BLOOD PRESSURE AND PULSE RATE REACTIONS *

SECOND PAPER

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During the late war there was an urgent need for some objective test by which the presence and the degree of circulatory disability could be measured. The observations on which this paper is based were made in the attempt to supply this need.

The great majority of men with organic heart disease were rejected before admission to the army, but under the regulations issued for the medical examination of drafted men, it was not possible to exclude those who suffered from the condition known as irritable heart, neuro-circulatory asthenia or effort syndrome, though a considerable percentage was later found to be incapable of active service. In England, Lewis developed a series of graduated drills by which the capacity of these men for service could be estimated. I used this system of exercises at Camp Lewis, both in normal individuals and in cases of irritable heart, but after some months it was discarded because the results were uncertain and the method required too much time. This was chiefly due, I believe, to the fact that Lewis was dealing with trained soldiers, while we had raw recruits. An essential for the success of his plan is that the exercises be carried out with sharpness and precision, so that the amount of effort entailed is more or less uniform. With untrained men, especially men suffering from cardiac, vasomotor and nervous symptoms, we did not succeed in attaining this uniformity. After a considerable experience of military discipline response to orders is almost involuntary. Without this experience movements are slowed and inadequately performed at the first onset of fatigue. The amount of work done varies rather with the psychic state of the individual than with his physical capacity. Time also was of importance in our case. Circumstances were such as to call for a decision in days rather than in weeks.

Efforts were made to use the exercise test recommended from Washington, D. C. This depends on the effect on the pulse rate of change of position and of hopping 100 times. Since no normal standards were given, the pulse rates of 1,000 normal recruits were counted throughout a test which was a slight modification of the official test. From these data statistical standards of normality were prepared.¹ But this method was also abandoned, for reasons which will be discussed in this paper. The pulse rate method was still further developed by

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1. Addis: J. A. M. A. **72**:181 (Jan. 18) 1919.

observing the effect of hopping, first 100, then 200, 300, 400 and 500 times without intermission, except for the short intervals during which the pulse was counted. This test demonstrated that even persons with marked symptoms were capable of the quite severe exertion which this amount of exercise entails, but otherwise it was not found to be of much value.

The test which was actually used developed as a result of a trial of Crampton's method² in which observations of systolic pressure as well as of pulse rate are made before and after rising from the recumbent to the erect position. First, while the subject is lying down, the pulse rate is counted for quarter minute intervals until a constant pulse rate is attained. The systolic pressure is then taken. After the subject stands up the pulse rate is again counted until it becomes constant and then the second systolic pressure reading is made. The results were found to be very variable, and in order to find the causes of this high degree of variability, a consecutive series of blood pressure and pulse readings were made simultaneously at quarter minute intervals before and after the subject altered his position. It then became clear that one reason for the inconstancy of the results lay in the varying time required for the pulse rate to become constant after standing. If this time were very short, a fall in systolic pressure was frequently found. If it took a little longer the pressure was often higher than when the subject was lying down and later still it fell to a more or less constant level. On examining the charted records of these experiments it was seen that the greatest change in systolic pressure occurred immediately after standing and that this effect was usually missed altogether by waiting until the pulse rate became constant.

The system of simultaneous pressure and pulse rate measurements was then applied to the test which had been used in the pulse rate work. Twelve consecutive pressures and pulse rates were taken, six before and six after exercise. Exercise consisted of 100 hops timed by a metronome striking at a rate of 120 to the minute. Pulse rates were counted over fifteen second periods, and each pressure reading was made as nearly as possible in the middle of these periods, which were timed by a watch attached to the sphygmomanometer. A mercury instrument was used which had a 5 inch canvas backed arm band under which a special stethoscope bell was fitted. One hand of the observer was thus free to make the air pressure adjustments while the other held the instrument. The systolic pressure was read at the first appearance of sound as the mercury fell. But in many cases after exercise the sound of the beat of the forcibly pulsating artery against the upper edge of the arm band could be heard, and the first accentuation of sound was then taken as the signal to read the systolic pressure. The diastolic pressure was read

2. Crampton: New York M. J. **98**:916, 1913.

at the commencement of the fourth phase, or at the disappearance of sound when no fourth phase could be distinguished. The blood pressures were taken by myself, the pulse rates by an assistant, usually with a stethoscope over the heart. Simultaneous counts by two assistants, one from the wrist and one from the heart, showed that a considerable degree of accuracy was quickly attained. Another assistant acted as recorder. The effect of change of position was determined both before and after exercise by having the subject lying flat on his back for the first three fifteen second periods and standing erect for the next three periods. The whole test required 230 seconds, the first forty-five lying down before exercise, the second forty-five standing before exercise, the hopping covered fifty seconds and was followed by lying and standing position for two forty-five second periods. Twelve observations were thus obtained from each subject under four successively changing conditions.

SECTION I

THE REACTION OF THE CARDIOVASCULAR SYSTEM IN NORMAL INDIVIDUALS

The average reaction of the cardiovascular systems of normal individuals and its variability under this test was first determined. For this purpose 300 soldiers between the ages of 21 and 31 were chosen. They stated that they were in good health at the time they were examined and none had suffered from any recent illness or infection. All had undergone two or more months of intensive physical training. The averages, coefficients of variation (the standard deviations expressed as percentages of their respective averages) are given in Tables 1 and 2. The frequency distributions for each of the twelve periods will be found in Table 11. The averages are charted in Figure 1.

The conditions of this test constitute in reality two different tests, for the cardiovascular system has to adapt itself to two diverse forms of strain. There is the strain of adjustment to muscular exercise which requires that the nervous system, the heart and the vessels should work together to send a greater supply of blood to the active muscles. And there is the strain, both before and after exercise, of adjustment to a sudden change from a horizontal to a vertical position of the body. This requires an intricate mechanism of adaption from the cardiovascular system to prevent the force of gravity from interfering with the uniformity of blood distribution. Therefore in using the pressure and pulse rate measurements on normal individuals for establishing standards we must distinguish between the effect of exercise and the effect of change of position, and must choose those periods in the series of measurements at which the effects are most marked. It is apparent

Fig. 1.—Normal Persons. Average blood pressure and pulse rate of 300 normal persons showing the effect of change of position and of exercise. The darker shaded areas represent the period during which the subjects were lying down, the lighter areas the period during which they stood up, while the unshaded portion indicates the time during which 100 beats were taken.

from Figure 1 that the most pronounced effect of exercise is seen in the first period after exercise, and that the change from the recumbent to the erect position occurs in the first periods after standing. These are the points at which the effects of the two strains should be estimated. The base from which the degree of effect should be measured is the third period in the lying position in the case of exercise, and the periods

TABLE 1.—AVERAGES OF 310 NORMALS

	Time in Seconds														
	0 15 30 45 60 75 90 140 155 170 185 200 215 230														
	Lying			Standing			Hop- ping	Lying			Standing				
Systolic.....	127	127	125	126	134	131		156	153	147	138	145	141		
Diastolic.....	78	80	81	86	90	91	68	74	78	84	87	87			
Pulse pressure	50	47	44	39	44	41	88	79	70	55	59	54			
Pulse rate.....	80	76	74	99	84	82	101	90	86	102	95	93			
P. P. × P. R.	3960	3700	3380	3950	3790	3380	9030	7190	6040	5630	5930	5050			

immediately preceding standing in the case of the strain induced by change of position. The average effect can be deduced by difference from the averages given in Table 1. Thus exercise raises the systolic pressure from 125 to 156, an increase of 31 mm. of mercury while standing raises the systolic pressure 1 mm. before exercise and is accompanied by a fall of 9 mm. after exercise. It would seem then that normality might be expressed as all those differences which lie within the range of the average differences + and — some measure of variability of the measurements.

