THE OXYGEN AND CARBON DIOXIDE CONTENT OF ARTERIAL AND OF VENOUS BLOOD IN NORMAL INDIVIDUALS AND IN PATIENTS WITH ANEMIA AND HEART DISEASE.

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The oxygen and carbon dioxide content of the blood in man, both in normal and in pathological conditions, has been studied but little, although the close relation of variations in the blood gases to alterations in the respiratory and circulatory mechanisms and to the blood flow has long been recognized. The difficulties of gas analysis upon small quantities of blood and in the technique of collection of samples have rendered the data hard to obtain and the reports in the literature scanty.

With the exception of a few observations by Hürter (1) upon arterial blood, all the data thus far published have been upon venous blood. Such, for example, have been the oxygen and carbon dioxide estimations of Morawitz and Röhmer (2) upon patients with anemia, those of Peabody (3) upon patients with lobar pneumonia, and those of Means and Newburgh (4) and of Lundsgaard (5) upon patients with cardiac disease.

Lundsgaard (6) has reported a series of thirty-eight determinations of the oxygen content of venous arm blood on twenty normal individuals. Hürter has reported four determinations of the oxygen and carbon dioxide content of normal human arterial blood. He found the arterial oxygen saturation to be between 93 and 100 per cent of the maximum capacity, which agrees with the values calculated from the dissociation curve for oxyhemoglobin.

The study of the arterial blood begun in 1912 by Hürter's analyses failed to develop further until 1918, when Stadie (7), continuing in the Hospital of The Rockefeller Institute the work on blood gases begun there by Lundsgaard and

Van Slyke (8), successfully undertook routine analyses of the arterial blood in patients with pneumonia.[1]

The determinations of Lundsgaard, which gave the most complete data available upon cardiac disease, were done with the recently devised blood gas apparatus of Van Slyke (8). The simplicity, accuracy, and speed attainable with this method render it a most satisfactory procedure. It has been employed in all the determinations here reported.

The collection of blood samples has been done in some instances by the method described by Lundsgaard for venous blood, and in others by the procedure recently described (9). Briefly, venous blood is collected without stasis from a vein at the bend of the elbow, and arterial blood from the radial artery, by means of a 20 cc. Luer syringe, to which is attached a sharp, short beveled needle.[2] The end of the syringe is rendered free from air by filling with 1 or 2 cc. of sterile paraffin oil, which is made to wet the barrel throughout its length. The technique of artery puncture is acquired easily, and when properly done causes little more discomfort to the patient than an ordinary venipuncture. The hand is held in the position recommended by Hürter (1), an assistant steadying it in place. After one has acquired a little practice, it is seldom necessary to use any local anesthesia. The only serious difficulty which may be met with is hematoma formation, which may take place from the opening in the artery. If proper steps to prevent it are neglected, a considerable extravasation of blood may occur and cause great pain and inconvenience. A small pressure bandage is applied tightly over the point of puncture immediately on withdrawal of the needle, and retained in place for at least an hour. Over 125 radial artery punctures and about 10 brachial artery punctures have now been done in this clinic, without any untoward effects.

Blood Gas Content in Normal Resting Individuals.

In Table I are given the results of oxygen determinations upon the arterial and venous blood of fifteen individuals with normal respiratory and circulatory apparatus, upon ten of whom simultaneous carbon dioxide analyses also were made. The artery punctures

[1] I am indebted to Dr. William C. Stadie for communicating to me his experience with the arterial puncture shortly after he had satisfied himself that it could be done without danger. In consequence of this, the present work on cardiac patients could be prosecuted simultaneously with Dr. Stadie's studies on pneumonia.

[2] The size used is No. 19 or 20 gauge, and the length 1½ to 2 inches. Care is taken that the needles are clean and very sharp.

TABLE I.

Arterial and Venous Oxygen and Carbon Dioxide Determinations upon Individuals with Normal Heart and Lung Findings.

