no history of congestive failure. Physical examination showed the heart normal in size. The sounds were of good quality and regular rhythm. Arteries were tortuous and thickened. The diagnosis was arteriosclerosis.

242. J. B. suffered from periodic attacks of constriction of the chest with epigastric pain and vomiting. Physical examination was negative except for marked arteriosclerosis. He was observed in one attack during which the electrocardiogram showed complete ventricular asystole of about 11 seconds duration. After discharge from the hospital, the patient showed almost daily attacks. He was unconscious during attacks. He had no signs of congestive failure. The diagnosis was Stokes-Adams syndrome, and myocardial degeneration.

295. M. C. complained of weakness of 6 months duration. He had frequently been troubled by painful joints for 15 years. Occasional palpitation with precordial pain was felt for several years which was associated with dyspnea on

exertion. At time of test he was unable to walk more than 600 feet without conspicuous dyspnea. There was no sign of congestive failure. Physical examination showed marked emaciation. Apex in fifth space was 9 cm. from midsternal line. The sounds were distant and regular. There was slight tortuosity of peripheral arteries. The diagnosis was myocardial degeneration and ? syphilis.

241. D. M. felt tiredness and shortness of breath on walking for the past 2 years. He gave no history of congestive failure. Physical examination showed the heart normal in size. The sounds were regular and distant. Conspicuous thickening of the peripheral vessels was noted. The diagnosis was marked generalized arteriosclerosis.

284, 312. J. G. entered the hospital because of dyspnea, anorexia, and weakness beginning 4 weeks previous to this test, when he developed severe attacks of nocturnal dyspnea, associated with a sense of pressure over the epigastrium. Physical examination showed orthopnea. The sounds were faint. A soft systolic murmur over the aortic area was heard. Brachial and radial arteries were sclerosed. Moist râles over both bases were heard. The liver edge was palpable and tender. Slight pitting edema over both ankles was present. At time of second test, no. 312, the patient was objectively and definitely worse. He was able to sleep flat on the right side though not on the left side, and was still troubled by paroxysmal nocturnal dyspnea. Physical examination was as before except that there was a presystolic gallop rhythm and moist râles could be heard everywhere over both lungs. There was marked pitting edema over the buttocks; the thighs and legs, and his face and arms were edematous. The diagnosis was arteriosclerosis and cardiac asthma.

360. M. H. entered the hospital complaining of increasing shortness of breath and swelling of the feet, twelve months in duration. At time of test he was orthopneic. Physical examination showed an enlarged heart with the maximum impulse in the sixth space 13.5 cm. from the midsternal line. The first sound was snapping and was followed by a soft systolic murmur. Conspicuous swelling and pitting edema of legs and signs of fluid in the right chest were found. Liver was not palpated. The patient's course was progressively downward and he died two weeks later. The diagnosis was generalized arteriosclerosis, and ? syphilis.

341. T. R. entered the hospital with conspicuous dyspnea, weakness and discomfort. At time of test there was no orthopnea although his dyspnea was extreme. The size of the heart was approximately normal and no murmurs were heard. The rate was rapid and regular. There were signs of fluid in the abdomen and in the right chest. Marked pitting edema was noted over the extremities. The diagnosis was myocardial degeneration and general anasarca.

B. With fibrillation of the auricles

362. M. C. entered the hospital complaining of palpitation and dyspnea eleven months in duration. Physical examination showed the left border of cardiac dulness 14 cm. from the midsternal line in the fifth interspace. The heart sounds were totally irregular. There was slight pitting edema over the ankles. The diagnosis was auricular fibrillation and arteriosclerosis.

411. M. M. entered complaining of shortness of breath and palpitation. On entry to hospital he showed the signs of congestive failure which responded well to treatment by rest and digitalis, so that at the time of test there was but slight pitting edema over the legs. The heart was slightly enlarged. No râles were heard over the lungs. The diagnosis was auricular fibrillation and generalized arteriosclerosis.