TABLE 2.—COEFFICIENTS OF VARIATION $\frac{\sigma}{\text{Av.}} \times 100\%$
300 Normals

	Time in Seconds														
	0	15	30	45	60	75	90	140	155	170	185	200	215	230	
	Lying Before Exercise, %			Standing Before Exercise, %			Hop- ping	Lying After Exercise, %			Standing After Exercise, %				
Systolic.....	13	13	12	13	13	12		13	13	14	13	13	13		
Diastolic.....	14	14	12	15	13	13		17	15	14	14	14	13		
Pulse pressure	28	28	29	27	27	26		21	22	23	29	25	26		
Pulse rate.....	16	16	16	16	18	18		15	15	15	15	17	17		
P. P. × P. R.	38	36	36	33	36	34		28	28	30	33	31	33		

But to adopt this plan would be to neglect the possibility that the level of the base may influence the difference induced by strain. It may be that exercise produces a greater difference in a subject with low blood pressure and slow pulse than it would if before the exercise the cardiovascular system were already in a state of hyperactivity. If that is so each initial level will have its own peculiar limits of variation.

In order to decide this preliminary question the data were arranged so that the amount of difference induced in subjects with initially low pressure and slow pulse could be compared with the difference produced in subjects with relatively high pressure and rapid pulse. The average

TABLE 3.—RELATION BETWEEN THE INITIAL LEVEL OF PRESSURE OR PULSE RATE AND THE EFFECT PRODUCED BY STRAIN

Group	Effect of Exercise			Effect of Change from Horizontal to Vertical Position					
	Number of Subjects in Group	Average of Group Before Exercise	Change in Average of Group After Exercise	Standing Before Exercise			Standing After Exercise		
				Number of Subjects in Group	Average of Group Before Standing	Change in Average of Group After Standing	Number of Subjects in Group	Average of Group Before Standing	Change in Average of Group After Standing
Systolic Pressure									
101-110	36	109	+30	36	109	+3			
111-120	78	118	+31	78	118	+2	25	119	-3
121-130	75	128	+33	75	128	-2	40	128	-5
131-140	50	138	+30	50	138	-4	54	139	-5
141-150	28	148	+32	28	148	-2	56	149	-11
151-160							47	158	-13
161-170							42	168	-15
Diastolic Pressure									
51- 60							24	59	+9
61- 70	61	69	-10	61	69	+7	86	69	+9
71- 80	110	79	-12	110	79	+6	105	78	+3
81- 90	80	88	-14	80	88	+4	61	88	+5
91-100	32	98	-17	32	98	+2			
Pulse Pressure									
21- 30	41	28	+46	41	28	+1			
31- 40	93	38	+44	93	38	-3			
41- 50	101	48	+41	101	48	-7	30	49	-8
51- 60	43	57	+43	43	57	-11	58	58	-10
61- 70							77	68	-13
71- 80							60	78	-16
81- 90							32	88	-19
91-100							22	98	-21
Pulse Rate per Minute									
53- 60	33	58	+27	33	58	+19			
61- 68	77	66	+26	77	66	+19			
69- 76	84	74	+29	84	74	+18	64	74	+16
77- 84	43	82	+24	43	82	+16	74	82	+16
85- 92	36	89	+29	36	89	+19	71	90	+14
93-100							38	97	+15
P. P. × P. R.									
1000-1999	37	1653	+4522	37	1653	+777			
2000-2999	85	2595	+5235	85	2595	+520			
3000-3999	107	3425	+5825	107	3425	+245	24	3543	+225
4000-4999	37	4375	+6075	37	4375	-40	53	4452	+ 83
5000-5999	25	5455	+6675	25	5455	-250	67	5404	-379
6000-6999							58	6343	-256
7000-7999							40	7412	-955
8000-8999							26	8419	-843

differences for groups with progressively increasing initial levels are given in Table 3.

In the case of the P. P. × P. R. product and also of the pulse pressure and the diastolic pressure, the absolute difference induced by strain is shown in Table 3 to be dependent on the level from which the difference is measured. On the other hand, the pulse rate figures show no

dependence and in the case of the systolic pressure there is an approximately uniform increase after exercise whether the initial level is low or high.

Porter and Richardson³ made a series of observations on various species of animals in order to determine the effect of variations in the initial level of mean blood pressure on the absolute increase caused by a uniform stimulus. They found that with initial pressures from 20 to 160 the increase was approximately the same whether the initial pressures were high or low. But neither their figures, nor those given here for the systolic pressure after exercise are sufficiently numerous to exclude the possibility of a slight degree of dependence. For the changes produced by strain in the different groups are as constant for the pulse rate as for the systolic pressure, and yet when a larger number of observations are dealt with it can be demonstrated that the higher the original level of pulse rate the less is the absolute increase produced

TABLE 4.—RELATION BETWEEN THE INITIAL LEVEL OF PULSE RATE AND THE EFFECT PRODUCED BY STRAIN. PULSE RATE PER MINUTE FROM DATA ON 1,000 NORMAL RECRUITS

Group	Effect of Exercise			Effect of Change of Position: Standing Before Exercise		
	Number of Subjects in Group	Average of Group Before Exercise	Increase in Average of Group After Exercise	Number of Subjects in Group	Average of Group Before Standing	Increase in Average of Group After Standing
60-69	193	65	+33	193	65	+20
70-79	305	74	+29	305	74	+17
80-89	358	83	+28	358	83	+15
90-99	78	94	+26	78	94	+14

by exercise and change of position. These data were obtained from counts made on 1,000 normal recruits under conditions very similar to those described here.¹

The "probable difference between the averages" of the increases of consecutive groups were calculated and were found to be several times smaller than the actual differences. This fact taken in conjunction with the existence here of a double series both showing a progressive decrease excludes the possibility that these differences can be accidental.

