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STUDIES ON THE VELOCITY OF BLOOD FLOW

IV. The Velocity of Blood Flow and Its Relation to Other Aspects of the Circulation in Patients with Arteriosclerosis and in Patients with Arterial Hypertension[1]

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This study undertakes the correlation of the clinical manifestations of arteriosclerosis and of hypertension with possible disturbances in the velocity of blood flow, venous and arterial pressures, and vital capacity of the lungs. In studying patients with arteriosclerosis and with evidences of myocardial degeneration it seemed desirable to divide them into two groups. The first group includes patients with regular ventricular rhythm; the second group presents observations on patients who showed total ventricular arrhythmia.

I. PATIENTS WITH ARTERIOSCLEROSIS AND WITH EVIDENCES OF MYOCARDIAL DEGENERATION

A. Patients with regular rhythm (table 1)

Rheumatic infection tends to strike, not only the valves, but also the myocardium. The consequent ill effects on the circulation are a result of these two lesions, the relative importance of which varies from patient to patient. In the group of patients presented here, the lesion is almost entirely myocardial and the rhythm is regular. Consequently, the study of such patients provides an opportunity to observe the practically uncomplicated effect on the circulation of but one factor, the myocardium.

All the patients in this group exhibited normal sinus rhythm, with

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TABLE 1

The velocity of blood flow and its relation to other aspects of the circulation in patients with arteriosclerosis and with evidences of myocardial degeneration with regular rhythm

Date	Test number	Name	Diagnosis	Age	Surface area	Temperature	Respiration	Pulse	Blood pressure		Venous pressure	Injected	Vital capacity	Vital capacity per square meter	Circulation time	Circulation time per square meter
									Systolic	Diastolic						
					sq. m.	°F.			*mm.* Hg	*mm.* Hg	*mm.* H_2O	*milli-curie*	*cc.*	*cc.*	*seconds*	*seconds*
June 21	215	D. C.	Myocardial degeneration; emphysema	77	2.04	n	22	54	108	55	35	3.0	3,200	1,640	22	11
January 14	71	T. H.	Arteriosclerosis; myocardial degeneration	53	1.82	98.2	24	95	150	80	0.5	4.2	2,100	1,150	25	14
January 9	48	D. M.	Arteriosclerosis	38	1.76	97.6	20	75	92	58	20	4.8	5,200	2,950	25	14
November 20	17	E. L.	Moderate arteriosclerosis, myocardial degeneration	51	1.92	98.0	23	75	134	74	58	2.9			29	15
January 13	63	J. W.	Arteriosclerosis; myocardial degeneration	57	1.60	98.2	26	70	108	72	25	5.1	2,800	1,800	25	15
December 5	21	L. C.	Moderate arteriosclerosis	44	1.67	98.2	20	60	110	55	45	1.8	3,200	1,900	25	15
															26	16
December 4	20	L. C.	Moderate arteriosclerosis	44	1.67	98.6	19	62	110	60	50	4.1	3,100	1,850	27	16
December 2, 1925	19	L. C.	Moderate arteriosclerosis	44	1.67	97.4	18	60	120	80	45	1.7	3,100	1,850	28	17
January 12	57	J. W.	Arteriosclerosis; diabetes	66	1.58	97.6	18	71	142	86	48	6.8	2,800	1,700	29	18
January 12	58	J. W.	Arteriosclerosis	64	1.55	97.6	16	71	124	54	−12	6.0	2,200	1,420	29	19
June 14	193	H. B.	Arteriosclerosis	64	1.60	n	24	68	114	54	−35	5.1	3,100	1,930	30	19
February 12	87	T. O.	Myocardial senile emphysema; arteriosclerosis	70	1.66	97.2	12	52	124	64	21	2.1	2,700	1,630	32	19

June 16	199	A. C.	Arteriosclerosis; myocardial degeneration	70	1.74	n	26	81	130	76	30	4.0	2,350	1,350	34	19
June 23	223	H. B.	Myocardial degeneration; arteriosclerosis; coronary thrombosis	52	1.66	n	18	60	114	64	65	5.8	2,650	1,500	33	20
December 16, 1925	33	J. O.	Chronic arthritis; moderate arteriosclerosis	58	1.71	98.1	22	74	114	65	0.8	3.2	4,400	2,570	34	20
March 16	168	F. S.	Myocardial degeneration	66	1.94	98.0	19	88	126	58	32	3.5	2,150	1,120	30	21
September 22	242	J. B.	Myocardial degeneration	57	1.77	96.6	18	64	118	80	−7	4.6	3,750	2,118	39	22
June 14	194	J. W.	Arteriosclerosis; diabetes	66	1.61	n	20	56	125	80	80	5.2	2,800	1,730	37	23
November 4	295	M. C.	Myocardial degeneration	72	1.46	98.4		62	75		35	4.0	1,550	1,062	39	27
September 22	241	D. M.	Arteriosclerosis; diabetes	78	1.57	95.2		72	114	50	15	4.8	3,200	2,038	45	28.7
December 29, 1925	36	J. G.	Myocardial degeneration; emphysema I.	79	1.63	96.2	18	76	116	65	30	10.6	2,950	1,800	48	29
October 28	284	J. S.	Arteriosclerosis; cardiac asthma	66	1.57	97.2		100	125	90	35	4.5	2,000	1,273	51	32
October 28	290	L.	Arteriosclerosis; arthritis	60		99		72				5.0	3,000		25	

clinical and electrocardiographic evidence of myocardial degeneration and of arteriosclerosis. The majority did not show the usual symptoms and signs of cardiac failure, although a history of weakness and restricted activity was obtainable in almost every case. A few complained of dyspnea or precordial pain at rest or on exertion, but were without other signs or symptoms of cardiac weakness. Only five patients (nos. 58, 36, 71, 168 and 384) showed, at the time of the test of the velocity of blood flow, evidences of congestive failure, namely, moist râles at the bases of one or both lungs. The patients

TABLE 2

A. Patients with signs of arteriosclerosis and of myocardial degeneration without history of cardiac failure and without signs of congestive failure

Test number	Circulation time	Circulation time per square meter	Vital capacity	Vital capacity per square meter body surface	Venous pressure
	seconds	*seconds*	*cc.*	*cc.*	*cm.* H_2O
17	29	15			5.8
19	28	17	3,100	1,850	4 5
33	34	20	4,400	2,570	0.8
48	25	14	5,200	2,950	2.0
87	32	19	5,700	1,630	2 1
98	31	17	2,850	1,530	1 2
193	30	19	3,100	1,930	−3.5
194	37	23	2,800	1,730	8.0
215	22	11	3,200	1,640	3 5
242	39	22	3,750	2,118	−7.0
290	25		3,000		
Average....	30	17.7	3,410	1,994	1 7

may be conveniently subdivided according to the signs or symptoms of congestive failure.

