STUDIES OF OXYGEN IN THE VENOUS BLOOD.

II. Studies of the Oxygen Unsaturation in the Venous Blood of a Group of Patients with Circulatory Disturbances.

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INTRODUCTION.

In a previous paper (1) a report was made of a series of determinations of the oxygen content of the venous blood from the vena mediana cubiti of twelve normal resting individuals. The difference between the oxygen in the venous blood and the total oxygen capacity of the hemoglobin (determined by Van Slyke's method (2) or Palmer's method (3)) was called the oxygen unsaturation of the venous blood. In this way the differences in hemoglobin concentration are eliminated.[1] It was found that the extent of oxygen unsaturation of the venous blood in normal individuals fell between 2.5 and 9 cc. per 100 cc. of blood. However, values above 8 volume per cent were only met with under special conditions; namely, when the blood was drawn in the morning immediately after the subject had been awakened from sleep. The blood was drawn from an arm vein without any stasis whatever, after the subject had rested for a half hour on a couch or bed. A sharp, not too pointed, needle was connected with a rubber tube to a glass pipette, 25 to 30 cm. long and $\frac{1}{2}$ cm. wide, which had a film of oxalate on the inside.[2] The blood (about 10 cc.) was sucked

[1] A determination of the oxygen in the venous blood without a simultaneous determination of the total oxygen capacity is just as incomplete as a determination of the urea in the blood or the nitrogen in the urine, without considering the intake.

[2] This was obtained by wetting the tube and rubber with a concentrated solution of oxalate and drying by an air current.

TABLE I.

Patients with Circulatory Disturbances Compensated at Rest.

Experimental.

Case No.	Determination No.	Age.	Bleeding.			Oxygen content of venous blood.					Hemoglobin (Palmer's method).	Calculated oxygen capacity (a).	Oxygen capacity (Van Slyke's method).	Oxygen unsaturation (a − v).	Pulse.	Respirations.
						Sample 1.		Sample 2.		Average (v).						
			Date.	Hour.	Condition.*	Hour.	Result.	Hour.	Result.							
		yrs.	*1917*				*vol. per cent*		*vol. per cent*	*vol. per cent*	*per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*		
1	1	41	Feb. 3	11.00 a.m.	1 hr.	11.20	17.72	11.30	18.70	18.21	123	22.70	21.96 22.34	4.49	98	18
2	2	56	May 22	10.30 a.m.	In bed.	11.35	12.98	11.55	13.50	13.24	100	18.50		5.26	80	22
3	3	47	Feb. 12	4.00 p.m.	5 min.	4.15	15.30	4.25	15.14	15.22	100	18.50	18.74 18.00	3.28	84	24
	4		May 6	2.30 "	1 hr.	2.50	14.24	3.15	14.26	14.25			16.90 17.10	2.75	100	22
4	5	29	May 8	2.10 p.m.	In bed.	2.40	5.44	3.00	5.42	5.43	44.5	8.23		2.80	84	22
5	6	49	May 22	9.20 a.m.	In bed.	10.50	9.19	11.08	9.35	9.27	92	17.02		7.75	80	22
	7		June 21	11.00 "	" "	11.45	12.24	12.00	12.24	12.24	89.5	16.54		4.30	80	20
	8		" 27	9.30 "	¾ hr.	10.30	11.88		11.46	11.67	88.5	16.37		4.70		
	9		" 30	10.20 "	¾ "	10.35	10.89	10.55	10.93	10.91	88.5	16.37		5.46	80	20
6	10	22	June 26	9.50 a.m.	½ hr.	10.50	11.78	11.20	12.04	11.91	103	19.06		7.15	82	20
7	11	25	Apr. 18	12 n.	In bed.	12.40	13.52	12.55	13.60	13.56	111	20.53		6.97	76	24
8	12	37	Feb. 12	12 n.	In bed.	12.15	15.30			15.30	111	20.53		5.23	75	18
9	13	31	Feb. 17	3.00 p.m.	In bed.	3.50	17.15	4.15	16.90	17.03	114	21.10		4.07	67	26
	14		Mar. 8	10.00 a.m.	" "	11.10	14.62	11.40	14.42	14.52	106	19.60		5.08	90	22
10	15	57	June 27	12.30 p.m.	½ hr.	2.10	12.89	2.30	12.47	12.68	105	19.44		6.76	96	28
11	16	25	May 31	3.00 p.m.	¾ hr.	4.00	11.65			11.65	89	16.46		4.81	56	28
12	17	18	Feb. 28	10.30 a.m.	In bed.	10.55	6.72	11.10	6.75	6.74	71.4	13.20		6.46	80	22