If an adequate number of observations were available, it might be found that a similar minor degree of dependence on initial level existed in the case of the systolic pressure also. But since in any case it has been shown that the original level is a factor in the change produced in all the other measurements, it is essential that the records after strain be grouped in accordance with the order of magnitude of the level before strain. It is only from the averages of these groups and

3. Porter and Richardson: *Am. J. Physiol.* **23**:131, 1908.

from a measure of their variability that a standard may be deduced which will be applicable to all cases no matter how variable the level before strain.

The usual measure of variability—the standard deviation—is applicable only in the event of the data grouping themselves in a “normal”

TABLE 5.—AVERAGES AND STANDARD DEVIATIONS AFTER STRAIN ARRANGED IN ACCORDANCE WITH THE LEVEL OF PRESSURE OR PULSE RATE BEFORE STRAIN

Group	Basal Average		After Strain						Basal Average		After Strain	
	Before Exercise or Standing		After Exercise				After Standing		Before Standing After Exercise		After Standing After Exercise	
	Third Period		First Period		Third Period		First Period		Third Period		First Period	
	No.	Av.	Av.	σ	Av.	σ	Av.	σ	No.	Av.	Av.	σ
Systolic Pressure												
101-110	36	109	138	± 13	131	± 12	112	± 11				
111-120	78	118	149	± 13	139	± 11	120	± 11	25	119	117	± 8
121-130	75	128	161	± 15	153	± 14	126	± 11	40	128	123	± 12
131-140	50	138	168	± 12	160	± 11	134	± 13	54	139	134	± 10
141-150	28	148	180	± 11	170	± 14	147	± 15	56	149	138	± 11
151-160									47	158	145	± 13
161-170									42	168	153	± 15
Diastolic Pressure												
51-60									24	59	68	± 9
61-70	61	69	60	± 9	69	± 8	76	± 8	86	69	78	± 9
71-80	110	79	68	± 9	76	± 8	85	± 9	105	78	82	± 7
81-90	80	88	73	± 10	82	± 11	92	± 10	61	88	94	± 9
91-100	32	98	81	± 11	89	± 10	99	± 7				
Pulse Pressure												
21-30	41	28	74	± 15	54	± 13	29	± 8				
31-40	93	38	83	± 15	65	± 11	36	± 8				
41-50	101	48	90	± 13	72	± 13	41	± 10	30	49	41	± 9
51-60	43	57	102	± 13	83	± 13	47	± 11	58	58	48	± 10
61-70									77	68	55	± 10
71-80									60	78	62	± 13
81-90									32	88	69	± 15
91-100									22	98	78	± 16
Pulse Rate												
53-60	33	58	85	± 10	71	± 6	76	± 10				
61-68	77	66	92	± 8	79	± 9	85	± 8				
69-76	84	74	102	± 11	86	± 8	92	± 8	64	74	90	± 10
77-84	43	82	106	± 11	89	± 7	97	± 7	74	82	98	± 8
85-92	36	90	118	± 11	100	± 9	109	± 10	71	80	104	± 10
93-100									38	97	112	± 9
P.P. $\times$ P.R.												
1000-1999	37	1653	6175	± 2093	4045	± 1183	2430	± 650				
2000-2999	85	2595	7830	± 1750	5180	± 1162	3115	± 830				
3000-3999	107	3425	9250	± 1769	6195	± 1338	3670	± 940	53	3545	3770	± 832
4000-4999	37	4375	10450	± 1766	7125	± 1225	4335	± 1255	24	4452	4535	± 924
5000-5999	25	5455	12130	± 1835	8500	± 1667	5205	± 1060	67	5404	5025	± 1027
6000-6999									58	6343	6087	± 1283
7000-7999									40	7412	6457	± 1500
8000-8999									26	8419	7576	± 1867

curve. The frequency distributions given in Table 11 show that this may be assumed to be the case for blood pressure and pulse rate observations.

In Table 5 the averages and standard deviations for the periods best adapted for revealing the effect of strain are given. The data in

Fig. 2.—Standards derived from observations on 300 normal persons. The upper part of the figure is a standard for the P.P. $\times$ P.R. product immediately after exercise. Since the effect of exercise depends in part on the level of the product before exercise, the P.P. $\times$ P.R. observed in the third period while lying down is plotted along the abscissa, so that the average and variability for any initial level may be determined. The chart shows that with an initial product of 2,000, the average product after exercise will be 6,860; with an initial product of 5,000, the product after exercise will be 11,100, and so on. If any observed product falls below the average on the upper curve (the average — 1.75 times the standard deviation), the odds are that one out of every twenty-five normals might have as low or a lower product. If the observed product falls on the lower curve (the average — 3 times the standard deviation) the odds are that only one out of every 740 normals would have as low or a lower product. The odds for products falling between the two curves are indicated on the chart. Products above the upper curve may very well be “normal.” Products below the lower curve are almost certainly “abnormal.” Those which lie between are doubtful in varying degree according to their degree of deviation from the average. The lower part of the figure is a similar standard for P.P. $\times$ P.R. products in the first period after standing before exercise.

the periods before strain were grouped in accordance with their order of magnitude, and the corresponding measurements after strain for each group were averaged and the standard deviation determined. Thus, for instance, the systolic pressures after exercise of all those individuals whose systolic pressure before exercise lay between 101 and 110, were averaged, and so on. From these figures standards of any degree of rigidity may be obtained. For practical purposes it may be regarded as almost certain that no normal individual will deviate more than ± 3.0 times the standard deviation from the average. The odds are that only one individual out of 740 will vary as much or more than this from the mean. With lesser degrees of deviation the chances that the measurement might be such as would be given by a normal individual rapidly increase until at the average ± 1.75 times the standard deviation the odds are that one out of every twenty-five normals might deviate so far from the average. Between these two limits lie the cases which may be either normal or abnormal, the chances varying according as they approach the lower or the upper limit. In cases beyond the limit farthest from the average there is practically no chance of normality; in cases nearer the average than the other limit, the chances of normality are so considerable that there is not sufficient reason for raising the hypothesis of abnormality.

In Figure 2 these limits are charted from smoothed curves derived from the data given in Table 5. In order to save space only the limits for the P. P. $\times$ P. R. product have been charted, and only the limits below the average after exercise and change of position have been defined because these standards are sufficient for the grading of the more important abnormalities found in the patients we examined. For cases with other types of abnormality similar standards can be easily prepared from Table 5.