246, 261. W. D., five years previously, following an operation, had shortness of breath, slight orthopnea, and swelling of legs and abdomen. Diagnosis at that time was auricular fibrillation, chronic myocarditis, coronary sclerosis and ascites. One week before admission he noted swelling of the ankles. At time of test no. 246, he had been completely digitalized and showed evidence of mild toxic effects such as vomiting. Physical examination showed the heart rhythm totally irregular, and the left border of dulness 13 cm. from the midsternal line in the fifth space. Bubbling râles were heard over bases of lungs. He was short of breath and unable to walk. The liver edge was palpable and tender. He showed slight pitting edema over the ankles and of subcutaneous tissues. At time of test no. 261, there was no edema of the legs. Vital capacity was not reliable because of nasopharyngitis. The diagnosis was myocardial degeneration; auricular fibrillation.

247. F. B. had dyspnea on moderate exertion and nocturnal paroxysmal attacks of precordial distress associated with shortness of breath. There was no history of congestive failure. Physical examination showed heart apex impulse in the fifth space, 11.5 cm. from the midsternal line. Sounds were distant. There was marked sclerosis of the peripheral vessels. The diagnosis was auricular fibrillation, arteriosclerosis and cardiac asthma.

IV. Patients with Arterial Hypertension

377. T. M. had never complained of any symptoms referable to his cardiovascular system. Physical examination showed the heart slightly enlarged. The sounds were normal and regular in rhythm. The diagnosis was essential hypertension.

272. E. M. had had attacks of dizziness, forcing him to lie down. These were associated with pain over the lower anterior chest, and palpitation. There was no swelling of ankles or puffiness of face. Physical examination showed tortuous retinal vessels, the left border of cardiac dulness 9.5 cm. in the nipple line in the fifth space. The pulses were equal, regular and synchronous, and the radial arteries, neither thickened nor sclerosed. Blood pressure during stay in hospital varied from 170 to 200 systolic and from 110 to 140 diastolic. Urine showed a specific gravity of 1004, no fixation, slight trace of albumin, no sugar and numerous red cells. Phthalein test of kidney function showed 57 per cent the first hour and 21 per cent the second hour. Wassermann test was negative. The diagnosis was hypertension and vascular nephritis.

307. B. M. had had for 6 years dizziness and headaches but no symptoms of cardiac decompensation. He had had arterial hypertension for at least 5 months. Physical examination showed puffiness about both eyes. The heart was enlarged to the left and a soft blowing systolic murmur was heard over apex. Lungs

were clear. Liver was not felt. Blood pressure at first determination, 5 months previously, was 188 systolic, and 90 diastolic. Urine was negative. The diagnosis was hypertension.

331. F. S. entered the hospital complaining of shortness of breath two months in duration. He was dyspneic, and orthopneic on entry, and showed edema of both legs. After five weeks of rest in bed in the hospital he improved so that at the time of test he was neither orthopneic or dyspneic, and showed no evidence of congestive failure. Physical examination showed a slightly enlarged heart and slight arteriosclerosis. Kahn test gave a negative reaction. The diagnosis was hypertension.

357. G. M. stated that his high blood pressure had been accidently discovered one year previously. He felt well and had been actively at work. Physical examination showed the heart slightly enlarged. The anterior posterior diameter of the chest seemed somewhat increased, and an occasional musical râle could be heard on expiration. The diagnosis was hypertension and ? pulmonary emphysema.

336. A. O. entered the hospital because of carcinoma of lip. His elevated blood pressure was discovered in the course of the routine physical examination. No symptoms were referable to the cardiovascular system. Physical examination showed slight cardiac enlargement and moderate thickening of the arteries and veins. The diagnosis was hypertension.

309. J. M. had had dizziness of 7 months duration but no dyspnea, orthopnea, or evidence of congestive failure. Nocturia 3 had been present for 7 months. Physical examination showed the apex impulse in fifth space, 12 cm. from the midsternal line. The heart rate and rhythm were normal and no murmurs were heard. Urine showed no fixation of specific gravity and very slight trace of albumin. The diagnosis was hypertension.