Case No.	Determination No.	Oxygen unsaturation (a − v).	Diagnosis and clinical notes.
		vol. per cent	
1	1	4.49	Arteriosclerosis; hypertensio (blood pressure 160–96); hypertrophy of heart; shortness of breath on exertion.
2	2	5.26	Arteriosclerosis; hypertension; hypertrophy of heart (blood pressure 296–130); some moist râles in both lungs.
3	3	3.28	Chronic interstitial nephritis; hypertension (blood pressure 180–130); hypertrophy of heart; shortness of breath on exertion.
	4	2.75	
4	5	2.80	Chronic interstitial nephritis; chronic uremia; hypertension (blood pressure 195–133); hypertrophy of heart; edema; ascites; stasis in lungs (edema); urea retention in blood.
5	6	7.75	Chronic nephritis; hypertension; cardiac hypertrophy (he was never incompensated at rest. He had shortness of breath on slight exercise). May 22. A few râles at base of lungs; no dullness. Blood pressure 218–126.
	7	4.30	June 21. No râles. Blood pressure 214–108.
	8	4.70	" 27. Pains behind sternum. No râles. Blood pressure 220–112.
	9	5.46	" 30. No râles.
6	10	7.15	Aortic insufficiency; hypertrophy of heart (blood pressure 300–90).
7	11	6.97	Mitral stenosis; auricular fibrillation.
8	12	5.23	Mitral stenosis and insufficiency; auricular fibrillation.
9	13	4.07	Mitral insufficiency; auricular fibrillation.
	14	5.08	
10	15	6.76	Auricular fibrillation; chronic myocarditis.
11	16	4.81	Chronic myocarditis. Pulse regular.
12	17	6.46	Pericarditis; some râles at bases of lungs.

* Condition indicates the length of time the patient has been resting in bed before the drawing of the blood.

TABLE II.

Patients with Incompensated Heart Failure.

Experimental.

Case No.	Determination No.	Age.	Bleeding.			Oxygen content of venous blood.					Hemoglobin (Palmer's method).	Calculated oxygen capacity (a).	Oxygen capacity (Van Slyke's method).	Oxygen unsaturation (a − v).	Pulse.	Respirations.
			Date.	Hour.	Condition.	Sample 1.		Sample 2.		Average (v).						
						Hour.	Result.	Hour.	Result.							
		yrs.	*1917*				*vol. per cent*		*vol. per cent*	*vol. per cent*	*per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*		
13	18	30	Feb. 10	11.00 a.m.	1½ hr.	11.20	6.56	11.35	6.50	6.53	93	17.18	16.65 16.69	10.14		
	19		" 19	10.05 "	In bed.	10.30	6.67	1.45	6.58	6.63	100	18.50		11.87	62	22
14	20	57	Feb. 9	3.10 p.m.	In bed.	3.30	8.60	3.50	8.84	8.72			22.52 22.46	13.76	96	36
	21		" 19	10.00 a.m.	" "	10.10	5.93	10.50	5.79	5.86	112	20.71		14.85	86	32
	22		Apr. 27	2.40 p.m.	" "	2.45	12.39	3.10	12.55	12.47	120	22.20		9.73	86	32
	23		June 26	10.00 a.m.	" "	10.10	7.16	10.40	7.62	7.39	106	19.61		12.22	77	24
15	24	64	Feb. 21	11.10 a.m.	In bed.	11.20	12.12	11.30	12.05	12.07	119	22.00		9.91	36	20
	25		Mar. 24	12 n.	" "	2.30	7.68	2.50	7.66	7.67	117	21.65		13.98	28	20
	26		Apr. 21	11.00 a.m.	" "	2.10	10.48	2.30	10.46	10.47	110	20.36		9.89	32	24

Case No	Determination No.	Oxygen unsaturation (a − v).	Diagnosis and clinical notes.
		vol. per cent	
13	18	10.14	Mitral stenosis and insufficiency; auricular fibrillation; adherent pericardium.
	19	11.87	Râles at base of lungs; cyanosis of face; distention of veins in neck; enlargement of liver; ascites; low diuresis.
14	20	13.76	Mitral insufficiency; arteriosclerosis; hypertrophy of heart.
			Feb. 9. Moist râles in both lungs; severe cyanosis; enlargement of liver; severe edema of legs.
	21	14.85	" 19. Same condition.
	22	9.73	Apr. 27. Has improved. Very slight cyanosis; no edema; marked enlargement of liver. Still some râles in lungs.
	23	12.22	June 26. Slight edema of legs; slight cyanosis. Still râles at base of lungs.
15	24	9.91	Heart block; syphilis of heart.
			Feb. 21. Slight cyanosis of lips; slight jaundice of scleræ and skin; râles in lungs; no edema; great enlargement of liver.
	25	13.98	Mar. 24. Same condition.
	26	9.89	Apr. 21. " "

TABLE III.

A Patient with Incompensated Heart Failure. Oxygen Determined by Barcroft's Method.

Experimental.