SECTION II

THE SIGNIFICANCE OF THE PRESSURE AND PULSE RATE REACTIONS OF NORMAL INDIVIDUALS

In this section the reasons why exercise and change of position should produce the effects on pressure and pulse rate which we have noted are considered. The effects under consideration will be more easily followed by reference to Figure 1. In this chart, and in all the others given in this paper, the darker shading indicates that the subjects were lying down, the lighter that they were standing. The unshaded portion of the chart is the period during which they were hopping. The curves indicate that exercise increases the pulse pressure, and the pulse rate, and markedly increases the P. P. $\times$ P. R. product. Changing from the recumbent to the erect position decreases the pulse pressure, increases the pulse rate, and has no very definite effect on the

P. P. $\times$ P. R. product. Before attempting any discussion of these general effects special experiments were carried out in which the influence of progressively increasing increments of work and the effect of several modifications in the position of the body were determined in

Fig. 3.—Lying. Average blood pressure and pulse rate reactions of a group of five normal persons observed while they were lying down in the intervals between increasing amounts of exercise. This figure should be compared with Figure 4 in which the reactions of the same group are charted from observations made while they were standing. It will be noted that there is little difference between the P.P. $\times$ P.R. curves in this figure and in Figure 4. But the pulse pressure is higher and the pulse rate lower in the lying than in the standing position.

the hope that the more pronounced pressure and pulse rate effects thereby produced would facilitate the interpretation of the results.

These special experiments were simply a continuation and intensification of the exercise conditions under which the average effects shown

in Figure 1 were obtained, combined with various alterations in the position of the body. Immediately after the completion of the observations following 100 hops, the subjects hopped 200 times and after the same intervals for the measurement of the pressure and pulse rate 300

Fig. 4.—Standing. Average blood pressure and pulse rate reactions of a group of five normal persons observed while they were standing up in the intervals between increasing amounts of exercise. This figure should be compared with the results on the same group observed while they were lying down. (Fig. 3). The systolic pressure curve shows what has been described as a "delayed rise in systolic pressure after exercise." This is seen only when the subjects are standing up. It is absent from Figure 3 where the subjects were lying down.

and then 400 and lastly 500 hops were taken. In this manner a consecutive series of observations were made covering a period of nearly half an hour and the effect of the progressively increasing amount of work was determined. The position of the body was varied by having

the subjects lie down or stand up between the hopping, or alternately lie down and stand up at intervals of forty-five seconds between the exercises. A group of five normal individuals were observed during lying, standing, and alternate lying and standing experiments, and in another group of three normals ten experiments were performed lying on a tilting table, head down, at an angle of 23 degrees and compared with ten experiments on the same subjects in which the readings were made while they were standing.⁴ The averages obtained in the experiments in which one position was continuously maintained are charted in Figures 3, 4, 5 and 6. The alternate lying and standing experiment will be given later when the effect of change of position is discussed.

The effect of exercise on the circulation has been studied by methods which allow of an accurate measurement of changes in the volume flow of blood, i.e., of the amount of blood flowing through the aorta per unit of time. This work has been recently collected and summarized by Bainbridge.⁵ It is agreed that two changes occur in the heart's action. There is an increase in the amount of blood discharged at each systole and there is an increase in the number of systoles. The combined effect of both of these changes is an increase in the amount of blood flowing per unit of time through the aorta. Lindhard⁶ and Newburgh and Means⁷ found that when muscular work was performed there was a direct proportion between the amount of work done, and the increase in the volume flow of blood. This gives the most complete and final answer to the question as to the general circulatory effect of exercise, so that all we need do is to study our pressure and pulse rate data in order to find whether there is anything in them which might have enabled us to arrive at the correct conclusion if we had had to depend on them alone. If the volume flow of blood had been measured, and plotted on our charts it would have shown a progressive increase with each increment of work, so that if any pressure or pulse rate data can be taken as an index of changes in volume flow the curves derived from them must also show this progressive increase.

When the charts are studied in the light of these facts it is clear that the only curves which can approximate to changes in volume flow of blood are the P. P. $\times$ P. R. curves. In all the four charts, irrespective of the position of the body, there is a progressive increase as the work done increases. This is what might have been expected on a priori grounds, for the volume flow of blood per minute is the product

4. An experiment of this sort was made on this group while they sat down between the exercises. The results closely resembled those obtained in the standing experiments.

5. Bainbridge: *The Physiology of Muscular Exercise*, 1919, Oxford Press.

6. Lindhard: *Arch. f. d. ges. Physiol.* **161**:233, 1915.

7. Newburgh and Means: *J. Pharmacol. & Exper. Therap.* **7**:441, 1915.

of the systolic output per beat and the number of beats per minute. The pulse pressure is produced by the systolic output of the heart, and, other things being equal, will increase in direct proportion to any increase in the amount of blood discharged into the aorta with each

Fig. 5.—Inverted Lying. Average blood pressure and pulse rate reactions derived from ten experiments on three normal persons while they were lying inverted on a table at an angle of 23 degrees in the intervals between increasing amounts of exercise.

This figure should be compared with the results on the same group observed while they were standing (Fig. 6). It shows that during inverted lying the pulse pressure is higher and the pulse rate lower than in the standing position, though there is not much difference between the $P. P. \times P. R.$ curves in the two experiments.

systole of the heart. The $P. P. \times P. R.$ product may, therefore, be expected to follow changes in volume flow of blood more closely than any other pressure or pulse rate measurements. These charts show that what might be expected does actually occur.

If any one of these charts is studied alone, it might appear that pulse pressure or pulse rate alone might follow the changes in volume flow, for both of these curves increase with each increment of work. But if the curves in the lying experiments are compared with the same curves in the standing experiments, it will be noticed that the position of the body has a considerable influence on the degree of increase

Fig. 6.—Standing. Average blood pressure and pulse rate reactions derived from ten experiments on three normal persons while they were standing in the intervals between increasing amounts of exercise. For comparison with Figure 5.

shown by each of them. In Table 6 the first readings after exercise in the lying position are compared with the first readings after exercise in the standing position, and in Table 7 the first readings after exercise while the subjects were lying inverted are contrasted with readings on the same subjects made at the same point while they were standing.

In both comparisons the pulse pressure increase is less and the pulse rate increase is greater when standing than when lying. The reason for these differences will be discussed when the effect of change of position is considered. They are mentioned here in order to show that neither pulse pressure nor pulse rate alone can be adequate measures of the effect of exercise because they are influenced by a factor extraneous to the exercise. So also it may be said that neither systolic output

TABLE 6.—EFFECT OF EXERCISE. COMPARISON OF OBSERVATIONS MADE IMMEDIATELY AFTER EXERCISE ON SUBJECTS WHO WERE LYING DOWN, WITH OBSERVATIONS MADE AT THE SAME POINT ON THE SAME SUBJECTS WHEN THEY WERE KEPT STANDING

Exercise: Number of Hops	Position	Blood Pressure		Pulse Pressure	Pulse Rate	P. P. × P. R.
		Systolic	Diastolic			
100	Lying.....	138	60	78	83	6,494
	Standing.....	147	70	77	111	8,556
200	Lying.....	150	52	98	95	9,324
	Standing.....	136	52	84	122	10,249
300	Lying.....	154	50	104	102	10,650
	Standing.....	140	45	95	131	12,436
400	Lying.....	158	50	108	107	11,600
	Standing.....	136	44	92	142	13,100
500	Lying.....	162	51	111	118	13,160
	Standing.....	132	38	94	151	14,500