Under the first heading (table 2) are grouped the patients without history of cardiac failure. In all of them physical activity was restricted on account of general weakness. They had not suffered from congestive failure. In the second subdivision (table 3) are those patients who, besides weakness, complained of dyspnea on exertion, but did not show signs of congestive failure. In the third subdivision (table 4) are those who exhibited signs of early congestive failure at the time of the test. That congestive failure was not marked is borne

out by the venous pressure measurements which were within the limits of normal.

In general, the patients in all three groups showed normal venous pressures, lowered vital capacities, and circulation times that were slightly, moderately, or greatly prolonged. Caution must be observed in the interpretation of the lowered vital capacities because

TABLE 3

B. Patients complaining of dyspnea on exertion but without congestive failure

Test number	Circulation time	Circulation time per square meter	Vital capacity	Vital capacity per square meter	Venous pressure
	seconds	*seconds*	*cc.*	*cc.*	*cm.* H_2O
57	29	18	2,800	1,700	4.8
63	25	15	2,800	1,800	2.5
70	30	18	3,100	1,920	2.5
199	37	23	2,350	1,350	3.0
223	33	20	2,650	1,500	6.5
241	39	27			
295	45	29	3,200	2,038	1.5
Average....	34	22	2,817	1,718	3.5

TABLE 4

C. Patients who showed at time of determination signs of congestive failure

Test number	Circulation time	Circulation time per square meter	Vital capacity	Vital capacity per square meter	Venous pressure
	seconds	*seconds*	*cc.*	*cc.*	*cm.* H_2O
36	48	29	2,950	1,800	3.0
58	29	19	2,200	1,140	−1.2
71	25	14	2,100	1,150	−0.5
168	30	21	2,150	1,120	3.2
284	51	32	2,000	1,273	3.5
Average....	36.6	23	2,280	1,296	1.6

practically all of the patients were of advanced years. All were over fifty except L. C. and D. M. (nos. 19 and 48), who were forty-four and thirty-eight years of age respectively, the average being sixty. Wintrich (2) in 1854, in the course of his study of the vital capacity of thirty-five hundred individuals, found that it tended to be slightly diminished between the ages of forty and fifty; while between the

ages of fifty and sixty years he observed great variation. The reduction in some individuals of advanced years is undoubtedly an expression of underlying emphysema, and is not necessarily due to circulatory causes. Advanced age in itself does not predispose to reduced velocity of blood flow, for we have found (3) that persons of approximately the same age as the patients here studied may have blood flow velocity within the limits of the normal observed in younger individuals.

Inspection of table 1 which includes all patients of the three groups shows that while the circulation times were in general prolonged, this prolongation was not conspicuous. Of the eighteen patients studied, only four showed arm to arm velocities of blood flow greater than thirty-five seconds. In seven patients, however, the circulation times were between twenty-five and thirty seconds. The vital capacities were all definitely reduced except in D. M. (no. 48), in whom it was normal, but whose circulation time was just outside of the normal limits. This patient did not give a history of cardiac failure but showed evidence of unusually advanced arteriosclerosis. In Group A (table 2) which includes patients without history or signs of congestive failure, there is considerable variation of the circulation time although, in general, its average prolongation runs parallel to the decrease in vital capacity. The venous pressures, on the other hand, are, in general, all within the limits of normal, as is to be expected in patients without conspicuous congestive failure. The patients of Group B (table 3) who complained of dyspnea on exertion without signs of congestive failure showed greater retardation of the velocity of blood flow and a more marked reduction in the vital capacity than patients of Group A.

Five patients (table 4) entered the hospital because of congestive failure. On rest in bed, and on administration of digitalis, they improved strikingly so that by the time the velocity of blood flow was measured, edema and dyspnea had disappeared and they showed only moist râles at the bases of the lungs. In patients who are regaining cardiac compensation, the increased venous pressure tends to disappear early, followed at first by a return of the velocity of blood flow to normal, and only later by a rise in the vital capacity. The time relationship between these three phenomena in patients showing

circulatory improvement is the reverse of that observed in patients with increasing failure. The measurement of the velocity of blood flow therefore affords information of prognostic value.

While there is a general relationship between the venous pressure, velocity of blood flow, and vital capacity, the results do not permit the formulation of a definite quantitative relationship. Whether such a relationship is possible is very questionable. The velocity of blood flow from arm to arm reflects the situation existing in the arm, and to a larger extent, in the lungs. But the clinical signs of congestive failure may be due to passive congestion of the liver, of the legs, or of other parts of the body. That there is a precise quantitative relationship between the velocity of blood flow through the lungs and in each and every other portion of the circulation is improbable. Vital capacity measurements, furthermore, do not lend themselves to precise interpretations in persons with generalized arteriosclerosis, because of the tendency to pulmonary emphysema.

In studying patients with evidences of arteriosclerosis and myocardial degeneration, we have been impressed by the relatively late appearance of dyspnea. This may be due in part to the fact that these persons frequently experience weakness as their earliest symptom, whereas patients with rheumatic or syphilitic heart disease whose blood velocity is similarly prolonged do not restrict their activities until compelled to do so by dyspnea. The spontaneous reduction of muscular activity, in patients with arteriosclerosis goes more or less parallel with impairment of heart muscle function so that these patients live within the limits of the functional capacity of their hearts and thus do not show symptoms of cardiac insufficiency. The late appearance of dyspnea may also be due in part to the presence of emphysema, for Scott (4) has shown that patients with emphysema are remarkably insensitive to concentrations of carbon dioxide which would be sufficient to cause overstimulation of the respiratory centers of normal persons.

