

## STUDIES ON THE VELOCITY OF BLOOD FLOW

### III. THE VELOCITY OF BLOOD FLOW AND ITS RELATION TO OTHER ASPECTS OF THE CIRCULATION IN PATIENTS WITH RHEUMATIC AND SYPHILITIC HEART DISEASE<sup>1,2</sup>

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#### INTRODUCTION AND HISTORICAL

Previous studies of the cardiovascular system have been in the main concerned with isolated aspects of the circulation. Since an accurate and trustworthy method for measuring the velocity of blood flow had become available, it seemed desirable to learn what changes in the velocity of blood flow occur in cardiovascular disease, and what the relation of such changes might be to other aspects of the circulation.

Preceding communications have described the details of the method employed (1), and the results obtained in studying the velocity of blood flow in a group of normal individuals (2). The velocity of blood flow was estimated by injecting active deposit of radium into the cubital vein of one arm, and recording the onset of the ionization effect when the active deposit of radium reached the arterial vessels about the elbow of the other arm. In a group of normal individuals the arm to arm circulation times ranged from fifteen seconds to twenty-five seconds. In a given normal individual, the circulation time, in successive measurements, was found constant within plus or minus two seconds. This paper presents a study of the circulation time in patients with cardiovascular disease, and attempts to establish the re-

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<sup>2</sup> Some of the determinations of later date in this and the subsequent communication were performed by means of a detecting device a description of which will shortly be published.

relationship between disturbance in the velocity of blood flow and other fundamental aspects of the circulation, such as the cardiac rate and rhythm, arterial and venous blood pressures, and the vital capacity of the lungs.

In the past, studies of certain fundamental features of the pathological physiology of compensated and decompensated heart disease have thrown indirect light on the velocity of blood flow. Such studies have included observations on venous pressure, vital capacity of the lungs, and minute volume output of the heart. The results of the more significant investigations by various observers in these fields may conveniently be summarized under the following headings.

#### *Venous pressure*

As has been pointed out in a previous communication (2) the wide variation in venous pressure measurements in normal individuals found by different observers necessitated establishing our own normal standards.

The most accurate method is that of Moritz and Tabora (3). According to them, normal venous pressure ranges from 1 to 9 cm. of water, but other investigators have obtained greater variations. Schott (4) in twelve normal individuals observed a range of 15 to 125 mm. of water. Bedford and Wright (5) observed a range of from 50 to 201 mm. of water. In a group of normal individuals, the venous pressure, by the method of Moritz and Tabora, was found by us (2) to range from 15 to 100 mm. of water. Observers who have studied patients suffering from disease of the circulatory system are in fair agreement that congestive failure is attended by a significant rise in the venous pressure. Moritz and Tabora found it as high as 20 to 32 cm. of water. Similarly, Frey (6) in 1902, Hooker and Eyster (7) in 1908, and Frank and Reh in 1911 (8), and Clark (9) in 1915 found a clear correspondence between the abnormal rise in venous pressure and the degree of cardiac decompensation. In compensated heart disease, various observers, Fuchs (10), Kroetz (11), Frank and Reh (8) found that it was within the limits of normal.

#### *Vital capacity of the lungs*

That the vital capacity of the lungs is diminished in cardiac decompensation, and that the diminution bears a close relationship to the

degree of decompensation was established by Peabody and Wentworth in 1917 (12). They measured the vital capacity of a group of normal individuals and established normal standards on the basis of height, weight and sex. In studying the vital capacity of a group of patients with various types of heart disease, they found close parallelism between the tendency to experience dyspnea and reduction in the vital capacity.

Reduction in the vital capacity may be due in part to the development of engorgement and increased pressure in the pulmonary circulation. It is therefore of interest to learn what the relationship between engorgement of the pulmonary circulation, as expressed in the vital capacity, is to engorgement of the peripheral circulation, as expressed in the venous pressure measurements, and what the relationship of both is to disturbance in the velocity of blood flow.

#### *Minute volume output of the heart*

With a failing circulation one might expect a decrease in the cardiac minute volume output. Plesch (13) found no diminution in six determinations on six patients showing fully compensated valvular disease. Lundsgaard (14) measured the minute volume output in ten patients according to the nitrous oxide method of Krogh and Lindhard. He found it reduced in patients with valvular lesions showing regular rhythm and congestive failure. It was normal in patients with compensated circulation and with regular rhythm.

In patients with circulatory failure, Lundsgaard (15) in 1918 and Harrop (16) in 1919 found a close relationship between venous oxygen unsaturation in the blood from the cubital vein and clinical evidence of circulatory failure. They inferred that a reduced velocity of blood flow in the arm was responsible for the increased oxygen unsaturation in the venous blood in their patients.

G. N. Stewart, (17), by means of the calorimetric method, also found the blood flow diminished in the hands of patients suffering from cardiac decompensation.