TABLE 7.—EFFECT OF EXERCISE. COMPARISON OF OBSERVATIONS MADE IMMEDIATELY AFTER EXERCISE ON SUBJECTS WHO WERE LYING INVERTED HEAD DOWN, WITH OBSERVATIONS MADE AT THE SAME POINT ON THE SAME SUBJECTS WHEN THEY WERE KEPT STANDING

Exercise: Number of Hops	Position	Blood Pressure		Pulse Pressure	Pulse Rate	P. P. × P. R.
		Systolic	Diastolic			
100	Lying inverted.....	144	72	72	81	5,830
	Standing.....	136	64	72	101	7,275
200	Lying inverted.....	153	66	87	88	7,650
	Standing.....	142	61	81	117	9,400
300	Lying inverted.....	160	65	96	101	9,660
	Standing.....	144	53	92	129	11,800
400	Lying inverted.....	167	63	104	105	10,900
	Standing.....	139	45	94	140	13,120
500	Lying inverted.....	167	58	110	116	12,770
	Standing.....	132	41	91	148	13,350

per beat nor pulse rate alone are measures of the actual changes in volume flow of blood. These changes can be measured only by the product of the systolic output per beat and the number of beats per unit of time. And in the data we have obtained in these experiments the only curves which can coincide with the progressive and uniform increase in volume flow of blood which we know occurred, are the curves derived from the product of the pulse pressure and the pulse rate.

It may be concluded, therefore, that an attempt to determine the effect of exercise on the circulation from pressure and pulse rate data alone could have been successful only if it had been based on the changes in the $P. P. \times P. R.$ product.

Fig. 7.—Alternate Lying and Standing. Average blood pressure and pulse rate reactions of a group of five normal persons observed while they alternately lay down and stood up during forty-five second periods in the intervals between increasing amounts of exercise. The darker areas are the periods when the subjects were lying down, the lighter the periods during which they were standing. It will be noted that the $P.P. \times P.R.$ curve is almost smooth after 100 hops but that on standing there is a progressively increasing break in the curve as the amount of exercise increases.

The foregoing interpretation of changes in blood pressure and pulse rate measurements after exercise in the light of knowledge of the actual circulatory effect may be used as a guide in discussing the effect of change of position, a question in which we have no decisive knowledge

to guide us and one in which we shall accordingly have to depend on pressure and pulse rate observations alone. One result of difference in body position has already been noted in the comparison between readings made in the lying and in the standing positions, but a more striking result was obtained when the subjects were required alternately to lie down and stand up at forty-five second intervals. This experiment was carried out on the same group of five individuals from whom the readings charted in Figures 3 and 4 were obtained. The results are shown in Figure 7. The darker areas in the chart are the periods when the subjects were lying down, the lighter the time during which they were standing.

The general result of these sudden alterations of position is that the smooth curves obtained in the experiments in which one position was continuously maintained are replaced by broken curves. The breaks recur regularly at each change of position. On standing, the systolic pressure drops and the diastolic pressure rises, so that there is

TABLE 8.—EFFECT OF CHANGE OF POSITION. COMPARISON OF OBSERVATIONS ON SUBJECTS IMMEDIATELY AFTER THEY STOOD UP WITH OBSERVATIONS ON THE SAME SUBJECTS AT THE SAME POINT IN THE EXPERIMENT ON CONTINUED STANDING

Exercise: Number of Hops	Position	Blood Pressure		Pulse Pressure	Pulse Rate	P. P. $\times$ P. R.
		Systolic	Diastolic			
100	Standing after lying.....	117	73	44	83	3,835
	Standing continuously.....	133	76	57	94	5,380
200	Standing after lying.....	111	71	40	97	3,870
	Standing continuously.....	143	74	69	98	6,736
300	Standing after lying.....	111	71	40	104	4,160
	Standing continuously.....	148	65	83	107	8,900
400	Standing after lying.....	110	73	37	118	4,350
	Standing continuously.....	140	68	72	114	8,180
500	Standing after lying.....	105	71	34	123	4,190
	Standing continuously.....	138	63	75	123	9,548

a great decrease in pulse pressure. But it will be noted that with every change in the pulse pressure curve there is a change in the contrary direction in the pulse rate curve. If these counterbalancing alterations in pulse pressure and pulse rate had exactly neutralized one another the P. P. $\times$ P. R. curve would have been quite smooth. But as the amount of exercise increases the breaks in the P. P. $\times$ P. R. curve become more and more marked. After 100 hops the curve is almost smooth, but after 500 hops there is a very pronounced fall during the first fifteen seconds after standing. So far as the product is concerned, these breaks in the curve immediately after standing are all that distinguish this alternate lying and standing experiment from the lying experiment or the standing experiment on the same subjects. In the latter the P. P. $\times$ P. R. curves are quite smooth, in this one they are broken. It is evident that in this experiment during the period immedi-

ately following the assumption of the erect position, and during this short period alone, some new factor becomes operative which has the effect of diminishing the $P. P. \times P. R.$ product. Elsewhere the product is smooth, and the changes in pulse pressure and pulse rate are such as have already been noted as characteristic of the standing as opposed to the lying position, and leave the $P. P. \times P. R.$ unaffected. But in this first fifteen seconds after standing something happens which is not present when the erect position is continuously maintained. This is evident from Table 8 in which the readings made during the first fifteen seconds after standing in the alternate lying and standing experiments are contrasted with readings made during the same period in the continued standing experiment.

In the figures from the continued standing experiment the $P. P. \times P. R.$ product increases from 5,380 after 100 hops to 9,548 after 500 hops. The increase after each increment of exercise is, it is true, not so uniform as in the experiments in which the lying position was maintained, but there is, nevertheless, evidence of a relation between the amount of work done and the magnitude of the product. But in the alternate lying and standing experiment the product at this period is only 3,835 after 100 hops and after 500 hops it is only 4,190; that is to say, the product is not only considerably lower throughout, but fails to show the increase on additional exercise which occurs in all the other experiments.

The correspondence we noted between changes in the $P. P. \times P. R.$ product and known changes in volume flow of blood allow us to interpret these abnormally low products as indicative of a failure of the cardiovascular system to supply the large amount of blood required on account of the amount of muscular work performed. We may, therefore, conclude that after exercise the sudden assumption of the erect position is associated with a transitory circulatory failure. The mechanism of the inadequate blood supply, however, cannot be deduced from the product alone but must be looked for in the pressure and pulse rate changes from which the low products are derived. Table 8 shows that in the alternate lying and standing experiment the pulse rate is essentially the same as in the continued standing experiment, so we may conclude that the circulatory failure is not due to an insufficient number of systoles of the heart. After 500 hops when the circulation rate is most defective, the pulse rate is exactly the same as in the continued standing experiment, and it is clear that at this and at all other points the product is low because of the low pulse pressure. After 500 hops, for instance, the pulse pressure is only 34 as against 75 on continued standing. We may, therefore, conclude that the circulatory failure is due to an insufficient systolic output at each contraction of the heart. Figure 7 shows how great is the drop in pulse pressure

towards the end of the alternate lying and standing experiment and also how rapidly the discrepancy is made good. We must, therefore, seek an explanation for an inadequacy in systolic output per beat occurring during the first fifteen seconds after the assumption of the erect position in normal individuals fatigued by exercise.