B. Patients with fibrillation of the auricles

The patients presented here (table 5) showed fibrillation of the auricles without antecedent rheumatic or syphilitic infection, but with signs of advanced arteriosclerosis.

The average circulation time of all patients with auricular fibrillation is approximately 100 per cent above the extreme upper limit of normal. The conspicuous slowing of the velocity of blood flow is in

TABLE 5

Circulation times and related measurements in patients with fibrillation of auricles and with history of dyspnea but without signs of congestive failure at the time of the determination

Test number	Circulation time	Circulation time per square meter	Vital capacity	Vital capacity per square meter	Venous pressure
	seconds	*seconds*	*cc.*	*cc.*	*cm.* H_2O
30	57	34	3,050	1,800	0.3
31	47	28	3,150	1,870	1.2
69	42	25	2,500	1,500	1.2
74	28	16	2,800	1,560	1.2
76	44	24	3,600	1,960	4.1
226	23	14	3,000	1,820	4.7
247	42	26	1,100	688	−3.0
Average....	40.4	23.9	2,743	1,600	1.4

TABLE 6

Circulation times and related measurements in patients with fibrillation of auricles, with history of congestive failure and with signs of congestive failure at the time of the determinations

Test number	Circulation time	Circulation time per square meter	Vital capacity	Vital capacity per square meter	Venous pressure
	seconds	*seconds*	*cc.*	*cc.*	*cm.* H_2O
23	55	29	3,100	1,650	7.0
90	46	38	2,650	1,490	2.4
100	39	18	2,250	1,030	5.2
113	34	14	2,400	1,130	−0.5
222	68	45	2,350	1,540	12.5
227	36	23	2,250	1,480	13.0
229	73	43	1,550	860	16.0
246	55	33	2,100	1,272	3.0
Average....	50.1	29.9	2,581	1,306	7.3

accord with the minute volume output studies of Lundsgaard (5). The degree of prolongation bears a definite relation to the clinical condition. These patients were suffering from more severe cardiac

TABLE 7

The velocity of blood flow and its relation to other aspects of the circulation in patients with arteriosclerosis and with evidences of myocardial degeneration with fibrillation of the auricles

Date	Test number	Name	Diagnosis	Age	Surface area	Temperature	Respiration	Pulse	Arterial pressure		Venous pressure	Injected	Vital capacity	Vital capacity per square meter	Circulation time	Circulation time per square meter
									Systolic	Diastolic						
					sq. m.	*°F.*			*mm. Hg*	*mm. Hg*	*mm. H_2O*	*millicurie*	*cc.*	*cc.*	*seconds*	*seconds*
June 25	226	O. C.	Arteriosclerosis	71	1.64	n		124	96	44	47	6.7	3,000	1,820	23	14
February 27	113	W. H.	Myocardial degeneration	67	2.12	98.6	20	74	126	50	−0.5	3.8	2,400	1,130	29	14
February 10	74	R. F.	Myocardial degeneration	49	1.79	99.2	21	88	124	64	12	2.8	2,800	1,560	28	16
February 18	100	W. H.	Myocardial degeneration	67	2.18	99.2	24	76	150	62	52	7.5	2,250	1,030	39	18
February 15	90	W. D.	Myocardial degeneration	55	1.78	97.0	26	46	84	50	24	3.6	2,650	1,490	38	21
June 25	227	J. U.	Myocardial degeneration	49	1.52	n	—	130	130	60	13	8.4	2,250	1,480	36	23
									140	70						
February 10	76	T. M.	Myocardial degeneration	50	1.83	98.0	23	72	136	56	41	4.1	3,600	1,960	44	24
January 14	69	D. M.	Myocardial degeneration	53	1.66	98.4	26	80	132	52	12	6.3	2,500	1,500	42	25
								90								
September 24	247	F. B.	Myocardial degeneration; arteriosclerosis	65	1.60	97.6		64	126	64	−30	3.4	1,100	687	42	26.2
December 29	31	J. W.	Myocardial degeneration	43	1.68	96.6	20	58	104	52	12	2.4	3,150	1,870	47	28
December 4, 1925	23	J. S.	Arteriosclerosis; marked myocardial degeneration	73	1.87	97.6	21	48	148	72	70	2.0	3,100	1,650	55	29
September 1, 1925	15	J. R.	Arteriosclerosis; myocardial degeneration	59	1.85				150	100		7.1	1,400	750	55	29
September 24	246	W. D.	Myocardial degeneration	55	1.65	98.1	n	42	94	65	30	7.8	2,100	1,272	55	33.3
December 16, 1925	30	J. W.	Myocardial degeneration	43	1.68	98.2	25	52	112	48	0.3	4.3	3,050	1,800	57	34
June 25	229	J. W.	Myocardial degeneration	45	1.75	n	24	112	88	78	16	6.4	1,550	860	73	43
June 23	222	J. U.	Myocardial degeneration	49	1.52	n	24	130	136	65	125	6.1	2,350	1,540	68	45

failure than patients with heart disease of similar etiology with regular rhythm. The question therefore arises whether the severity of the cardiac failure in these patients is due to the abnormal rhythm, or whether the severity of the cardiac failure and the abnormal rhythm at least in certain patients are both expressions of grave myocardial damage. As is well known, auricular fibrillation itself is not necessarily the cause of the clinical signs and symptoms of circulatory decompensation.

The exact degree to which the abnormal rhythm and the myocardial lesion each contributes to the decompensatory state must vary from individual to individual. Further studies are being attempted to investigate these factors more precisely.

Examination of the circulation times of the two groups shows that the degree of retardation is less in those individuals who were without signs of congestive failure than in those patients who showed positive signs at the time of the test. The average circulation time of patients with auricular fibrillation, but without signs of congestive failure, was 40.4 seconds or approximately 68 per cent above the extreme upper limit of normal, while it was 50.1 seconds or approximately 108 per cent above the extreme upper limit of normal in patients with these signs.