#### *Velocity of blood flow*

E. Koch (18) in 1922, injected fluorescein into the vein of one arm, and by observing the time of its arrival in the corresponding vein of

the other arm, found the circulation time prolonged in patients with edema and other signs of failure. The circulation time of patients whose circulation was compensated was slightly prolonged in the majority of determinations, and was within the limits of normal in a smaller group of individuals. His results indicate only a general relationship between velocity of blood flow and degree of decompensation. The limitations of the fluorescein method, and the difficulty of estimating the exact onset of the appearance of the fluorescein is subject to the considerations discussed in a preceding communication (2).

Since previous studies of the blood flow in man have been in the main concerned with isolated aspects of the circulation, and since we wished to study the relationship of various circulatory phenomena both to each other and to the velocity of blood flow as measured by our method, we undertook the following investigation.

#### METHODS

In a group of patients with pathological signs and symptoms of the circulatory system, careful histories and physical examinations were made. The height, weight and vital capacity were ascertained. The ventricular rate was counted before and after measurement of the velocity of blood flow. Electrocardiographic tracings were frequently taken to elicit further evidence of cardiac abnormality. In some patients who exhibited abnormal rhythm, electrocardiographic tracings were taken during the time of the test. The venous pressure was measured immediately before the active deposit of radium was injected. The blood and urine were examined. The velocity of blood flow from the cubital vein of one arm to the cubital arterial vessels of the other arm was measured. The procedure used in measuring the velocity of blood flow was that described in a previous communication (1).

The results of these measurements are presented below. The findings are tabulated according to the etiology of the disease regardless of the state of compensation. A short abstract of the history and physical examination with especial reference to the cardiorespiratory system is appended. Positive findings are given, and negative data are omitted. We have correlated the velocity of blood flow with the actual degree of compensation as observed clinically.

#### I. THE VELOCITY OF BLOOD FLOW AND ITS RELATION TO OTHER ASPECTS OF THE CIRCULATION IN PATIENTS WITH RHEUMATIC HEART DISEASE

Rheumatic heart disease affords a better opportunity to study certain phases of circulatory pathology than heart disease due to other

causes since the time at which the cardiac involvement commences can often be established by the history of antecedent rheumatic fever. The rheumatic infection tends, moreover, to attack the valves, and so produces physical signs which enable one to recognize the valvular damage and the rate of development of subsequent changes. But it is common experience that the degree of valvular damage indicated by the physical signs is not necessarily closely related to the symptoms experienced by the patient. In a given individual with regular rhythm, the degree of circulatory disability represents the accumulated effect of both valvular and myocardial damage. We have therefore attempted to learn what effect rheumatic fever has on the velocity of blood flow and other related aspects of the circulation independent of any valvular changes by studying a group of patients who had had rheumatic fever, but who showed no physical signs of valvular involvement. In contrast to this group of patients we have investigated the effect of rheumatic heart disease after it has involved the valves, and finally we have studied the effect of the rheumatic process after it has, in addition, caused absolute arrhythmia of the heart action.

In the analysis of circulatory failure it is of importance to learn whether changes in the arterial and venous pressure, vital capacity of the lungs, and velocity of blood flow which manifest themselves in different types of heart disease always occur in the same sequence or whether they bear a different relation according to the etiology of the circulatory disturbance. What these relations are, we have attempted to ascertain.

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

We have classified our patients suffering from the sequelae of acute rheumatic fever into three groups (table 1). Group A consists of patients who, at the time of admission to the hospital, suffered from acute rheumatic fever, but who, at the time of measurement of the velocity of blood flow, were free from the signs and symptoms of the acute inflammatory process. They presented evidence of valvular lesions. The first three patients had recently recovered from acute rheumatic fever. The only evidence of myocardial involvement was the irritability of the heart. The arm to arm velocity of blood flow was normal or



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

September 30, 1926.....	258	D. S.	Mitral stenosis and insufficiency, aortic insufficiency	40	1.60	64	130	85	40	—	2,300	1,430	30	19	
October 19, 1926.....	265	S. C.	Mitral stenosis and insufficiency aortic insufficiency	26	1.5	97.4	75	105	40	42	5.5	2,650	1,766	29	19.3
June 23, 1926.....	224	S. C.	Mitral stenosis and insufficiency, aortic insufficiency, adhesive pericarditis	26	1.53	n	56	128	64	105	6.9	2,550	1,660	34	22
September 1, 1925.....	13	F. M.	Mitral stenosis and insufficiency	43						10.2				53	
August 28, 1926.....	12	F. M.	Mitral stenosis and insufficiency	43			55			3.8				55	
							65								

\* To conform to the level of the right auricle, 50 mm. should be added to these figures.

slightly more rapid than that found in normal persons. This finding is an objective confirmation of the clinical observation that patients after rheumatic fever just as after other acute infectious diseases often exhibit signs of cardiac hyperactivity with forcible, rapid precordial pulsation, a tendency to low blood pressure, a somewhat increased pulse pressure, flushed face and skin, and an absence of signs of decompensation. The subjective complaints of precordial pain and tenderness, and palpitation of the heart may also be interpreted as a result of this overactivity of the heart.