Case No.	Determination No.	Age.	Bleeding.		Oxygen content of venous blood.			Total oxygen capacity (Barcroft's method(a)).	Oxygen unsaturation (a − v).	Pulse.	Respirations.
			Date.	Condition.	Sample 1.	Sample 2.	Average (v).				
		yrs.	*1916–17*		*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*		
	27	17	Oct. 25	In bed.	2.40		2.40	15.30	12.90		
	28		Nov. 20	" "	2.48		2.48	17.20	14.72	120	40
16	29		" 21	" "	3.36	1.36	2.36	17.56	15.20	122	40
	30		" 22	" "	2.40		2.40	17.56	15.16	124	44
	31		Jan. 12	" "	6.86		6.86	17.20	10.34	120	30

Clinical.

Case No.	Determination No.	Oxygen unsaturation (a − v).	Diagnosis and clinical notes.
		vol. per cent	
	27	12.90	Mitral and aortic insufficiency. Oct. 25, 1916. Moderate cyanosis; few râles at base of lungs.
	28	14.72	Nov. 20. Lungs clear.
16	29	15.20	" 21. Cyanosis.
	30	15.16	" 22. Enlargement of liver.
	31	10.34	Jan. 12, 1917. Less cyanosis; no râles; slight enlargement of liver.

up in the pipette, from which it was discharged into a cylinder, 2 cm. in diameter, below a layer (2 cm.) of mineral oil to prevent oxidation. The last (upper) 1 or 2 cc. of blood, a part of which had been oxidized, were put in a separate dish and used for a Palmer determination of the hemoglobin. Samples of blood for a Van Slyke determination were taken from the cylinder after careful stirring.[3]

Observations on Patients.

This paper is a report of a preliminary investigation on the oxygen unsaturation in patients with circulatory disturbances. Thirty-

[3] For further technical details see Paper I of this series (1).

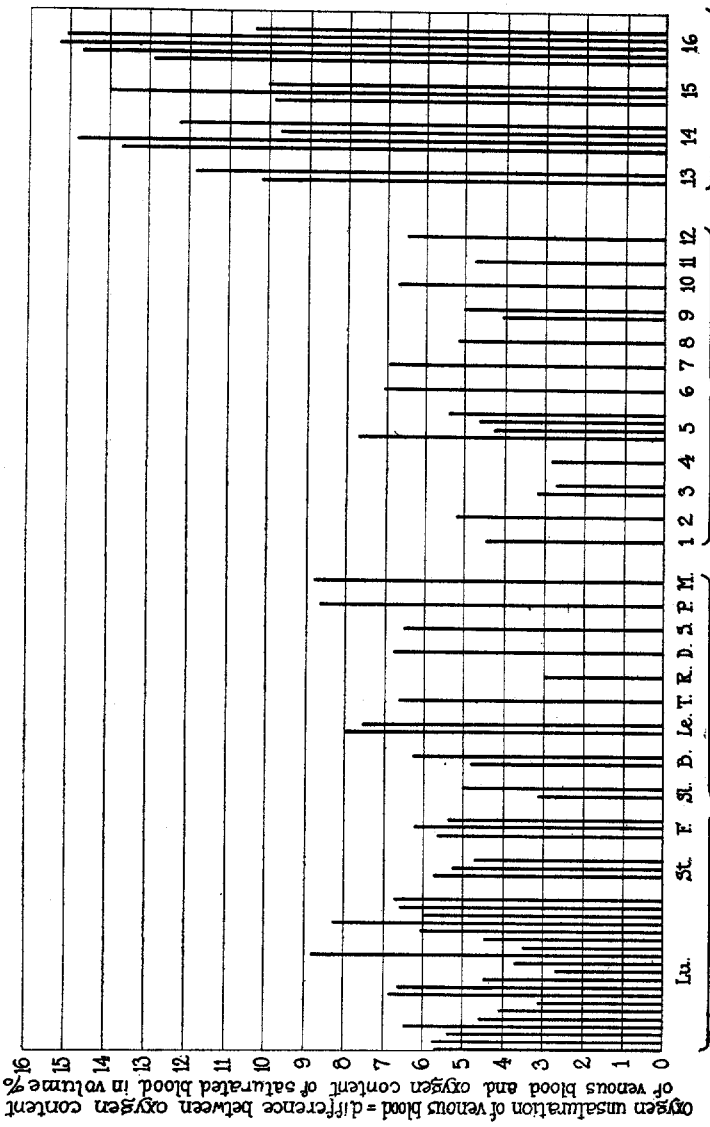
one determinations were made on sixteen patients with different kinds of circulatory disturbances. The procedure in preparing the patients and the technique in drawing the blood have been exactly as described in the first paper. Van Slyke's method (2) has been used for the determination of the oxygen in all but one case (No. 16 in Table III) where Barcroft's method has been used.

The patients have been divided into two groups according to the condition of the circulation. Results and clinical notes from patients with clinically compensated heart lesions have been tabulated in Table I. The data from the cases with incompensated circulatory conditions are collected in Tables II and III. As in Paper I, we have tabulated both the oxygen content of the venous blood samples and the extent of the oxygen unsaturation.