II. THE VELOCITY OF BLOOD FLOW AND ITS RELATION TO OTHER ASPECTS OF THE CIRCULATION IN PATIENTS WITH ARTERIAL HYPERTENSION

Consideration of the dynamic factors concerned in the maintenance of arterial blood pressure shows that if the hypertensive state be due to a preponderant increase in cardiac energy, and the peripheral resistance be not proportionately increased, we might expect the velocity of blood flow to be increased. If, however, the peripheral resistance is relatively more increased than the cardiac energy developed, and particularly if the elasticity of the vessels be diminished, we might expect that with the production of the hypertensive state, the velocity of blood flow would be lessened. Were there an exact balance of these opposing factors the blood velocity would be unaltered. Since factors such as cardiac energy, elasticity of the vessel walls, and peripheral resistance cannot be measured directly in man, the following study was undertaken in the hope that measurement of

the velocity of blood flow, which is a resultant of many complicated dynamic factors, might aid in our understanding of the mechanism of hypertension.

The data obtained in studying patients with hypertension are divided into three groups (table 8). Group A consists of patients without any evidence of circulatory failure at rest at the time of the tests, and in whom the velocity of blood flow was normal. Group B also includes patients who did not exhibit symptoms or signs of circulatory failure at rest or on exertion, but in whom there was definite slowing of blood flow. Group C presents patients with decreased velocity of blood flow but with symptoms or signs of circulatory failure.

The blood pressures of the patients of Group A, with one exception, at the time of test, varied from 160 mm. Hg to 220 mm. Hg systolic and from 76 mm. Hg to 114 mm. Hg diastolic. L. S. (no. 96), whose blood pressure was normal, had suffered from dizziness and headaches for three years. His physician had told him that his blood pressure was elevated and at the time of entry to the hospital the systolic blood pressure was 195 mm., the diastolic 50 mm. The finding of signs of cardiac enlargement, in the absence of any signs of cardiac failure, suggested that the blood pressure of this patient had been elevated for some time. The velocity of blood flow and the vital capacity were normal. Were his blood pressure to fall without any diminution in the peripheral resistance the velocity of blood flow might be expected to become slowed. That it did not become slowed suggests that his capillary resistance was due to functional causes rather than to persistent structural alteration such as widespread capillary occlusion due to arteriosclerosis. E. M. (no. 272) complained of breathlessness only on exertion, had never suffered from congestive failure, and his circulation was compensated at the time of test.

The presence of a normal velocity of blood flow in the patients of Group A is of considerable interest. In no patients with hypertension did we find an increased velocity of flow. 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.

Group B consists of patients in whom there was a slowing of the blood flow, but who were able to continue their daily duties without

TABLE 8

The velocity of blood flow and its relation to other aspects of the circulation in patients with arterial hypertension

Date	Test number	Name	Diagnosis	Age	Surface area	Temperature	Respiration	Pulse	Arterial pressure		Venous pressure	Injected	Vital capacity	Vital capacity per square meter	Circulation time	Circulation time per square meter
									Systolic	Diastolic						
Group A. Patients with compensated circulation at time of test, whose velocity of blood flow was within normal limits																
					sq. m.	*°F.*			*mm. Hg*	*mm. Hg*	*mm. H_2O*	*millicurie*	*cc.*	*cc.*	*seconds*	*seconds*
November 6	307	B. N.	Hypertension	57	1.77	98.1		80	180	102	105	4.5	3,500	1,980	15	8.4
November 6	305	H. M.	Hypertension	70	1.74	98.1		90	194	104	200	7.0	2,700	1,552	18.5	10.6
February 10	80	D. C.	Essential hypertension	39	1.65	98.2	19	52	00	114	22	2.5			19	12
February 17	96	L. S.	Periodical hypertension	49	1.79	98.4	20	68	132	88	42	0.9	4,050	2,260	21	12
October 21	272	E. M.	Hypertension	46	1.81	98.2		82	184	110	80	5.0	3,800	2,099	22	12.1
January 12	56	B. G.	Hypertension	53	1.63	96.8	21	75	160	76	20	1.63	3,100	1,900	21	13
Group B. Patients with compensated circulation, whose velocity of blood flow was prolonged																
November 6	309	J. M.	Hypertension	57	1.79	98.6		58	205	104	85	4.0	3,650	2,039	26	14.5
November 6	308	M. C.	Hypertension	57	1.86	97.2		66	192	116	85	4.0	4,400	2,368	31	16.6
November 6	304	M. S.	Hypertension	61	1.76	98.2		76	190	112	65	4.0	2,900	1,648	30	17
June 16	198	J. M.	Hypertension	52	1.68	n	16	67	204	116	60	4.4			30	18
January 13	61	H. B.	Hypertension, auricular fibrillation	51	1.98	98.5	36	96	220	116	25	2.7	2,900	1,460	37	19

Group C. Patients with decompensated circulation, whose velocity of blood flow was prolonged

June 28	233	P. S.	Hypertension; auricular fibrillation	56	1.96	n	26	82	190	110	45	8.5	2,500	1,270	31	16
November 4	296	M. B.	Hypertension	50	1.60	98.4		94	214	110	135	4.5	4,100	2,560	29	18.4
October 26	278	J. G.	Hypertension	53	1.92	97.2		120 140	165	110	90	3.5	2,800	1,538	34	18.6
June 21	214	R. M.	Arteriosclerosis; auricular fibrillation	61	1.64	n	19	104	220	134	40	4.0			31	19
November 5	300	M. B.	Hypertension	50	1.60	99.2		92	180	95	125	4.0	4,000	2,500	33	20.6
June 14	196	G. H.	Arteriosclerosis; hypertension; auricular fibrillation	71	1.62	n	20	74	200	100	25	4.6	2,150	1,330	36	22
January 14	68	W. H.	Arteriosclerosis; myocardial degeneration; hypertension; auricular fibrillation	50	1.87	97.8	26	38 45	190	95	−0.5	3.0	2,700	1,440	49	26

experiencing any symptoms of circulatory insufficiency. We lay emphasis on these observations because they constitute the only instances in which we have encountered such prolongation in any group of patients without signs or symptoms of circulatory failure. It is possible, that the abnormal increase in blood pressure constitutes a compensatory mechanism enabling the normal gaseous exchanges to take place in spite of the subnormal velocity of blood flow.