Two of the patients, R. L. and F. C. (nos. 18 and 292), had had previous attacks of rheumatic fever, whereas S. G. (no. 213) was suffering from his first attack. We can offer no explanation why in patient R. L. (no. 18) the venous pressure was slightly elevated and the vital capacity was reduced. In patients S. G. and F. C. (nos. 213 and 292), who had both experienced shortness of breath on exertion during the preceding year but in whom the circulation was entirely compensated at the time of test, the vital capacities, the venous pressures and the electrocardiographic tracings were normal. In these patients the increase in ventricular rate tended to be associated with a more rapid blood flow, a relationship which is similar to a tendency noted in our previous study of normal individuals (2).

In contrast to these patients, F. G. (nos. 218, 221, and 267) was suffering from chronic myocardial failure without evidence of valvular disease as a result of rheumatic fever experienced several years previously. His tests are of particular interest. Four months before his first entry, he was troubled by breathlessness and orthopnea. He had never noted swelling of the legs. He was cyanotic and dyspneic. The physical signs of the heart were normal, except that at times a soft systolic murmur was heard over the apex, and there was slight cardiac enlargement. The electrocardiographic tracings showed sinus arrhythmia with left ventricular preponderance, T3 inverted, a depressed S-T interval, S3 notched, occasional premature ventricular beats, partial heart block (P-R 0.20 second). The first two measurements were made on successive days when his condition was essentially the same. The ventricular rates corresponded closely, inasmuch as they were 38 at the first test and 40 at the second. The vital capacities were 2950 cc. (1770 cc. per square meter) on the first occasion, and



2850 cc. (1700 cc. per square meter) on the second. His condition improved and he left the hospital. Although he refrained from work and felt comfortable at rest, dyspnea on exertion continued. A few days before his second entry he suffered a recurrence of orthopnea and was forced to use four pillows. On admission there was marked dyspnea and orthopnea. The physical signs were unchanged except that the heart rate was rapid and there were squeaking rhonchi over the bases of the lungs. The circulation time was thirty-one seconds, or nineteen seconds per square meter of body surface. The vital capacity was 2000 cc. or 1200 cc. per square meter of body surface. Although his ventricular rate was 142, or about three and a half times its previous rate, his circulation time was found to be practically the same as it had been on a previous occasion. These observations are an exception to our general findings since his circulation time at the time of the third measurement when the clinical condition was worse was only two seconds longer than that found on two previous occasions. The discrepancy may be due to the fact that the ventricular rate at the time of the first two observations was 38 and 40, whereas at the time of the third test it was 142. One week later, the patient developed acute dyspnea, was unable to walk, his heart rate was 120 per minute, venous pulsations in the neck appeared, the liver edge was very tender at the level of the umbilicus. It was the general opinion that he had no evidence of valvular involvement and that his condition was one purely of rheumatic myocarditis. In this instance, therefore, we had the opportunity to observe in a young patient the uncomplicated effects of damage to the myocardium.

*B. Patients with rheumatic valvular heart disease with regular rhythm*

Group B consists of patients suffering from valvular disease of rheumatic origin with regular rhythm. The arm to arm circulation times were normal or slightly prolonged with the exception of T. Th. (nos. 10 and 11) whose circulation was severely decompensated at the time of the determination. The vital capacities of these patients were reduced, the venous pressures were normal.

A comparison of the clinical findings with the results of the circulatory measurements establishes the following relationship. Five of these patients, T. H. (no. 219), K. N. (no. 287), W. O'B. (no. 283),

A. M. (197), H. B. (no. 167), never had suffered congestive failure; their vital capacity was, however, reduced; the arm to arm velocity was within the limits of normal, and the venous pressure was normal. W. O'B. (no. 283) typifies our findings in this group. He was 42 years of age at the time of the test and had had rheumatic fever at the ages of fourteen and twenty-four. He had never been aware of any circulatory disturbance although at the time of a life insurance examination, eighteen years previously, he had been informed that he had aortic and mitral valvular heart disease. During the past eighteen years he led an active life as a salesman without restricting his activities. He showed the clinical signs of mitral stenosis and regurgitation and aortic stenosis and regurgitation. The systolic blood pressure was 142 and the diastolic 42. Although the patient had a definite history of valvular disease for eighteen years, it is noteworthy that his circulatory function was entirely adequate for a fairly vigorous life. His normal circulation time is in accord with his functional activity. Nevertheless his vital capacity, and similarly the vital capacities of the other patients whose circulation was compensated were definitely reduced.