Cardiac inhibition will not explain it for the pulse rate quickens. It cannot arise from cardiac failure because the output increases again almost at once. It can then only be due to a temporary decrease in the amount of blood flowing back to the heart, and the blood which failed to return to the heart must have been held in the capillaries or in the veins. The low systolic pressure is sufficient evidence that it was not in the arteries. But why should a pooling of blood in the capillaries or in the veins occur, as Figure 7 shows it does, only when the subjects changed from the lying to the standing position? A purely mechanical answer would be that in the erect position the blood from the greater part of the body—the abdomen and limbs—has more difficulty in getting back to the heart because it has to run up hill against the force of gravity. But the differences shown in Table 8 between the alternate lying and standing results and those obtained in the continued standing experiment cannot be accounted for in this way, because at the period chosen for the comparison the force of gravity was equally operative in both experiments. The effect of gravity may, nevertheless, be an adequate explanation of the differences in the pulse pressure and pulse rate levels in Tables 6 and 7, and in Figures 3 and 5, as compared with Figures 4 and 6. In these experiments the only essential difference in the conditions was that in the lying position the force of gravity was in abeyance while in the standing position it was at work. We may conclude, therefore, that the lower pulse pressures and the higher pulse rates observed in the standing position are due to the action of gravity, and they may be interpreted as indicating smaller systolic outputs at each beat of the heart with a larger number of systoles. It is allowable to surmise that this result is brought about by a lowering of pressure in the large veins due to a diminution of the force of the stream of blood returning to the heart when it has to flow uphill against gravity. This decrease in venous pressure would be followed by a decrease in the filling of the heart during diastole, and so to smaller systolic outputs per beat. The increased pulse rate is a compensatory process, designed to counteract the necessary fall in venous pressure and to prevent any decrease in the volume flow of blood.

Another secondary manifestation of the effect of gravity is the "delayed rise in systolic pressure" which is seen only in the standing experiments. (Figs. 4 and 6). This is surely mainly an effect of

gravity and not as has sometimes been supposed⁸ a result of cardiac weakness. Nor is a temporary pooling of blood in the vessels of muscles suddenly relaxed by the cessation of exercise—a suggestion offered by Cotton, Rapport and Lewis⁹—an entirely satisfactory explanation, for there is no delay in the systolic rise after exercise in the lying experiments in which muscular relaxation must have been more complete than in the standing position. It may be accounted for as the result of a relatively smaller output per beat immediately after exercise due to the combined effect on the venous pressure of gravity and a delayed return of blood held in capillaries dilated by the exercise. As these dilated vessels recover their tone the pressure in the large veins rises, the systolic output per beat increases, and thereby the delayed rise in systolic pressure is produced.

But the force of gravity cannot be the only explanation of the real though transient circulatory failures shown by the breaks in the P. P. $\times$ P. R. curve in Figure 7, because there is no such failure in the continued standing experiments, although the fact that the breaks occur only during the first fifteen seconds after the change to the erect position would appear to show that it is one of the determining factors. But that gravity itself is not enough is indicated in Figure 7 by the fact that the product curve is almost smooth after 100 hops. It is only after 200 hops that a definite break occurs, and as the amount of work increases, the breaks become more and more pronounced so that the amount of muscular exertion which preceded the standing appears to have been a contributing factor in causing the delay in the return of blood to the heart. It should be remembered that the amount of work entailed in these exercises was very considerable, and although the subjects chosen were strong and healthy, they showed very evident signs of fatigue before the end of each experiment. Another proof that fatigue is a necessary factor is the absence of any break in the P.P. $\times$ P.R. curve shown in Figure 1 which was obtained from 300 normal subjects after 100 hops.

Why should a transient decrease in the return of blood to the heart occur when normal individuals, who are tired with muscular work, stand up? The phenomenon suggests a temporary failure under fatigue of some regulatory mechanism concerned with the adaption of the circulation to withstand the force of gravity. Krogh's¹⁰ demonstration of the power for active contraction possessed by capillaries, and Hooker's¹¹ proof that the capacity of the capillaries and venules is controlled through the central nervous system make it seem not

8. Barringer and Teschner: *Arch. Int. Med.* **16**:795 (Dec.) 1915.

9. Cotton, Rapport and Lewis: *Heart* **6**:269, 1916.

10. Krogh: *J. Physiol.* **53**:359, 1920.

11. Hooker: *Am. J. Physiol.* **54**:30, 1920.

unlikely that the blood which failed to return to the heart in our experiments may have been held in the capillaries and venules because under the influence of fatigue the central nervous system did not induce so quickly as usual the constriction of these vessels which would have prevented any pooling of blood within them under the influence of gravity.

The circulatory abnormalities shown in Figure 7 can be explained under the terms of this hypothesis. They arise under conditions of fatigue from muscular exertion as the result of a failure in the central nervous system. The failure consists in an inability to produce with sufficient promptitude the increase in the tone of the capillaries which is required to meet the effect of the force of gravity on the blood when the erect position is suddenly assumed. As a consequence the capillaries dilate before the strain, the blood is pooled in the tissues, there is a marked fall in venous pressure, in diastolic filling of the heart, and so in systolic output per beat and in pulse pressure. The systolic pressure falls because of the relatively small amount of blood forced into the aorta. The diastolic pressure rises because of the increased peripheral resistance caused largely by the banked up blood in the tissues. The delay in the return of blood to the heart is not entirely counteracted by the compensatory increase in pulse rate, and the heart for the moment fails to send an adequate supply of blood through the aorta to the body. These transitory circulatory failures are marked by the breaks in the P.P. X P.R. curve in Figure 7.

Finally, it may be well to meet an objection which may be raised on general grounds. The reaction of the cardiovascular system to exercise is so smoothly and faultlessly accomplished that it may seem improbable that there can be any failure to meet the mechanically much smaller strain involved in overcoming the force of gravity. But the perfection of the adaption to exercise is the fruit of uncounted ages of evolution. The adaption to the erect position is only recent and is not yet complete.