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 in patients with normal blood pressure suffering from circulatory failure. The degree of slowing was approximately that observed in patients with cardiovascular failure due to other causes. In seven of the patients (nos. 198, 296, 304, 305, 307, 308 and 309) the venous pressure was definitely above the extreme upper limit of normal, a phenomenon which has been observed by others (6) (7) (8).

The existence of two groups of patients with hypertension similar to our Groups A and B which cannot be differentiated clinically was also observed by Boas and Frant (9). They found that in one group the capillary pressures were normal, whereas in the other the capillary pressures were elevated. Since we have not measured the capillary pressures of our patients we cannot state whether the two groups differentiated by Boas and Frant correspond to the two groups observed by us. The fact that in none of the patients with hypertension did we observe increased velocity of blood flow suggests, perhaps, that the primary change in hypertension occurs in the peripheral blood vessels and that rise in the arterial tension is a secondary reaction on the part of the body aimed to maintain adequate blood supply to the tissues. For, were the primary change the elevation of the blood pressure, one would expect to find a period when patients with hypertension show an increased velocity of blood flow. This, however, has not been observed. In some patients, on the contrary, the velocity of blood flow is retarded without clinical evidence of decompensation, and we suspect that in these patients the adjustment on the part of the heart to the opposed peripheral resistance was incomplete.

SUMMARY

1. In this and the preceding (3) communication, eighty-seven measurements of the arm to arm circulation time by the radium C method, on male patients with cardiovascular diseases are presented, and an attempt is made to establish the relationship between the velocity of blood flow and other fundamental aspects of the circulation such as the vital capacity of the lungs, the venous and arterial pressures, and the cardiac rate and rhythm.

2. The method as described in a preceding communication has been found adequate for the study of the various aspects of cardiovascular disease.

The velocity of blood flow and its relation to other aspects of the circulation were studied in patients: I. With arteriosclerosis and with evidences of myocardial degeneration

1. Twenty-three measurements of the arm to arm velocity of blood flow and related aspects of the circulation were carried out on twenty patients who showed a regular cardiac rhythm.

2. All these patients showed normal venous pressures, lowered vital capacities and circulation times that were slightly, moderately, or greatly prolonged, according to the degree of circulatory insufficiency.

3. Sixteen measurements of the arm to arm velocity of blood flow and related aspects of the circulation in thirteen persons with fibrillation of the auricles showed that while the retardation of blood flow corresponded to the clinical evidences of cardiac decompensation, the prolongation of the circulation time was greater in proportion to the degree of circulatory decompensation than might be expected on the basis of our tests on similarly decompensated patients who showed a regular rhythm.

II. With Hypertension

1. Eighteen measurements of the arm to arm velocity of blood flow and related aspects of the circulation are presented on seventeen patients suffering from arterial hypertension.

2. Patients with hypertension who exhibit no evidence of circulatory disability may be divided into two groups: in one, the velocity

of blood flow is within the limits of normal, whereas in the other, the velocity of blood flow is retarded.

3. In no patients with hypertension was an abnormally rapid velocity of blood flow observed.

4. In seven patients without evidences of congestive failure, the venous pressures were found to be elevated.

5. Patients with hypertension who show congestive failure have a retardation in the velocity of blood flow similar to that of patients with a corresponding degree of circulatory failure but with a normal blood pressure.

CONCLUSIONS

1. Whereas the arm to arm circulation time in normal, resting, male individuals ranged from eleven to twenty-four seconds, it varied between eleven and seventy-three in male patients with compensated and uncompensated cardiovascular disease.

2. The average arm to arm circulation time in fifty-three normal male individuals was eighteen seconds, whereas the average in eighty-six determinations in patients with cardiovascular disease was thirty-three seconds.

3. The average arm to arm circulation time in those patients who showed no symptoms or signs of circulatory decompensation at the time of test averaged twenty-four seconds, whereas patients exhibiting symptoms or signs or cardiac failure showed an average arm to arm circulation time of thirty-eight seconds.

4. The fact that the average circulation time in normal persons was eighteen seconds, and in patients with compensated cardiovascular disease, was twenty-four seconds, indicates that a retardation in the velocity of blood flow occurs in general before symptoms or signs become manifest.

5. In general, the degree of cardiac decompensation at the time of the test was closely related to the degree of retardation of the velocity of blood flow.

6. Prolonged circulation times always occurred in the presence of a failing circulation, except in one group of patients with arterial hypertension in whom a prolongation of the velocity of blood flow was observed, and who had never shown evidence of circulatory embarrassment.

7. Patients with auricular fibrillation showed a disproportionate prolongation of the blood flow compared with patients with a similar degree of circulatory decompensation but with a regular rhythm.

8. At the onset of circulatory failure, the retardation in the arm to arm velocity of blood flow appeared earlier than the increase in the venous pressure, and somewhat later than the reduction in the vital capacity. In patients with improving circulatory function the venous pressure first returned to normal. This was followed by a return of the velocity of blood flow to within the limits of normal, and somewhat later the vital capacity became normal.

9. When the velocity of blood flow was measured several times in the same patient, it was found that the retardation of the velocity of flow preceded clinical evidence of increasing cardiac failure; and conversely, an increase in the velocity occurred before clinical evidence of improvement appeared.

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APPENDIX

Ia. Abstracts of Histories and Physical Examinations of Patients with Arteriosclerosis and Evidences of Myocardial Degeneration with Regular Rhythm

215. D. C. entered the hospital complaining of dizziness. Six days before entry he suffered what was evidently a cerebral hemorrhage with fainting, vomiting and vertigo. Following this he had been somewhat disoriented until he entered the hospital. During his stay in the hospital his neurological signs of cerebral hemorrhage improved. Occasionally, on physical examination, auricular fibrillation was found. Heart was not enlarged to percussion, sounds were faint but of fair quality. Discharge diagnosis—cerebral hemorrhage; arteriosclerosis; paroxysmal auricular fibrillation; chronic myocarditis.