One case (No. 4, Table I) is particularly worth mentioning, because it shows the importance of using the oxygen unsaturation and not the absolute value of the venous oxygen alone in comparing the results from different individuals. This patient was brought to the hospital in a very sick condition. He was suffering from dyspnea and palpitation and had ascites and severe edema of the legs. The oxygen in his venous blood was 5.43 volume per cent, which was much lower than any figure obtained on normal individuals, and even lower than most of the figures obtained from patients with incompensated heart disturbances. The total oxygen-combining power of his blood was very low, however, 8.23 volume per cent, instead of the usual average 18.50 volume per cent. The oxygen unsaturation of his venous blood is therefore not only within normal limits but even rather low. The clinical diagnosis was chronic nephritis, chronic uremia, hypertension, hypertrophy of the heart, anemia, and urea retention. The edema was considered to be entirely of nephritic origin. Therefore, in respect to both the clinical picture and the findings in the blood, he is to be grouped in Table I, with patients free from incompensated circulatory disturbances. On the other hand, we may find a high figure for the venous oxygen combined with a high extent of unsaturation (No. 13, Table II) on account of a high content of hemoglobin.

Text-fig. 1 is a diagrammatic representation of the figures for the oxygen unsaturation in normal individuals[4] and in the patients re-

[4] Reported in Paper I (1).



TEXT-FIG. 1. Oxygen unsaturation of venous blood in a group of normal subjects[4] and of patients reported in the present paper.

ported in the present paper. It will be seen that the upper limit for the unsaturation in normal individuals (9 volume per cent) is not exceeded by any figure obtained from patients belonging to the clinically compensated group. The figures are all distributed in the same haphazard manner over the area between 2.5 and 9 volume per cent.

The figures from the incompensated group show quite another picture. They are all above the upper normal limit, lying between 9.7 and 15.2 volume per cent.

No attempt has been made to subdivide the cases according to the anatomical or physiological form of the heart lesion. There may be a difference, but the cases and the number of determinations in each case are much too few to allow any such attempt. The only statement which can be made is that the oxygen unsaturation of the venous blood from patients with clinically compensated heart conditions has been found within the normal limits, whereas the oxygen unsaturation in some patients with incompensated heart lesions has been above the upper normal limit. It is probably not even justifiable to make this a general statement. It seems probable that patients with non-stationary conditions might fall outside this rule. We might suppose that a patient with an incompensated, but improving heart lesion could show normal figures. On the other hand, a patient with a heart lesion where incompensation is developing might possibly show increased oxygen unsaturation before the ordinary clinical symptoms had developed. If that should be true, the determination of the oxygen unsaturation would be of great clinical significance. Series of determinations on a single patient might throw light on this question.

DISCUSSION.

There is extensive clinical and experimental evidence for assuming that the condition of the heart has a certain influence on the extent of oxygen unsaturation of the venous blood. A previous investigation on the blood flow (minute volume) in patients with incompensated heart lesions (4) has shown a considerable decrease in the minute volume of the heart compared with that found in normal individuals

(5–14), and in most cases of compensated heart lesions[5] (4, 6, 11, 15). The figures obtained by Stewart (16) by determining the blood flow in the hands and feet point in the same direction. A retarded circulation, the rate of oxygen consumption not being decreased, may logically be assumed to result in a necessarily increased oxygen unsaturation of the average venous blood. The question is whether one is justified in assuming a retarded circulation from an increased oxygen unsaturation in the blood drawn from the vena mediana cubiti or from another superficial vein.

We know that other factors than the output from the heart may influence the extent of unsaturation in the blood from an arm vein. These factors are[6] (1) the possible unsaturation of the arterial blood; (2) variations in the metabolism of the tissues drained by the vein tapped as compared with the rest of the body; (3) variations in the rate of blood flow through the tissues drained compared with the rest of the body.

We are unfortunately, for the time being, only to a limited extent able to measure, control, or eliminate the influence of these factors.

(1) The dissociation curve for oxyhemoglobin shows that the blood must become nearly saturated (usually about 96 to 98 per cent) during the passage through the lungs, provided that all the blood flowing through comes into equilibrium with the alveolar air. This has been proved experimentally on animals by several investigators, and in

[5] Plesch (6) and Means and Newburgh (11) found normal figures for the blood flow in all their patients with compensated heart lesions, whereas Lundsgaard (4) found diminished minute volume not only in incompensated cases but in some patients with compensated heart conditions (mitral stenosis and auricular fibrillation), which agrees with Stewart's (16) observations for the local blood flow.