SECTION III

PRESSURE AND PULSE RATE REACTIONS IN PATIENTS

1. *Irritable Heart.*—The normal standards given in the first section of this paper were used in grading the reactions of men suffering from the condition known as irritable heart. A characteristic type of deviation from normality was constantly found. But the degree of deviation in any given patient varied somewhat from day to day or even from hour to hour, and in order to diminish this variability the average of five tests on different days was usually taken. A large number of records were thus obtained from these patients in the routine work of attempting to determine their capacity for military

Fig. 8.—Irritable Heart Patients. Average blood pressure and pulse rate reactions derived from 580 observations on 156 cases of irritable heart. It will be noted that the breaks in the P.P. $\times$ P.R. curve on standing before and after exercise are similar to the breaks in the product curve in normal persons after they are exhausted by exercise (Fig. 7), for in both cases these breaks are mainly due to a drop in systolic pressure. Compare with Figure 1.

service. The averages of 580 tests on 156 patients who had been classified as belonging to what has been called the constitutional division of the irritable heart group are given in Table 9 and charted in Figure 8.

In these patients the basal and daytime levels of pressure and pulse rate have already been shown to coincide fairly closely with the normal.¹² Yet in spite of this essential normality in initial level a comparison of Figure 8 with the curve of the average normal reactions in Figure 1 demonstrates a marked departure from the normal. It is therefore a latent abnormality which becomes apparent only under strain.

Further, it is an abnormality which is revealed only by a specific type of circulatory strain, i.e., the strain involved in the adaption required when the erect position is assumed. Figure 8 shows that the average reaction to exercise is entirely normal. The variability is somewhat greater and in some cases there was what we called

TABLE 9.—AVERAGE BLOOD PRESSURE AND PULSE RATE OBTAINED FROM 580 OBSERVATIONS OF 156 PATIENTS WITH CONSTITUTIONAL NEUROCIRCULATORY DISABILITY

	Time in Seconds													
	0	15	30	45	60	75	90	140	155	170	185	200	215	230
	Lying			Standing			Hop- ping	Lying			Standing			
Systolic.....	131			129	113	135	131		158		152	121	142	140
Diastolic.....	76			79	84	90	91		65		73	84	86	88
Pulse pressure	55			50	28	45	40		93		79	36	56	52
Pulse rate.....	85			82	107	96	95		116		99	123	110	108
P. P. × P. R.	4680			4120	2990	4320	3900		10788		7750	4440	6160	5620

"overaction", i.e., an excessive increase in the P.P. X P.R. product after exercise, but on the average these patients have an adequate and normal response to the strain of exercise. On the other hand, the average curves show a pronounced deviation from the normal immediately after standing both before and after exercise. In some individual cases the difference from the normal was slight; in others, it was very marked and constant. And, in general, there seemed to be a fairly close relation between the severity of the symptoms and the degree of difference from the normal.

The nature of the abnormality is clearly shown in Figure 8. After standing there is a break in the P. P. × P. R. curve where it is almost smooth in normal individuals. These breaks are due to a sudden fall in pulse pressure and this again is mainly due to a drop in systolic pressure. If Figure 7 is consulted it will be seen that the abnormality

12. Addis: Arch. Int. M. 29:539 (April) 1922.

in the patients is in every respect similar to the failure in circulation which occurs in normal individuals when they are required to stand up after being tired with a considerable amount of muscular work. The only difference between the patients and the normals consists in the fact that this failure appears in the patients before they have done any work at all, while in normal individuals some degree of exhaustion by muscular exertion is required before any failure can be demonstrated.

The cause of this particular form of circulatory failure has already been discussed in Section II. The conclusion was reached that the immediate cause is a failure of the capillaries to affect the adjustment of capacity required to counterbalance the force of gravity when the vertical position is assumed, and that the primary cause is a failure of the complex coordination of impulses which is required of the central nervous system. This explanation, which is in accordance with the facts observed in normal persons, is also in harmony with the signs and symptoms observed in the patients. As their hands hang down

TABLE 10.—BLOOD PRESSURE AND PULSE RATE REACTIONS IN A PATIENT WITH HYPERTENSION AND SYMPTOMS SUGGESTIVE OF CARDIAC WEAKNESS

	Lying			Standing			Hopping 25 Times	Lying			Standing		
Systolic.....	220	210	215	205	210	205		200	210	210	220	220	220
Diastolic.....	150	160	150	150	155	145		135	145	150	150	155	155
Pulse pressure	70	50	65	55	55	60		65	65	60	70	65	65
Pulse rate.....	104	100	104	104	100	100		108	104	104	108	100	92
P. P. × P. R.	7280	5000	6760	5720	5500	6000		7020	6760	6240	7560	6500	5980

they are engorged, cyanosed and cold. This is the visible evidence of the delay and accumulation of blood in their capillaries. As they rise from the recumbent position, they become dizzy and faint. This is evidence of a failure in the return of blood to the heart and consequent temporary cerebral anemia. Their palpitations, precordial pain, and dyspnea, when conditions call for a sudden increase in the volume flow of blood, are the result of an inadequate and irregular return flow of blood to the heart from their dilated capillary areas. And there was ample clinical evidence in these patients of the nervous instability which I believe is at the root of the condition. A neuro-pathic family history is almost constant in the constitutional type, and a great variety of symptoms could be traced ultimately to defective nervous control. Later as men began to return from France many were seen who presented the same symptoms found in the constitutional type, though in less marked degree. They showed the same pressure and pulse rate reactions, though the fall in pulse pressure on standing was less pronounced. Psychic or physical exhaustion seemed to be the cause in this group of acquired cases and in them

Fig. 9.—Cardiac Failure. The blood pressure and pulse rate reaction of a patient who showed evidence of an early stage of cardiac decompensation.