71. T. H. complained of dyspnea, orthopnea and congestive failure for 8 weeks. He was cyanotic at time of admission. P.E. (on admission)—heart was pushed to right and the rhythm was totally irregular. There was fluid in both chests and thoracentesis was performed 3 times. Patient received novarsurol and quinidin and became regular. P.E. (time of test)—heart was regular and the circulation compensated. No evidence of fluid observed. Few moist râles at left base elicited. Diagnosis—myocardial degeneration; arteriosclerosis.

48. D. M.: No cardiac history obtained. Thickened radial and brachial arteries palpated. Diagnosis—slight arteriosclerosis.

17. E. L. had had no definite history of decompensation. Sclerosed radial and brachial arteries were prominent. The heart sounds were distant. Diagnosis—moderate arteriosclerosis.

63. J. W. complained of dyspnea. P.E. (date of test)—distant heart sounds heard with rough systolic murmur over precordium, loudest over aortic area. Moderate sclerosis of peripheral vessels was present. Diagnosis—arteriosclerosis; myocardial degeneration.

19, 20, 21. L. C. had had no history of decompensation. The heart sounds were distant. Moderately tortuous radial and brachial arteries palpated. Diagnosis—moderate arteriosclerosis.

57. J. W. had had increasing dyspnea for 3 years. He suffered from diabetes for 4 years. The heart was normal except for systolic murmur over precordium. No history of congestive failure. Beaded peripheral arteries felt. Diagnosis—conspicuous arteriosclerosis; myocardial degeneration.

58. J. W. had dyspnea and gastric and precordial distress for 3 years, congestive failure 2 months previously with nocturnal dyspnea. Precordial pain was radiating to the left shoulder. P.E. (at time of test) showed moderate cardiac enlargement. The heart sounds were distant with rough systolic murmur over precordium which was loudest at 2nd right costal space. Moist râles at both bases were heard. Conspicuous sclerosis of brachials, radials and temporals were noted. Diagnosis—myocardial degeneration; generalized arteriosclerosis.

193. H. B. had had no cardiac history. He entered the hospital because of bleeding gums and perifollicular hemorrhages which were diagnosed as scurvy. At time of test patient had improved and signs of scurvy had disappeared. P.E. showed distant heart sounds. Size of heart was normal, sounds, regular. There was conspicuous sclerosis of the femoral, radial and temporal arteries. Diagnosis—general arteriosclerosis.

87. T. O. gave no history of cardiac decompensation. P.E.—size of the heart was normal; the sounds were inaudible. Rough systolic murmur over the apex was heard. All the palpable vessels were markedly sclerosed. The thorax was fixed and the breath sounds were distant. Diagnosis—arteriosclerosis ; senile emphysema.

199. A. C. complained of weakness, loss of weight, shortness of breath on exertion. There was no dyspnea or orthopnea, and no history of congestive failure. P.E.—size of the heart was normal; sounds were not heard. Pulse was regular. Conspicuous thickening and tortuosity of peripheral vessels was present. The chest was barrel-shaped and fixed. The breath sounds were distant. Diagnosis—generalized arteriosclerosis; myocardial degeneration; senile emphysema.

223. H. B. suffered for several years from dyspnea on exertion, and from occasional precordial pain. One year ago he felt dizzy, had sharp precordial pain radiating to the left chest, palpitation, sounds regular but distant. The white blood cell count was 22,000 per cubic mm. and therefore coronary thrombosis was suspected. He was fairly comfortable until 4 weeks previous to present admission, when after a short walk, he developed a rather sudden, marked sensation of suffocation. He was relieved by nitroglycerine and was well except for short dyspnea. On the day of admission, after a walk, the sense of suffocation rather suddenly returned. He vomited once. He had a tight sensation over the upper chest, soreness but not pain, and felt as if he were in extremis. P.E.—Appeared to be "in extremis", with marked dyspnea, orthopnea and slight cyanosis. Heart was slightly enlarged. Sounds were distant, many crepitant râles at both bases. No edema noted. White blood cell count was 28,800. He improved gradually. At time of test his circulation was compensated at rest, no signs of congestive failure were noted, though he was very weak and dyspneic on slight exertion. Electrocardiogram showed signs of coronary occlusion. Diagnosis—coronary thrombosis; arteriosclerosis.

33. J. O. had had history of painful swollen right knee 37 years before admission. At time of admission he suffered from painful joints. P.E.—apex impulse

was not felt. The left border of cardiac dullness was 9 cm. Sounds were regular and of good quality. Temporal arteries were tortuous, and the brachial and radial arteries were moderately sclerosed. Diagnosis—infectious arthritis; arteriosclerosis.

168. F. S.: Following rest in bed for 8 weeks he became short of breath. P.E. (at the time of test)—the heart size and sounds were normal. There was flatness with suppressed breath sounds on left. Râles at both bases were heart. Pitting edema of legs was present. Diagnosis—myocardial failure.

242. J. B. suffered from periodic attacks of constriction of the chest with epigastric pain and vomiting. P.E.—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, patient showed almost daily attacks. He was unconscious during attack. He had no signs of congestive failure. Diagnosis—Stokes-Adams syndrome; myocardial degeneration for 9 months.

194. J. W. had had no history of cardiac decompensation. P.E.—apex beat was not visible and not felt. The left cardiac border dullness was in the 5th space, 9 cm. The sounds were of good quality. Rough systolic murmur with palpable thrill over aortic region was heard. Brachial and radial arteries were hard, tortuous and beaded. There was no edema of the ankles. Diagnosis—generalized arteriosclerosis.

29. R. B. gave no cardiac history. P.E. (date of test)—Heart sounds were muffled. Premature ventricular beats from different foci were shown by the electrocardiogram. Cardiac impulse was not seen or felt. Conspicuous sclerosis of the radial and brachial arteries was observed. Diagnosis—general arteriosclerosis; myocardial degeneration.

295. M. C. complained of weakness of 6 months' duration. Frequently he was 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 yards without conspicuous dyspnea. There was no sign of congestive failure. P.E.—showed marked emaciation. Apex in 5th space was 9 cm. from midsternal line. The sounds were distant and regular. There was slight tortuosity of peripheral arteries. Diagnosis—myocardial degeneration; syphilis.

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

36. J. G. complained of weakness, shortness of breath, and chronic cough of several months' duration. P.E.—there was slight orthopnea. The heart was apparently normal. The arteries were sclerosed. Persistent moist râles at bases were heard. Diagnosis—myocardial degeneration; emphysema.