In contrast to the five patients just cited J. V. (no. 191) had been troubled with shortness of breath on exertion in the past, but he showed no evidence of congestive failure. The velocity of blood flow was nevertheless reduced. Patient T. Th. (nos. 10 and 11) is the only person in this group who shows conspicuous prolongation of the circulation time and marked reduction in vital capacity; it should be observed that, similarly, he is the only one of the group who exhibited signs of congestive failure at the time of the test.

The appended abstracts of the histories and physical examinations of these patients show that the site and extent of the valvular involvement varied from patient to patient, and that the degree of valvular involvement did not correspond with the degree of circulatory competence.

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

The three patients in this group showed, beside unmistakable clinical evidence of valvular damage, fibrillation of the auricles with complete

irregularity of the ventricular rhythm. They gave histories of dyspnea on exertion but only one patient, F. M. (nos. 12 and 13), showed physical signs of congestive failure at the time the velocity of blood flow was determined. Râles could be heard throughout both sides of his chest, especially at the bases. Determinations nos. 12 and 13 are repeated tests when his clinical condition was stationary; it should be noted that the conspicuous slowing of the velocity of blood flow is in accord with the clinical findings. S. C. (nos. 265 and 224) and D. S. (no. 258) who showed a more rapid velocity of blood flow had regained circulatory compensation at the time of test and their improvement is reflected in the shorter arm to arm circulation times.

#### *Discussion*

Changes in blood flow may depend then (1) on valvular damage, (2) on muscular injury, (3) on both, and (4) on the presence of an abnormal rhythm. Our observations demonstrate that it is not the localization or the type of valvular lesion which determines the functional severity of circulatory failure, but rather the capacity of the myocardium to compensate for the increased burden imposed. We have not yet studied whether one type of valvular lesion predisposes to earlier myocardial insufficiency than another.

In general, the results of the study of patients in group A establishes the fact that rheumatic infections, without valvular pathology, may be associated with normal or increased rapidity of blood flow provided the circulation is compensated. If valvular lesions develop, without evidence of circulatory insufficiency, one may find that the velocity of blood flow is normal; the venous pressure is not elevated, while the vital capacity may be diminished.

When early symptoms of circulatory insufficiency, such as palpitation and dyspnea on exertion, manifest themselves, the vital capacity is reduced, and the velocity of blood flow is definitely retarded; but the venous pressure may still remain within the limits of normal. If, however, signs of congestive failure appear, the venous pressure becomes elevated, and the vital capacity and the velocity of blood flow still further deviate from the normal.

This sequence of events in circulatory insufficiency exhibited in the course of rheumatic fever is of both theoretical and practical interest.

The facts gathered in the preceding section indicate that the immediate effect of rheumatic infection leads to overactivity of the heart, which with the attendant vasodilatation, results in a tendency to an increased velocity of blood flow. It is of particular interest to learn that the velocity of blood flow is within the limits of normal in such patients, and that it may remain so even after the valves have undergone changes sufficient to give unmistakable clinical evidence of their distortion. With the development of mitral valvular disease, the vital capacity may be reduced, while the velocity of blood flow from arm to arm is still normal or slightly prolonged. The reduced vital capacity in the absence of signs of congestive failure has led to the suggestion that the limitation in the excursion of the lungs may be due to diminished elasticity because of engorgement of the pulmonary vessels under somewhat increased pressure, and is not necessarily referable to the respiratory center. This suggestion is in accord with the early appearance of dyspnea in patients with mitral stenosis. In some of our patients the diminution in vital capacity occurred while the velocity of blood flow was normal. While the arm to arm velocity expresses the speed of blood flow through the arms as well as through the pulmonary circulation, the finding of a normal circulation time in the presence of lowered vital capacity suggests that the blood flow through the upper lung fields may be accomplished with normal speed, whereas engorgement and reduced speed of blood flow occurs in the lower portions of the lung.

Since our method enables us to measure only the speed of the fastest particle, we cannot offer evidence to support on this hypothesis all the phenomena which occur. But the early reduction in vital capacity, the early appearance of breathlessness, the presence in the early stage of the disease of a normal arm to arm velocity of blood flow, the common clinical experience of finding râles first at the bases of the lungs—all these facts are in accord with the hypothesis that the velocity of blood flow through the upper portions of the lung fields may be normal whereas that through the more dependent portions may be retarded, because of the "sedimentation" (19) of the blood. Measurement of the minute volume output of the heart in such patients, and a closer estimation of the pulmonary circulation time, would be of great interest in this connection. A later communication will deal more specifically with this question.

## II. THE VELOCITY OF BLOOD FLOW AND RELATED ASPECTS OF THE CIRCULATION IN PATIENTS WITH SYPHILITIC HEART DISEASE

Whereas rheumatic fever usually attacks the mitral valve, syphilis involves most frequently the aorta and aortic valve. In mitral valvular disease, with the engorgement of the pulmonary circulation, dyspnea is an early symptom, whereas in aortic valvular insufficiency dyspnea does not appear until later in the course of the disease when the left ventricle is no longer able to carry on the increased amount of work. Dyspnea is of more serious prognostic significance than the same symptom appearing in patients with involvement of the mitral valve.