305. H. M. had had occasional shortness of breath of 2 weeks duration, and a choking sensation the night before admission. He had had several similar attacks during the previous 2 months but no symptoms of congestive failure. There had been nocturia 2–3 of one month's duration. Physical examination showed edema of conjunctivae and eyelids, and the heart, moderately enlarged. The sounds were regular and of fair quality. No murmurs were heard. There was no evidence of sclerosis. Non-tender liver edge was palpable two fingers below costal margin. There was no orthopnea. Urine showed no fixation of gravity, and a slight trace of albumin. There was no nitrogen retention, no signs of arteriosclerosis or congestive failure. The diagnosis was hypertension.

296, 300. M. P., beginning 5 years before entry, had had attacks of pain in chest radiating to left arm, associated with dyspnea. Three weeks before entry, paroxysms of pain and dyspnea became more frequent and more severe. Paroxysms lasted about 3 minutes and were agonizing. Physical examination showed peripheral vessels sclerosed and tortuous, heart not enlarged and no signs of congestive failure. Urine showed a slight trace of albumin and hyaline casts with a slight tendency toward fixation of specific gravity. The diagnosis was hypertension and chronic nephritis.

308. M. C. had had precordial pain of several years duration, with occasional palpitation. Patient never stopped his work. There was no dyspnea or orthopnea and no evidence of congestive failure. Physical examination showed heart apex in fifth space, 12 cm. from the midsternal line, no murmurs and no thrills. The lungs showed the signs of emphysema. Liver was not felt. Radial and brachial arteries were sclerosed and somewhat tortuous. There were no signs of congestive failure. Urine was entirely normal with no fixation of gravity. The diagnosis was arteriosclerosis and hypertension.

304. M. S. had no cardiac history but was troubled by dizziness and headaches. Hypertension was discovered accidentally. Physical examination was entirely normal. Urine was clear with no fixation of specific gravity. There was no nitrogen retention. The diagnosis was hypertension.

427, 429. C. B. had noticed slight dyspnea on exertion beginning two years before entry which had gradually become progressively worse. His blood pressure had been elevated for at least three years. Three weeks before entry his feet and legs began to swell. Physical examination at the time of the first test showed conspicuous congestive failure with cyanosis and dyspnea. There was generalized anasarca. The heart was moderately enlarged. His weight was 162 pounds. The patient was rapidly digitalized and within twelve hours passed large amounts of urine, his ventricular rate slowed, and his vital capacity increased. His weight at the time of the second test was 146 pounds. The edema had lessened conspicuously, though light pitting edema was still evident over the lower extremities and the buttocks. The diagnosis was hypertension; cardiac decompensation.

423. P. F. had never been troubled by symptoms referable to his cardiovascular system, but on rest in bed first developed swelling of the legs. Physical examination was negative save for the elevated blood pressure and edema of the legs. The diagnosis was hypertension.

278. J. G. had had attacks of abdominal pain, and frequent attacks of severe nocturnal dyspnea, lasting 10 to 15 minutes. Heart was normal in size, sounds regular and of good quality. Faint systolic murmur over the mitral area. Pulses were equal and of increased tension. Liver edge was felt 3 fingers below the costal margin, moderately tender. There was no edema over the extremities. Urine showed a tendency toward fixation of specific gravity, slight trace of albumin, occasional hyaline and cellular casts. There was no nitrogen retention. 'Phthalein output was 45 per cent in 2 hours. The diagnosis was hypertension; vascular nephritis.

334. E. A. had suffered a complete right hemiplegia two years previously from which he had only incompletely recovered. For sixteen months he had suffered from shortness of breath on exertion with nocturnal dyspnea, of one month duration. He had known that his blood pressure was elevated for two and a half years. Physical examination showed sounds of poor quality, rapid, and absolutely irregular in rhythm. No murmurs were heard. The radial arteries were thickened. The diagnosis was auricular fibrillation, arteriosclerosis and hypertension.