284. J. G. entered the hospital because of dyspnea, anorexia, and weakness beginning 4 weeks previously, when he developed severe attacks of nocturnal

dyspnea associated with a sense of pressure over the epigastrium. P.E.—There was 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. Diagnosis—arteriosclerosis; cardiac asthma.

290. N. L. gave no history of congestive failure. P.E.—the heart was normal in size. The sounds were of good quality and regular in rhythm. Arteries were tortuous and thickened. Diagnosis—arteriosclerosis.

IB. Abstracts of Histories and Physical Examinations of Patients with Arteriosclerosis and Evidences of Myocardial Degeneration with Fibrillation of the Auricles

226. O. G. gave no cardiac history and had never experienced shortness of breath. P.E.—showed the heart moderately enlarged, the sounds rapid and distant and totally irregular in rhythm. The lungs were hyperresonant and expansion was diminished. Marked thickening of all palpable arteries was present. Both legs were amputated from the thigh. Diagnosis—arteriosclerosis; auricular fibrillation; myocardial degeneration.

100, 113. W. H. gave a history of cardiac failure of one year with marked orthopnea and dyspnea. At time of test he showed orthopnea and dyspnea. P.E.—Left border of cardiac dullness was 13 cm. from the midsternal line. The heart sounds were distant and totally irregular. There was no pulse deficit. Both bases were flat. Moist râles were heard over the lungs. Pitting edema of wrist, arms and legs was present. Diagnosis—myocardial degeneration; auricular fibrillation.

74. R. F. had had occasional shortness of breath with attacks of pain over precordium radiating to the left shoulder, arm, and hand. The pain was never sharp, but rather dull and numb. Slight dyspnea on exertion had been present during the previous few weeks. P.E.—showed the left border of cardiac dullness 12.5 cm. from the midsternal line, sounds of good quality, and no murmurs. Blood pressure (on entry) was 190 systolic and 100 diastolic. No evidence was present of congestive failure. Diagnosis—auricular fibrillation.

90, 246. R. F. five years ago, 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. He improved under digitalis and was able to work. Two days before admission he became dyspneic and orthopneic. Legs and abdomen were not swollen. P.E.—The apex impulse was not felt. The left border of cardiac dullness in 5th space was 13 cm. from the midsternal line. Systolic murmur was heard over precordium; totally irregular rhythm was present with the apex rate, 80, radial rate, 72. Bubbling râles were heard over both bases at time of Test 90. Nails were slightly cyanotic. His circulation was compensated; a rough systolic murmur was heard over the precordium. Patient was discharged from the hospital and was well

until a month before second admission and Test 246, when he experienced cough and dyspnea. One week before this 2nd admission he noted swelling of the ankles. At time of Test 246 he had been completely digitalized and showed evidence of mild toxic effects such as vomiting. P.E. was essentially the same as at previous test, except that he showed slight pitting edema over the ankles and of the subcutaneous tissues. He was short of breath and unable to walk. Liver edge was palpable and tender. Diagnosis—myocardial degeneration; auricular fibrillation.

222, 227. J. U. had had rapidly increasing marked dyspnea, soreness over epigastrium, cough, swelling of abdomen and legs of two months' duration. P.E. showed orthopnea, dyspnea and cyanosis. Veins of neck were distended. The left border of cardiac dullness was 12 cm. from the midsternal line. Sounds were distant, rapid and totally irregular. Arteries were soft. The abdomen was large and the liver edge firm, 5 fingers below the costal margin. There was pitting edema of the lower extremities and over the buttocks. At the time of test the patient was still markedly decompensated with orthopnea, cyanosis and signs of congestive failure. Hgb. 100 per cent. Diagnosis—myocardial degeneration; auricular fibrillation.

76. T. M. had had fatigue for 1 year and dyspnea and paroxysmal palpitation for 3 months, orthopnea and nocturnal dyspnea for 2 months. P.E. showed orthopnea with rapid breathing, the left border of cardiac dullness being 12.5 cm. from the midsternal line, the heart rate 140, with a pulse deficit of 35. Sounds were weak and totally irregular, and a short blowing systolic murmur was present. Both bases were dull. Liver edge was felt 3 cm. below right costal margin. There was pitting edema of both ankles. The circulation was compensated at time of test. There was no edema. The heart rate was 72 with no pulse deficit. Slight pitting edema was present over buttocks. Diagnosis—myocardial degeneration; auricular fibrillation.

69. D. M.: No history of congestive failure. At time of test patient was in moderate distress. P.E. showed the left border of cardiac dullness 12 cm. from the midsternal line. Systolic and early diastolic murmurs were heard over apex, with a loud first sound. Rhythm was totally irregular. The apex rate was 120, with a pulse deficit of 15. Diagnosis—mitral stenosis; 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, P.E. showed heart apex impulse in the 5th space, 11.5 cm. from the midsternal line. Sounds were distant. There was marked sclerosis of the peripheral vessels. Diagnosis—myocardial degeneration; auricular fibrillation; arteriosclerosis; cardiac asthma.

30, 31. J. W. had had shortness of breath and weakness, 8 months prior to admission, and precordial pain, orthopnea and nocturnal dyspnea. There was no history of congestive failure. Repeated electrocardiographic tracings showed spontaneous changes to normal rhythm, flutter and auricular fibrillation. P.E. at time of test showed the heart slightly enlarged, sounds of fair quality. Radial,

brachial and temporal arteries were tortuous and thickened. The circulation was compensated. Diagnosis—myocardial degeneration; auricular fibrillation.

23. J. S. had had increasing dyspnea for 3 years, orthopnea for 2 years. On admission he showed marked cyanosis and general anasarca, and slight jaundice. The vital capacity was 1350 cc. At time of test there were no signs of congestive failure except râles at both bases. The left border of cardiac dullness was 17 cm. from the midsternal line in the sixth interspace. Absolute irregularity of ventricular rate was noted. Systolic murmur was heard over apex. The brachial and radial arteries were rigid. Ronchi and râles were heard at both bases. Diagnosis—general arteriosclerosis; chronic myocarditis; auricular fibrillation.