Examination of the data in table 2 reveals the fact that the patients exhibit lowered vital capacities, and that the velocity of blood flow was reduced beyond the limits of normal in all patients except in patients J. P. (no. 240), J. C. (no. 293), and T. B. (no. 91). The venous pressure was found slightly elevated or within the limits of normal.

It is of considerable interest that in J. P. (no. 240) and T. B. (no. 91), both of whom complained of precordial pain and shortness of breath, the velocity of blood flow was within the limits of normal. Neither patient had had at any time signs of congestive failure. The occurrence of these symptoms when the blood flow is normal suggests that in syphilitic aortitis, breathlessness and cardiac pain may be due to a reflex nerve mechanism, and not necessarily to congestion of the pulmonary bed. The findings in patients suffering from rheumatic heart disease favor this hypothesis, for it was observed that if dyspnea was present at the time of the test the circulation times were invariably prolonged.

Further evidence of the lack of relation of pain and dyspnea on the one hand, and of underlying disturbance in the dynamics of the circulation on the other, in patients suffering from syphilitic heart disease is shown by the results of the tests in J. H. (no. 203). He entered the hospital because of lobar pneumonia. He did not show pain, dyspnea, or other signs or symptoms of cardiac decompensation, although the vital capacity was low, and the velocity of blood flow was definitely reduced. It is noteworthy that objective measurements of the velocity of blood flow gave evidence of impaired function before

TABLE 2  
*The velocity of blood flow and its relation to other aspects of the circulation in patients with syphilitic heart disease*

Date	Test number	Name	Diagnosis	Age	Surface area sq. m.	Pulse	Arterial pressure		*Venous pressure mm. H <sub>2</sub> O	Injected milli-curies	Vital capacity cc.	Vital capacity per square meter	Circulation time seconds	Circulation time per square meter seconds
							Systolic mm. Hg.	Diastolic mm. Hg.						
1926														
September 2.....	240	J. P.	Aortic aneurysm		1.72	84	140	75	75	10.0	2,500	1,453	17	9.8
November 4.....	293	J. C.	Aortic insufficiency	52	1.92	82	162	0	—	4.0	2,750	1,434	20	10.4
January 15.....	91	T. B.	Aortic insufficiency	54	1.74	63	150	40	15	4.3			19	11
October 26.....	276	J. C.	Aortic insufficiency	52	1.92	70	136	0	35	5.0	2,700	1,406	22	11.4
June 16.....	203	J. H.	Aortic insufficiency	54	1.77	87	124	38	52	2.6	2,950	1,660	26	15
January 27.....	112	W. H.	Aortic insufficiency	53	1.69	80	192	0	50	5.0	3,150	1,860	29	17
October 21.....	271	A. S.	Aortic aneurysm	57	1.58	89			75	4.5	2,300	1,455	32	20.2
June 14.....	192	I. K.	Aortic insufficiency	48	1.67	96	130	48	40	5.4	1,600	958	35	21
September 22.....	243	W. H.	Aortic insufficiency	54	1.62	88	110	40	—	5.4	3,000	1,851	36	22.2
September 2.....	239	W. H.	Aortic insufficiency	54	1.66	88			28	6.9	2,550	1,550	43	25.9
June 28.....	234	W. H.	Aortic insufficiency	54	1.66	96	125	50					48	29

\* To conform to the level of the right auricle, 50 mm. should be added to these figures.

he himself was aware of symptoms and before any clinical signs appeared, because six months later he entered the hospital, cyanotic, dyspneic, orthopneic and edematous. He died five days after admission and autopsy showed the presence of cardiac hypertrophy, aortitis, and aortic insufficiency of syphilitic origin. In this instance, therefore, the circulation time was the first sign of heart failure and was therefore of prognostic significance.

The two patients, I. K. (no. 192) and W. H. (nos. 112, 243, 239, and 234) who showed the greatest retardation of blood flow, entered the hospital with pronounced signs of chronic passive congestion. I. K. (no. 192), who showed pulmonary congestion and swelling of both legs at entry, improved markedly and when his circulation time was measured he had no edema, no orthopnea and no dyspnea. The circulation time was thirty-five seconds, the vital capacity was 1600 cc. and the venous pressure was at the extreme upper limit of normal.