[6] One condition which might theoretically affect the oxygen saturation of the hemoglobin in arterial blood is incompensated acidosis, defined by Hasselbalch and Gammeltoft (17) and Van Slyke and Cullen (18), in which increased free carbonic acid causes an increase in the actual hydrogen ion concentration of the blood. As shown by Barcroft (19) this increase reduces the percentage oxygen saturation of hemoglobin under a given oxygen tension. Since, however, the differences thus caused in oxygen saturation become considerable only under reduced oxygen tension, it appears improbable that this factor, even in the infrequently occurring incompensated acidosis, exerts a significant effect on the degree of saturation of the arterial hemolgobin.

man it has been investigated by Hürter (20). By drawing blood from a radial artery and using Barcroft's method for determination of the oxygen, he found in four normal individuals a saturation of 94, 92, 99, and 100 per cent. In four patients with compensated heart lesions the saturation was 90, 100, 90, and 95 per cent. In one patient with incompensated aortic insufficiency and stasis bronchitis in the lungs the saturation was 81 per cent. In another patient with incompensation (Pick's disease) it was 92 per cent. A patient with patent ductus Botalli showed 88 per cent saturation. One with diffuse bronchitis showed 94 per cent, and another with phthisis, 88 per cent. Two patients with lobar pneumonia showed 81 and 79 per cent saturation. Barcroft[7] found a saturation of 94 per cent in a normal individual.

In several of my patients with compensated heart lesions moist râles have been heard at the base of the lungs. This circumstance does not seem to have influenced substantially the amount of oxygen in the venous blood. These râles are probably to a great extent just a proof that the air really does go down into the alveoli of the lowest part of the lungs. The whole problem is not sufficiently investigated to allow a decision concerning the influence of the involvement of the lungs in the individual case. We may confine ourselves to making a careful examination of the lungs in every case, hoping that special investigation of the unsaturation in patients with lung diseases or experimental studies will give us sufficient information.

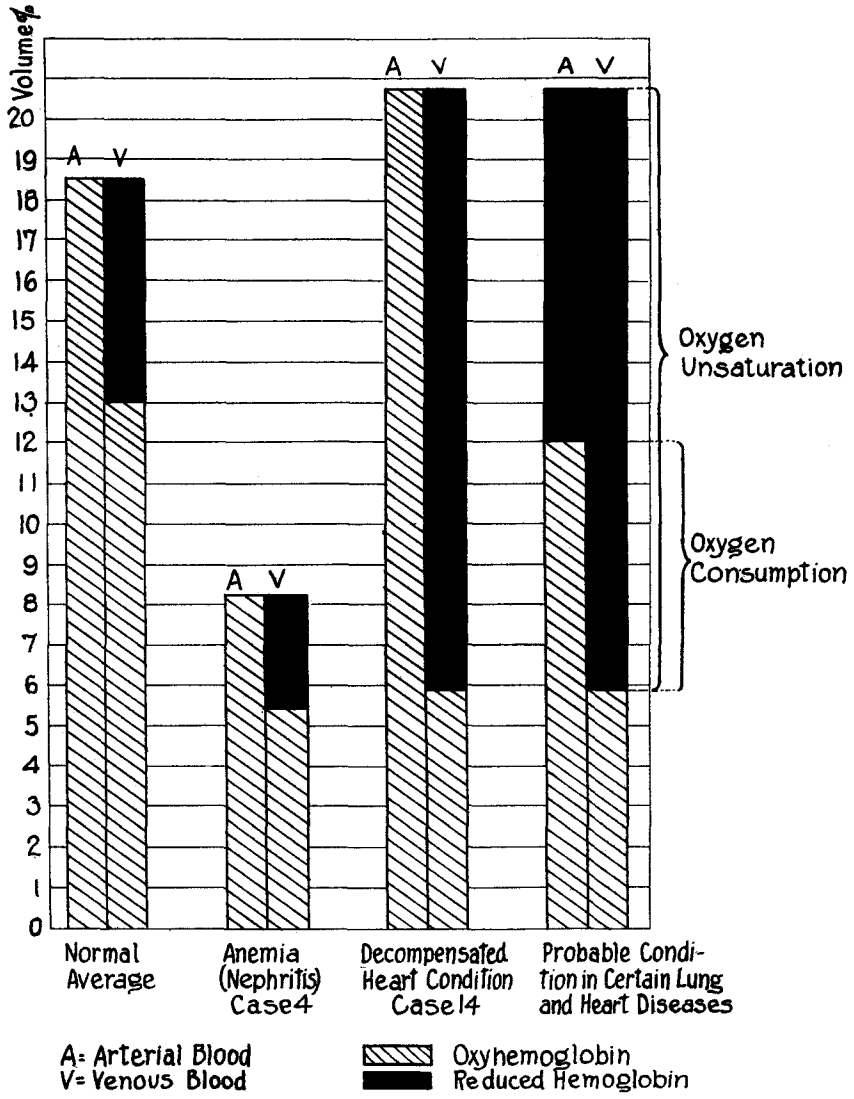
(2) The variations in the metabolism are presumably minimized by performing the experiments under definite conditions, such as muscular rest and digestive inactivity. The basal metabolism is a rather constant figure for the normal individual at rest and is, as shown by Lusk and his associates, chiefly dependent on the surface of the body. It has been shown by Peabody, Meyer, and Du Bois (21) that the metabolism in patients with compensated heart and cardiovascular lesions is within the normal limits. In patients with incompensated heart lesions, particularly in patients suffering from dyspnea, however, the metabolism was found materially increased, in some cases about 50 per cent. An increase in the metabolism necessarily means