II. Abstracts of Histories and Physical Examinations of Patients with Hypertension

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. P.E. 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, 90 diastolic. Urine was negative. Diagnosis—hypertension.

305. H. M. had had 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. P.E. showed edema of conjunctivae and eyelids, and the heart was moderately enlarged. The sounds were regular and of fair quality. No murmurs were heard. There was no evidence of sclerosis. The chest was fixed and flat. Non-tender liver edge was palpable two fingers below costal margin. There was no orthopnea. Urine showed no fixation of gravity, a slight trace of albumin. There was no nitrogen retention, no signs of arteriosclerosis or congestive failure. Diagnosis—hypertension.

80. D. C. had had no history or signs of cardiac decompensation. He entered because of accidental fall. The systolic blood pressure of 240 was discovered accidentally 4 years ago. The left border of cardiac dullness was 12 cm. from the midsternal line. Sounds were normal. Diagnosis—hypertension.

96. L. S. had had no signs or symptoms of decompensation and occasional headaches and dizziness for 3 years. The blood pressure at entry was 195 systolic and 50 diastolic. P.E. showed the heart slightly enlarged, with the left border of cardiac dullness 12 cm. from the midsternal line. A_2 was accentuated. Peripheral arterial vessels were tortuous. Diagnosis—arteriosclerosis; hypertension.

272. E. M. had had attacks of dizziness, forcing him to lie down, associated with pain over the lower anterior chest and palpitation. Patient was neurotic. There was no swelling of ankles or puffiness of face. P.E. showed tortuous retinal vessels, the left border of cardiac dullness 9.5 cm. in the nipple line in the 5th space. There was a slight systolic murmur over the tricuspid area in standing position. The pulses were equal, regular and synchronous, and the radial arteries, neither

thickened nor sclerosed. Lungs were normal. 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; 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. Diagnosis—hypertension; vascular nephritis.

56. B. G., for 2 years had been easily excitable, had occasional palpitation and shortness of breath for a few months. He noticed occasional edema of the left ankle. P.E. showed the left border of cardiac dullness 11.5 cm. in the 5th space, heart sounds regular. A_2 accentuated, and a soft systolic murmur over the precordium. At the time of test the circulation was compensated. Diagnosis—hypertension.

309. J. M. had had dizziness of 7 months' duration but no dyspnea, orthopnea, or evidences of congestive failure. Nocturia 3 had been present for 7 months. P.E. showed the apex impulse in 5th 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, very slight trace of albumin. Diagnosis—hypertension.

233. P. S. had had repeated attacks of shortness of breath with congestive failure during preceding 2 years, and breathlessness and swelling of ankles and legs of 3 weeks' duration. P.E. on admission showed chest increased in anterior posterior diameter with numerous moist and musical râles heard anteriorly and posteriorly. At time of test there was slight dyspnea and fluid in right chest. Heart showed the left border of dullness well outside nipple line, no enlargement to right, action totally irregular, pulse deficit of 5. There was pitting edema of ankles and buttocks. Diagnosis—myocardial decompensation (mild); general arteriosclerosis; auricular fibrillation; chronic myocarditis; pulmonary emphysema.

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. P.E. showed heart apex in 5th space, 12 cm. from the midsternal line, no murmurs, 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. Diagnosis—arteriosclerosis; hypertension.

304. M. S. had no cardiac history but was troubled by dizziness and headaches. Hypertension was discovered accidentally. P.E. was entirely normal. Urine was clear with no fixation of specific gravity. There was nitrogen retention. Diagnosis—hypertension.

198. J. M. had been in good health and gave no history of weakness, dyspnea or congestive failure. He came into hospital because of fainting for the first time. P.E. showed the heart apex in 5th space and the left border of dullness 11 cm. from the midsternal line. Sounds were regular and normal. There was slight thickening of brachial arteries. Diagnosis—hypertension.

296, 300. M. B., 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. P.E. showed peripheral vessels sclerosed and tortuous, heart not enlarged. No signs of congestive failure. Urine showed slight trace of albumin and hyaline casts with a slight tendency toward fixation of specific gravity. Diagnosis—hypertension; chronic nephritis.

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 was heard 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 cast. There was no nitrogen retention. 'Phthalein output was 45 per cent in 2 hours. Diagnosis—hypertension; vascular nephritis.

214. R. M., for 6 weeks, had had marked dyspnea and swelling of extremities. P.E showed the heart markedly enlarged. Sounds were loud, A_2 was accentuated. Rhythm was totally irregular. There was fluid in both chests and in the abdomen, and pitting edema over lower extremities. Patient improved markedly under rest and digitalis and rhythm became regular spontaneously. At time of test there were no signs of congestive failure. His circulation was compensated at rest. Hgb. 70 per cent. Diagnosis—hypertension; auricular fibrillation.

61. H. B. had no history of congestive failure but had pain over left chest. P.E. at time of test showed diffuse pulsation over precordium, with the left border of cardiac dullness 11.5 cm. in the 5th space. Vessels were slightly sclerosed. Diagnosis—hypertension; auricular fibrillation; myocardial degeneration.

196. G. H. One week previous to admission ankles and knees began to swell and there was slight shortness of breath on exertion. Patient was weak and unable to walk. P.E.—heart was moderately enlarged, the second aortic sound was loud and ringing, and a loud, blowing systolic murmur replaced the first sound over the apex. Conspicuous thickening of radial, brachial and femoral arteries, and slight pitting edema of ankles was present. Diagnosis—myocardial degeneration; arteriosclerosis; hypertension.

68. W. H. had had dyspnea and attacks of precordial pain for 6 years, and orthopnea and bloody sputum for 2 months. At time of test patient was in distress, orthopneic, and dyspneic, but had no edema or congestion of lungs. Patient suffered from paroxysmal nocturnal dyspnea. P.E. at time of test showed rapid breathing, heaving cardiac impulse in the 6th space, 14 cm. from the midsternal line, a systolic murmur with very loud booming first sound, and the aortic second sound ringing. The rhythm was totally irregular, rate 70, no pulse deficit. Urine was negative. No nitrogen retention was present. Diagnosis—hypertension; auricular fibrillation.