The other patient, W. H. (nos. 112, 243, 239, and 234), who showed conspicuous retardation of blood flow, is especially interesting for repeated measurements were obtained several times during his stay in the hospital when his clinical condition showed definite changes. Previous to his first test he had experienced paroxysmal nocturnal dyspnea for one year. On the morning of the test day his circulation was compensated and he was free from breathlessness. The velocity of blood flow was twenty-nine seconds; the vital capacity was 3150 cc. On June 28, 1926, the time of the second test, he still experienced pronounced orthopnea and showed edema of the ankles, two features which had not been present at the time of the first test. Clinically, therefore, his condition was worse. His circulation time was found to have become prolonged to forty-eight seconds, the vital capacity was reduced to 2550 cc. and the venous pressure was 2.8 cm. of water, still within the limits of normal. Sixty days later his clinical condition improved, for, though orthopneic, edema of the ankles had disappeared. His circulation time was shorter, i.e., forty-three seconds. Under full doses of digitalis and rest in bed, the shortness of breath disappeared and he was able to walk about the ward and three weeks later, his circulation time was thirty-six seconds and his vital capacity, 3000 cc. In short, the circulation time ran parallel to the other signs.

*Discussion*

These observations on syphilitic patients indicate that there is a definite relationship between the degree of circulatory sufficiency and the arm to arm velocity of blood flow, and that the latter measurement is an objective and in general a quantitative index of the state of the circulation. Observations before, during and after the disappearance of the signs of chronic passive congestion indicate that the circulation time and the vital capacity may show deviations from the normal before the clinical signs of congestive failure appear. In patients whose condition is improving the velocity of blood flow and the vital capacity may increase before the clinical evidences of chronic passive congestion disappear and the patient has regained full circulatory compensation. Contrary to our general experience, certain patients with syphilitic aortitis although they exhibit symptoms of breathlessness, precordial pain and lowered vital capacities, nevertheless have normal velocities of blood flow. These findings support the opinions of those investigators who believe that pain and dyspnea in patients with syphilitic aortitis may be due to a reflex mechanism rather than to chronic passive congestion.

A further discussion of the general aspects of the findings presented in this communication will be given in another paper (Paper V of this series).

## SUMMARY

I. The velocity of blood flow and its relation to other aspects of the circulation have been studied in patients with rheumatic heart disease. We have divided these patients into the following groups:

*A. Without valvular damage.* 1. Six measurements of the arm to arm circulation time, of the venous pressure, and of the vital capacity were made on four patients and correlated with the clinical findings.

2. Three patients showed a normal or slightly increased velocity of blood flow which was associated with clinical evidences of exaggerated cardiac activity following acute rheumatic fever.

3. Three measurements of the velocity of blood flow in a young adult who exhibited the symptoms and signs of severe myocardial damage due to rheumatic fever, showed a slightly prolonged velocity of blood flow.



*B. With valvular disease and regular rhythm.* 1. Eight measurements of the circulation time and related aspects of the circulation were made on seven patients.

2. The circulation times were normal, or slightly, or greatly prolonged according to the clinical condition of the patients.

3. Patients who had never shown the signs or symptoms of cardiac decompensation had circulation times that were within the limits of normal. The vital capacities of these patients tended to be reduced; the venous pressures were normal.

4. Our observations indicate that the circulation times in patients with rheumatic heart disease reflect the dysfunction due to myocardial damage.

5. The site and extent of the valvular heart disease varied from patient to patient. 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 disease and fibrillation of the auricles.* 1. In five measurements of the velocity of blood flow on three patients the circulation times ranged from twenty-nine seconds to fifty-five seconds, and bore a definite relationship to the degree of circulatory compensation.

II. The velocity of blood flow and related aspects of the circulation were studied in patients with syphilitic heart disease.

1. Eleven measurements of the arm to arm circulation time, of the venous pressure, and of the vital capacity were made on seven patients suffering from lesions of the aortic valve.

2. In all patients in this group in whom the circulation time was prolonged beyond the extreme upper limit of normal there was evidence of circulatory failure. The vital capacities were reduced; the venous pressures were normal or slightly elevated. The degree of prolongation of the circulation time corresponded to the degree of circulatory failure.

3. When cardiac pain and dyspnea, but no signs of congestive failure are present, normal velocity of blood flow may be present. This suggests that breathlessness and cardiac pain may, in patients with syphilitic aortitis, be due to a reflex mechanism, and that they are not necessarily due to congestion of the pulmonary bed.

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## APPENDIX

## I. ABSTRACTS OF HISTORIES AND PHYSICAL EXAMINATIONS OF PATIENTS WITH RHEUMATIC HEART DISEASE

213. S. G. had fever and painful and swollen joints five months previously. At time of test, occasional fleeting pains were referable to various joints. There was no history of cardiac failure. P.E. was negative except for a faint systolic murmur over the apex. Heart rate was slightly rapid, 80-95. Diagnosis—acute rheumatic fever.

18. R. L. had had repeated attacks of tonsillitis and swelling of joints during previous twelve years. He entered hospital with swollen ankles. Later, his knees and right hip became painful and tender. P.E.—heart sounds were of good quality. Both knees and ankles were swollen, hot and tender. Hgb. 90 per cent. W.B.C. 11,500. Diagnosis—acute rheumatic fever.