[7] Barcroft (19), p. 177.

an increase in the consumption of oxygen. This, however, is not necessarily followed by an increase in the deoxidation of the oxyhemoglobin. That depends upon the output of the heart, which may or may not be changed. Lindhard (9), for instance, has shown that the increased consumption of oxygen during exercise only partially shows itself in the decreased oxygen in the venous blood; the increased blood flow compensates for a great part of the increased consumption. Lundsgaard (15) has shown that an increased oxygen consumption on account of exercise in two clinically compensated patients with heart block only to a very small degree could be compensated for by increasing blood flow. The reason for this was that it was impossible for the heart to increase the pulse rate and probably very difficult to increase the volume output per beat, which even in rest was about 150 cc. It is, therefore, probable that an increase in metabolism in a patient with a weak heart will increase the deoxidation of the venous blood, particularly in the venous blood coming from the heart muscle and the respiratory muscles. An increase in temperature may have a similar effect.

(3) About the only figures available on this point are those of Stewart (16). He has calculated from measurements of the heat given off by the hands and feet the rate of local blood flow and found that the flow in the right hand or foot is approximately equal to that in the left. Significant differences were encountered only when there was definite local cause, as aneurysms, diabetic gangrene, local edema, etc. It will probably be possible to throw light upon this question by simultaneously drawing blood from different veins and under different conditions, as tried in a number of cases by Means and Newburgh (11). Investigations on the local blood flow by Hewlett (22), using a modified Brodie method, have shown that there is a considerable temperature interval where the blood flow remains approximately constant when the surrounding temperature is changed.

The influence of the hemoglobin percentage, of the output from the heart, and of a possible unsaturation of the blood in the lungs is shown in diagram form in Text-fig. 2. The first double column represents the conditions in a person with normal hemoglobin, normal blood flow, and total saturation of the arterial blood. The next



TEXT-FIG. 2. Diagrams showing how the hemoglobin percentage, the output from the heart, and the saturation of the arterial blood influence the oxygen in the venous blood.

shows the findings in a patient with very low total oxygen capacity[8] but with normal circulation and full saturation in the lungs. A comparison of these two sets of figures shows the impossibility of using the zero as the base-line. The differences in hemoglobin must be accounted for. That is what we have done by using the term oxygen unsaturation, which is represented by the black area. The next two pairs of columns show how the extent of oxygen unsaturation can be affected in the same way by different causes. The third column represents the condition in a heart case (No. 14, Table II) when the total oxygen capacity was a little above normal; the saturation in the lungs was supposed to be normal. The cause of the increased oxygen unsaturation in this case was a slow circulation. The fourth column represents a hypothetical case with the same degree of oxygen unsaturation of the venous blood. However, the cause is ascribed to a considerable extent of unsaturation of the arterial blood. These two examples show the possible inaccuracy of using the word oxygen consumption in the same sense as oxygen unsaturation.

Oxygen Unsaturation Compared with Oxygen Consumption (Blood Flow).

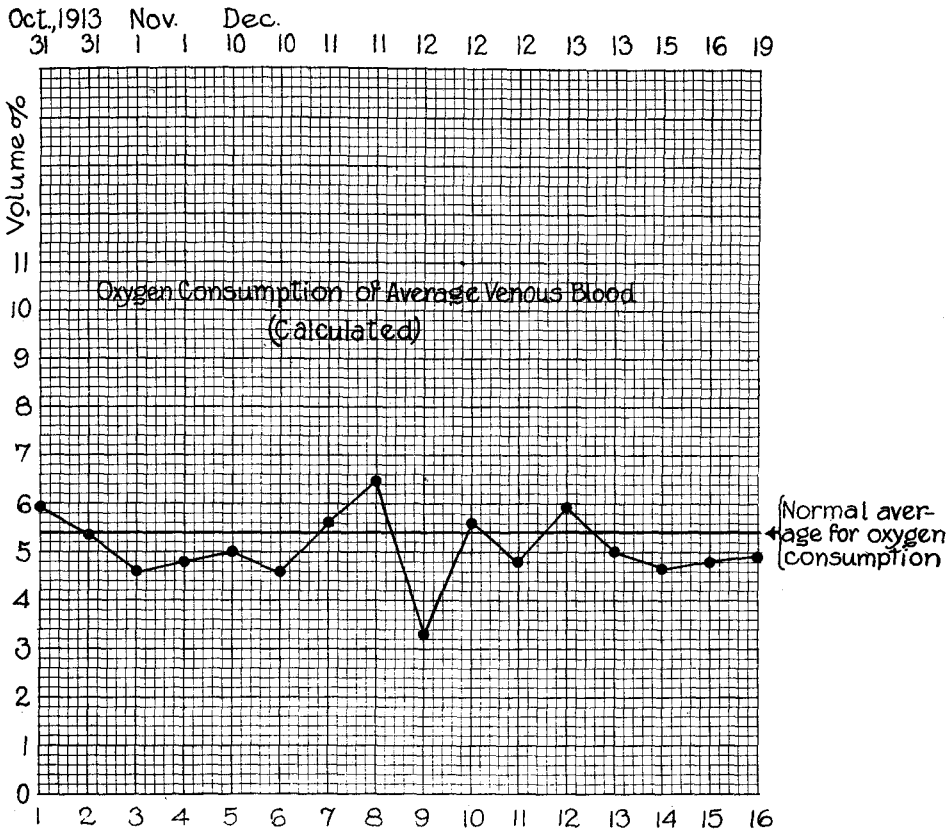
It is of interest for the interpretation of our results that the minute volume (output per minute from the heart) was determined 4 years ago in the Medical Clinic of the University of Copenhagen[9] on the same subject (the writer) on whom twenty determinations of the oxygen unsaturation of the venous blood have been done (Text-figs. 1 and 4).[10] From the figures indicating the values for the minute volume we can calculate the oxygen consumption of the average venous blood.[11]