TABLE 11.—FREQUENCY DISTRIBUTIONS OF PRESSURE AND PULSE RATE OF 300
NORMAL INDIVIDUALS

Time in Seconds															
	0	15	30	45	60	75	90	140	155	170	185	200	215	230	
	Lying			Standing			Hop- ping	Lying			Standing				
Systolic Pres- sure															
81- 90	7	6	6	2	1	2		1	2	2					
91-100	18	17	19	22	6	11		2	2	2		4	2	4	
101-110	32	33	30	45	23	24		2	4	7	18	8	11		
111-120	65	76	78	82	54	61		8	13	25	37	27	30		
121-130	83	73	75	66	73	82		26	34	40	65	47	69		
131-140	47	53	50	45	72	62		50	45	54	71	72	62		
141-150	31	25	28	19	31	29		46	55	56	48	56	59		
151-160	12	11	4	13	18	19		56	56	47	28	32	30		
161-170	2	3	3	3	10	6		49	53	42	16	34	19		
171-180	3	3	1	2	8	3		36	20	13	8	11	7		
181-190				1	2	0		13	8	5	5	7	6		
191-200					2	1		8	4	4		3	3		
201-210								1	3	3		0			
211-220								2	1	3		1			
Diastolic Pres- sure															
31- 40								1							
41- 50	5	3				4		27	8	2	1				
51- 60	19	15	12	7	4	13		82	46	24	20	7	4		
61- 70	84	65	61	35	21	110		105	106	86	41	31	31		
71- 80	103	110	110	84	58	57		48	85	105	107	88	84		
81- 90	69	77	80	99	113	113		32	39	61	79	101	98		
91-100	16	26	32	54	70	70		5	13	18	39	48	53		
101-110	4	3	5	19	24	29			3	4	12	20	24		
111-120		1		2	9	13					1	4	6		
121-130					1	1						1			
Pulse Pressure															
11- 20	2	5	5	20	2	6		1	1	1			2		
21- 30	28	29	41	74	45	73		1	1	3	20	7	14		
31- 40	70	86	93	105	101	110		0	5	11	46	34	49		
41- 50	99	106	101	67	96	75		6	9	30	73	71	85		
51- 60	61	48	43	25	37	26		14	35	58	81	83	84		
61- 70	20	15	13	9	11	9		41	59	77	39	60	39		
71- 80	12	8	2		5	1		60	71	60	23	26	16		
81- 90	8	3	2		1			59	59	32	12	13	9		
91-100					2			66	42	22	5	3	2		
101-110								31	13	4	1	3			
111-120								16	4	1					
121-130								4	0						
131-140								0	1						
141-150								0							
151-160								1							
Pulse Rate															
45- 52		5	8	1	3	4									
53- 60	14	26	33	2	7	10				5		5	6		
61- 68	38	67	77	7	31	36		3	13	17	1	9	14		
69- 76	80	85	84	35	70	79		10	43	64	18	30	36		
77- 84	78	53	43	51	75	69		39	61	74	23	39	46		
85- 92	43	40	36	78	53	42		47	71	71	56	66	60		
93-100	24	11	12	54	18	20		75	62	38	60	54	52		
101-108	8	8	7	37	23	21		39	22	18	59	48	41		
109-116	2	2		17	10	15		48	21	8	43	21	21		
117-124	2			12	8	4		16	4	3	15	15	14		
125-132	1			5	2			18	2	2	20	10	9		
133-140				1				1	0		4	3	1		
141-148								3	1						
149-156								1							
P. P. × P. R.															
1000- 1999	17	25	37	17	15	20		1	1	1		1	4		
2000- 2999	53	75	85	86	78	108		2	3	8	16	15	18		
3000- 3999	93	99	107	84	95	97		2	8	28	32	35	63		
4000- 4999	58	58	37	69	61	40		6	23	50	72	70	75		
5000- 5999	47	25	25	34	29	28		19	51	72	73	67	63		
6000- 6999	13	13	7	6	13	6		34	71	53	43	52	38		
7000- 7999	12	3	1	1	6	1		52	44	40	32	28	23		
8000- 8999	6	1	0	2	2			42	32	28	15	17	8		
9000- 9999	0	1	1	0	1			38	36	13	10	5	6		
10000-10999	0			0				33	22	7	7	8	1		
11000-11999	1			1				35	7			2	1		
12000-12999								15	2						
13000-13999								11							
14000-14999								9							
15000-15999								0							
16000-16999								1							

the nervous element was strongly marked. The condition is also common as a temporary sequel to all varieties of acute infection, and the same type of abnormality in pressure and pulse rate reactions is found. I believe that the same cause is at work in these three groups of cases. But we have no satisfactory term to describe the condition. Irritable heart is unsuitable because the heart itself is not at fault. Effort syndrome is not entirely satisfactory because muscular effort is not essential for the production of the symptoms. Neurocirculatory asthenia does not quite fit because weakness is no necessary part of the picture. The term neurocapillary stasis is suggested because it calls attention to the mechanism which causes the syndrome.

Cardiac Disability.—The records in cases of compensated valvular defects show no abnormality apart from the well known effects of aortic insufficiency on the diastolic pressure. In definitely decompensated cases the test is inapplicable. But in a few patients who showed evidence of an early stage of decompensation interesting records have been obtained. In Table 10 and Fig. 9 are given the pressure and pulse rate of a man, age 44, with chronic diffuse nephritis and a moderate degree of impairment of renal function who was beginning to complain of dyspnea on exertion and occasionally a little edema around the ankles. The heart was enlarged but no evidence of dilation was found.

The striking point about this record is its uniformity, which results from the absence of any appreciable change in pressure and pulse rate after exercise. This was not due to an insufficient amount of exercise. The number of hops had to be restricted because of the patient's dyspnea. It suggests, rather, the forced uniformity which would result if the heart, before the exercise, were already doing all it could. It resembles the phenomenon seen in renal decompensation where a constant rate of excretion is maintained under conditions which induce great variation in normal persons.

SUMMARY

SECTION I

1. Blood pressure and pulse rate curves showing the effect of exercise and of change of position were obtained from 300 normal persons.

2. It was found that the degree of change in pressure and pulse rate produced by exercise and change of position is in part dependent on the level of pressure and pulse rate existing before the exercise or change of position. This fact must be taken into account in computing standards of normality. A separate standard for each initial level must be established.

3. Averages and standard deviations for pressure and pulse rate reactions to exercise and change of position are given for a series of initial levels.

SECTION II

1. Blood pressure and pulse rate observations on normal persons before and after progressively increasing amounts of muscular work were charted as curves and examined with a view to finding whether it was possible to derive from them any indication of the regular increase in the volume of blood flow which is known to occur after each increase in muscular exertion. It was found that the systolic and diastolic pressure curve showed no constant relation to the work, and that the degree of increase in the curves of pulse pressure and pulse rate varied not only with the work but also with the position of the body. The only curves which under all conditions corresponded to changes in volume flow of blood were the curves derived from the P.P. X P.R. product.

2. When normal persons were tired by muscular work, and were required to change from the recumbent to the erect position, there was an immediate, though transient, decrease in the P.P. X P.R. product curve. The correspondence between changes in volume flow of blood and changes in the magnitude of the product, the nature of the pressure changes which gave rise to the decrease in the product, and recent evidence as to the contractility and innervation of the capillaries, are taken as a basis for the hypothesis that the product changes indicate a failure of the circulation to meet the effect of the force of gravity. The circulatory failure is secondary to a failure of the central nervous system to produce with sufficient promptitude the increased tone of the capillaries and venules which is necessary in order to prevent an accumulation of blood in the tissues when the erect position is assumed.

SECTION III

1. The type of abnormality in pressure and pulse rate reactions and the signs and symptoms found in patients suffering from the irritable heart syndrome support the hypothesis that the cause of the condition is a failure of nervous control over the capillaries.

2. It is suggested that when weakness of the heart is the primary cause of circulatory failure the pressure and pulse rate curves show an unusual degree of uniformity in level before and after exercise. This uniformity results from an absence of the increase in pulse pressure and pulse rate after exercise which is one of the manifestations of that increase in volume flow of blood with which the normal heart meets the requirements of muscular exertion.