292. F. G.: For two months there was 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. P.E. was entirely negative at time of test. Diagnosis—acute rheumatic fever.

218, 221, 267. F. G. had had numerous attacks of tonsillitis in the past, but there was no history of rheumatic fever. Four months previous to tests 218 and 221, he developed gradually increasing dyspnea. Orthopnea appeared three weeks before test with occasional pain over the epigastrium. There was no history of congestive failure. P.E.—apex in the 5th space. Left border dullness 12 cm. The sounds were regular. Systolic murmur heard at apex. Lungs were clear. Liver was not palpable. There was no edema. Electrocardiographic tracing at time of first test showed left ventricular predominance, T3 inverted, S3 notched, depressed S-T interval, occasional ventricular extra systoles. These signs may have been due to digitalization. After discharge from hospital, patient was unable to work and dyspnea on exertion continued. A few days before second entry he became suddenly orthopneic and entered the hospital showing marked arterial pulsations in the neck. The heart action was rapid. Otherwise signs were the same as at previous entry. There were squeaking râles over chest posteriorly. The liver was not palpable and there was no edema of the ankles. Diagnosis—rheumatic myocarditis.

219. T. H. had had repeated attacks of swollen and painful joints for thirty-eight years, but no history of shortness of breath or congestive failure. P.E.—Heart was enlarged. Left border dullness 12 cm. First sound over apex was loud and booming and the second sound over the pulmonic area was accentuated and loud. Short presystolic murmur preceded the first sound. Blowing systolic murmur was heard over entire precordium. The liver edge was palpated about 4 cm. below costal margin at entrance but was not palpated at time of test. R.B.C. 4,005,000. Hgb. 75 per cent. Diagnosis—subacute rheumatic fever with mitral disease.

287. K. N. entered hospital because of pain and swelling of both knees. He had had acute rheumatic fever five weeks' duration which responded to treatment by salicylates. At time of test there were no joint signs or symptoms. P. E.—heart: Left border dullness 11.5 cm. from midsternal line in 5th space. First sound was accentuated. Soft systolic murmur at the apex was transmitted to the axilla. There were no diastolic murmurs. Diagnosis—mitral insufficiency.

283. W. O. had had rheumatic fever twenty-eight years and eighteen years previously. At time of insurance examination eighteen 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. P.E. 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 5th 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 murmurs were heard. Loud, long diastolic murmur heard along the left border of sternum and faint diastolic murmur over aortic area. Corrigan pulse and Duroziez' sign were present. Diagnosis—mitral stenosis and regurgitation; aortic regurgitation.

197. A. M. had no definite past history of acute rheumatic fever. No dyspnea or congestive failure. P.E. showed a presystolic murmur with loud, short first sound, the elbow joint red, tender and swollen. Diagnosis—acute rheumatic fever; mitral stenosis and regurgitation.

167. H. B. had had repeated attacks of rheumatic fever for the past ten years. There was no history of dyspnea or congestive failure. He entered hospital with an acute flare-up of rheumatic fever. P.E. showed the apex impulse in 6th interspace, left border of cardiac dullness 14 cm. from the midsternal line. Double murmurs were heard at apex and over aortic area with systolic thrill transmitted to the large vessels of the neck. The lungs were normal. Liver was not palpated. X-ray showed heart enlargement both to the left and to the right. R.B.C. 4,570,000, Hgb. 60 per cent. Diagnosis—rheumatic endocarditis with mitral stenosis and insufficiency, and with aortic stenosis and insufficiency.

10, 11. T. Th. had had increasing dyspnea and weakness for three years, with several attacks of congestive failure. P.E. showed Cheyne-Stokes breathing, left border dullness 17.5 cm. from the midsternal line in the 6th space. Faint systolic blow was heard over the precordium. Chest was broad and definitely

emphysematous in type. Tender liver edge felt 10 cm. below right costal margin in the nipple line. Blood pressure was 148 systolic and 100 diastolic. Numerous râles were heard over both chests. Pitting edema of both legs was present. Electrocardiographic tracings showed right bundle branch block. Diagnosis—rheumatic myocarditis; mitral stenosis and insufficiency.

258. D. S. had had shortness of breath, attacks of sharp lancinating pain over the heart for several years, following exertion, which was non-radiating, and lasted a minute or two. There was marked orthopnea. One week before entry he coughed up blood-streaked sputum. P.E. showed left border of cardiac dullness 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. Hgb. 75 per cent. Diagnosis—mitral stenosis and insufficiency.