[8] Compare Morawitz and Röhmer's observations (23).

[9] Lundsgaard (14), p. 397.

[10] See Table I, Paper I (1).

[11] The details of Krogh and Lindhard's method can be seen in the papers quoted in the bibliography, particularly in 8, 10, 13, and 14. The principles are the following: The subject is connected with an easily movable spirometer which contains a mixture of air and nitrous oxide. He mixes his lung air with the air in the spirometer during three to five respirations. He then stops breathing for a few seconds. Before and after the apneic period a sample of alveolar air is



TEXT-FIG. 3. Diagram showing the oxygen consumption of a normal man calculated from sixteen determinations of the minute volume of the heart by Krogh and Lindhard's nitrous oxide method.[12]

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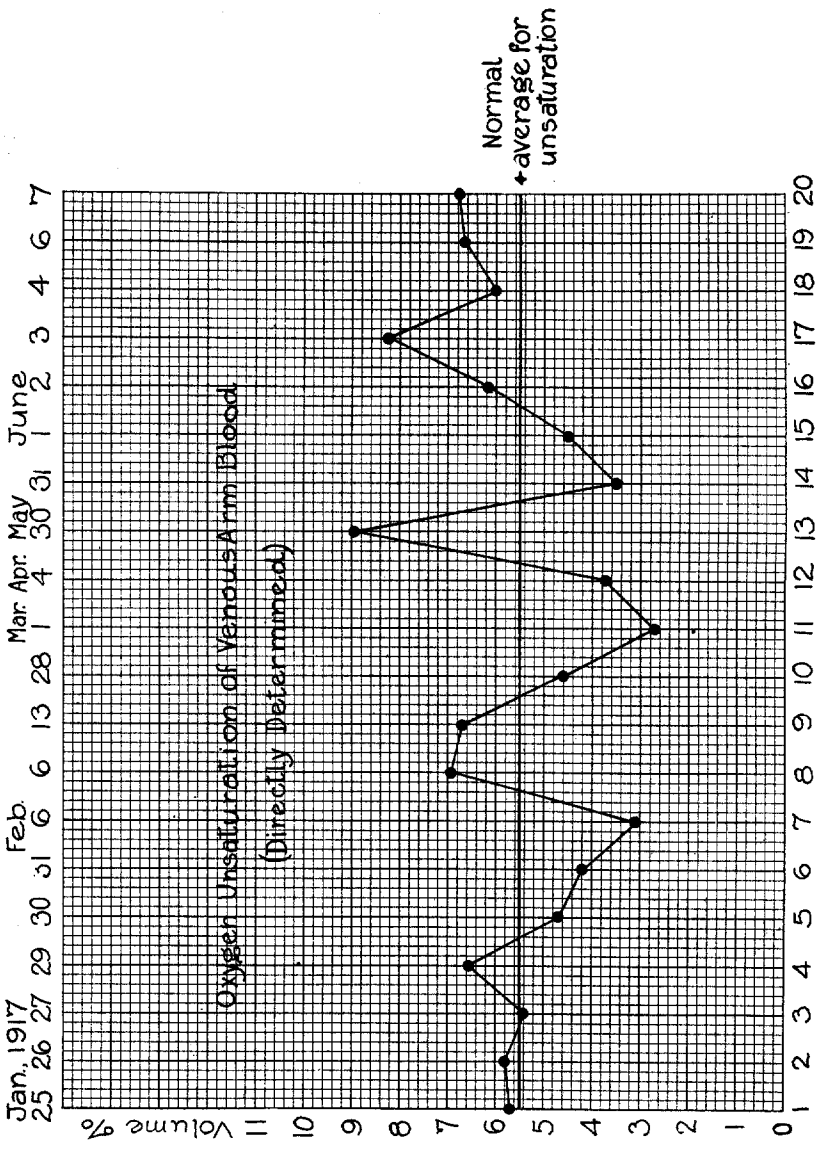
drawn and analyzed. On account of absorption by the blood flowing through the lung capillaries the second sample will contain a smaller amount of nitrous oxide and of oxygen than the first. The percentage in the difference between the two samples multiplied by the volume of air in the lungs will indicate the amount of blood flowing through the lungs during the period between the two samples. The output of blood per minute from the heart (the mihute volume) can then be calculated. Knowing the quantity of oxygen taken up by the blood during the same period we can calculate the amount of oxygen taken up by the body per 100 cc. of blood. When a correction is introduced for a possible abnormality in the percentage of hemoglobin this amount will indicate the oxygen consumption by the average blood. An extensive critical study of Krogh and Lindhard's method is published by Sonne (24). He expresses doubt about the reliability of the method on account of possible imperfect mixture of the lung air, not only in patients but in normal individuals as well. Krogh and Lindhard (25) have later on admitted that the difficulties shown by Sonne may exist.

[12] See Lundsgaard (14), p. 397, where the data for the blood flow can be obtained.



TEXT-FIG. 4. Diagram showing the oxygen unsaturation of the venous arm blood from the same normal subject[4] as in Text-fig. 3 (see also Text-fig. 1).

Sixteen determinations of the blood flow were made at that time. The calculated figures for the oxygen consumption in these sixteen experiments are given in Text-fig. 3. The average oxygen consumption for a considerable number of blood flow experiments on normal people (5.4 volume per cent) is indicated.

In Text-fig. 4 are given the figures for the oxygen unsaturation of the venous arm blood of the same subject (the writer) as reported in the first paper of this series.[10] The average line in this diagram (5.5 volume per cent) is from thirty-eight determinations of the oxygen unsaturation on eleven normal people reported in Paper I and shown in Text-fig. 1 in this paper. It will be seen how closely the values for the oxygen unsaturation determined by Van Slyke's method agree with the values for the oxygen consumption calculated from the determinations of the blood flow with Krogh and Lindhard's method. The average figures for normal individuals are the same (5.5 and 5.4 volume per cent). The variations for the two series on the same person agree rather closely. The variations in the oxygen unsaturation are more extensive than the variations in the values for the average consumption; *i.e.*, for the blood flow. This is probably due to variations in the local blood flow in the arm from which the blood is drawn. It is worth mentioning that the subject on whom the determinations were done has a very labile circulatory system (respiratory arrhythmia, changeable pulse, dermographia).[13] The significance of this agreement is that the amount of oxygen lost by 100 cc. of blood in passing through the forearm is approximately the same as the average loss in passing through the other body tissues. In view, nevertheless, of the undoubted possibility that the disturbing factors discussed may influence the unsaturation, we are not justified at present in interpreting unsaturation figures in terms of minute volume of the heart. What we believe we can do, is to fix the limits of the oxygen unsatura-

[13] The conception that the extensive variations in the oxygen unsaturation in this particular case are principally due to vasomotor changes, is supported by an observation (Cohn and Lundsgaard, personal communication) on the relation between the brachial blood pressure and the blood pressure in the arteries of the finger. The tension in the digital arteries was found more variable than in other normal subjects and more variable than the blood pressure nearer the center.

tion in subjects with normal circulation and normal lungs and study the variations observed in carefully controlled patients with symptoms of abnormal circulation and in patients with respiratory abnormalities. From the data thus obtained we may empirically standardize the figures for the oxygen unsaturation, learn the pathological conditions that affect it, and thus add it to the armamentarium that assists the clinician in accurate diagnosis. The empirical evolution of blood pressure measurement, made possible by accumulation of many determinations on clinically controlled patients, has shown how a quantitatively measurable factor, even though imperfectly explained physiologically, may prove to be of value in clinical medicine.

SUMMARY.

1. Thirty-one determinations of the total oxygen-combining power and the oxygen in the venous blood from vena mediana cubiti of sixteen resting patients are reported.

2. The difference between the total oxygen capacity of the hemoglobin and the oxygen in the venous blood, the oxygen unsaturation, is calculated.

3. In twelve patients with compensated heart lesions the unsaturation was found within normal limits, between 2.5 and 8 volume per cent.

4. In four patients with incompensated heart disease the values for the unsaturation were all above the normal limit, from 9.7 to 15.2 volume per cent.

5. A general discussion of the problem of interpreting the results is given.

6. A comparison is drawn between the oxygen consumption calculated from direct determination of the blood flow on a normal subject (the writer) and the oxygen unsaturation determined 4 years later on the same subject. A close agreement between the two series of values exists.

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