224, 265. S. C. complained of shortness of breath. He had had rheumatic fever in childhood but had been well until nine years previously, when, after pneumonia, he developed moderate shortness of breath for eight months. During the six months before entry he experienced slight precordial pain on exertion with shortness of breath and palpitation. P.E. at time of test 224 showed the heart markedly enlarged, and all sounds obscured by murmurs. Blowing systolic and diastolic murmurs were heard over the apex, soft systolic and diastolic murmurs over the 2nd interspace. A thrill was felt all over the precordium with marked systolic retraction. The heart rate was slow, and the rhythm totally irregular. Suggestive Broadbent sign was present. No râles were heard over the chest. Liver was not palpable. There was no evidence of pitting edema. Hgb. 90 per cent. The patient left the hospital but was soon troubled by a choking sensation associated with pain in the epigastrium. He had frequent palpitation and dyspnea on exertion, and orthopnea for one week before second admission to hospital. Three days before this second 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 second test, 265, he was still slightly orthopneic. P.E. showed heart signs as before noted, and moist râles over left base. Diagnosis—rheumatic pericarditis; auricular fibrillation.

12, 13, F. M. had had dyspnea for three months, orthopnea ten days. There was no history of congestive failure. P. E. showed left border of cardiac dullness 14 cm. from the midsternal line, systolic and diastolic murmurs and a thrill at apex. There were moist râles over both chests, especially at bases. Diagnosis—mitral stenosis and regurgitation; auricular fibrillation.

## II. ABSTRACTS OF HISTORIES AND PHYSICAL EXAMINATIONS OF 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. P.E. was negative except for a systolic murmur

over the base. By x-ray, aneurysm of the aortic arch was observed. Diagnosis—aneurysm of the aortic arch.

276, 293. 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. P.E. at time of first test showed marked arterial pulsations visible in the neck, a heaving apex impulse over the 5th space, left border of cardiac dullness 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. P.E. was as before noted. Diagnosis—*aortic insufficiency; syphilis.*

91. T. B. had had marked dyspnea and cough for nine months but no edema of legs. P. E. (date of test) showed orthopnea, blood-streaked sputum, no signs of edema, pain over aortic region, and cardiac hypertrophy with a diastolic murmur over 2nd left interspace. Diagnosis—*aortic insufficiency.*

203. J. H. entered hospital suffering from lobar pneumonia. He gave no history of cardiac decompensation. Signs of aortic insufficiency were discovered at routine examination. Apex impulse was seen in the 6th interspace. The left border of cardiac dullness was 12 cm. from the midsternal line. Short systolic and long diastolic murmurs were heard over base, especially over left border of the sternum. Wassermann test was positive. Diagnosis—*post-pneumonia and aortic insufficiency.*

112. W. H. had had progressive dyspnea with paroxysmal nocturnal attacks for one year, orthopnea and epigastric pain for two weeks before admission. P.E. showed the apex in the 6th space, 12 cm. from the midsternal line, and systolic and diastolic murmurs over base. Brachial and radial arteries were sclerosed. Corrigan pulse was present. No signs of congestive failure were present. Diagnosis—*aortic insufficiency; syphilis.*

271. A. S. entered hospital because of dyspnea and abdominal pain five weeks in duration, with sharp, precordial non-radiating pain. He noted slight swelling of legs and was troubled by cough. He was forced to use two or three pillows at night, but rapidly improved under rest and digitalis. P.E. showed the sclerae slightly jaundiced, and the heart in 5th space 13 cm. from midsternal line. A double murmur was heard over the aortic area and the tender edge of the liver was felt 3 fingers breadth below right costal margin. There was no edema of legs. The lungs were normal. He could walk on the level without stopping. Kahn test was positive. Fluoroscopy showed aneurysm of the ascending aorta. Diagnosis—*aneurysm of ascending portion of arch of aorta.*

192. I. K. had had shortness of breath and swelling of legs for four weeks, and orthopnea for two weeks. A few days before entry, there was marked swelling of both legs. P.E. showed a heaving diffuse apex beat with maximum intensity in the 6th space where left border dullness was 12 cm. from the midsternal line.

Supracardiac dullness over the left 2nd space was 4 cm. To the right of the sternum there was a palpable impulse. The sounds were regular and of good quality. Over the right of the sternum in 2nd space there were soft systolic and diastolic murmurs. Marked edema of lower extremities at time of entrance was noted. At time of test there was no dyspnea or edema. X-ray examination showed shadow of an aortic aneurysm. Hgb. 110 per cent. R.B.C. 4,300,000. Diagnosis—aortic aneurysm with probable aortic insufficiency.

234, 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 two weeks. P.E. showed at time of admission respiratory distress with orthopnea, the apex impulse in the 6th space, left border of cardiac dullness 13 cm. from midsternal line. Double murmurs were heard over the aortic area. Tender liver edge was felt 3 fingers below costal margin. Slight edema of both ankles was present. At time of 1st test, 234, there was marked dyspnea, orthopnea, slight pitting edema over ankles. At time of 2nd test, 239, there was orthopnea, no congestive failure and he was able to walk slowly. At time of 3rd test, 243, his circulation compensated fairly well at rest and he was able to walk. He felt stronger. Diagnosis—aortic insufficiency; syphilis.