Individual No.	Oxygen capacity (A).	Oxygen content of arterial blood (B).	Percentage saturation of arterial blood $\left(\frac{B}{A} \times 100\right)$.	Arterial oxygen unsaturation (A-B).	Oxygen content of venous blood (C).	Oxygen consumption (B-C).	Carbon dioxide content of arterial blood.	Carbon dioxide content of venous blood.	Temperature.	Pulse.	Respirations.	Blood pressure.	Remarks.
	vol. per cent	*vol. per cent*		*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	°F.			*mm.*	
1	23.74	23.04	97.5	0.70	17.61	6.43	51.76	57.15	98.6	78	20	$\frac{125}{80}$	
2	17.23	17.19	100.0	None.	14.62	2.57	54.69	56.71	98.6	86	22	$\frac{132}{84}$	
3	16.26	15.31	94.3	0.95	10.52	4.79	52.89	55.88	101.4	88	22	$\frac{120}{80}$	
4	20.60	19.83	96.3	0.77	13.46	6.37	46.46	51.70	98.4	84	20	$\frac{115}{78}$	
5	18.69	17.75	95.1	0.94	15.09	2.66	44.84	48.27	99.0	84	22	$\frac{130}{84}$	
6	20.60	19.79	96.0	0.81	12.74	7.05	49.67	54.56	98.8	80	20	$\frac{128}{78}$	
7	14.42	13.89	96.3	0.53	10.77	3.12	48.13	52.18	98.8	82	20	$\frac{120}{80}$	
8	24.67	24.08	97.6	0.59	15.81	8.27	50.55	58.74	98.6	84	22	$\frac{130}{78}$	
9	18.91	17.87	94.4	1.04	13.94	3.93	53.31	60.43	98.8	86	20	$\frac{126}{85}$	
10	21.23	21.01	99.0	0.22	13.08	7.93	44.58	50.90	98.6	82	20	$\frac{120}{80}$	
11	22.02	20.94	94.9	1.08	15.04	5.90			98.4	80	24	$\frac{130}{85}$	
12	21.46	21.23	98.9	0.23	14.66	6.57			98.6	78	22	$\frac{150}{95}$	
13	20.85	19.78	94.8	1.07	14.09	5.69			98.8	78	20	$\frac{128}{80}$	
14	22.08	21.00	95.0	1.08	13.98	7.02			98.6	76	20	$\frac{132}{78}$	
15	19.76	18.92	95.8	0.84	15.17	3.75			98.6	84	22	$\frac{140}{80}$	Right radial artery.
	19.58	19.10	97.6	0.48					98.6	84	22		Left brachial artery.
	19.82	19.24	97.2	0.58					98.6	84	22		Left radial artery.

for the three determinations made on the last case in the table, No. 15, upon the right radial artery, the left radial artery, and the left brachial artery, in the order named, were all done in the space of about 12 minutes and were made with the purpose of furnishing some experimental evidence of variations, if any exist, in the blood gas content of differently placed arteries.

The term oxygen unsaturation was introduced by Lundsgaard in his studies upon the venous blood, to indicate the difference in volumes per cent between oxygen content and oxygen capacity. It is used here in the same sense.

The term oxygen consumption, used by various writers, is applied here to the difference between the oxygen content of the arterial and of the venous blood in volumes per cent.

All the blood specimens for the determinations reported in this paper were uniformly collected in the following way: The artery puncture was first done and was immediately followed by the vein puncture. Both were done within a space of 3 to 5 minutes. The pulse and respirations were then counted. The blood pressure was that recorded on the history chart as the day's reading. All the individuals were bed patients unless otherwise indicated, and the specimens were taken at least 2 hours after the preceding meal.

The determinations were done immediately after collection, in nearly all instances those for carbon dioxide by one worker and those for oxygen by the other, simultaneously. When one person has done both series, those for carbon dioxide were made first because of the tendency of the carbon dioxide to diffuse out into the protecting paraffin oil layer.

The oxygen capacity readings were in all cases checked by colorimetric determinations by the Palmer method. However, the readings taken were the average of those made directly with the blood gas apparatus.

The factors producing variation in the oxygen content of arterial blood, aside from the obvious change in the oxygen-carrying capacity of the blood due to the varying content in hemoglobin, are chiefly those due to alterations in the lung ventilation (the alveolar oxygen tension), to pathological changes in the lung tissue, and to variations due to the rate of the blood flow, as found in normal individuals, for example, between the periods of rest and exercise.

It seems unlikely that variation in the phases of normal respiration in man usually effects as large changes as have been credited to it (1 to 2 volumes per cent).

The following observation upon the oxygen content of the arterial blood in an individual with Cheyne-Stokes respirations (the period of apnea was of 45 seconds duration), certainly producing a much greater interference with the blood gas exchange in the lungs than is caused by the normal variation in the phases of respiration, offers some evidence on this point.

Specimen.	Oxygen capacity.	Oxygen content of arterial blood.	Percentage saturation of arterial blood.
	vol. per cent	*vol. per cent*	
At height of period of dyspnea.....	24.61	24.51	100 (right radial artery).
" middle " " " apnea.....		22.00	89.4 (left " ").

In spite of the marked disturbance in the breathing, the difference in the arterial oxygen content between the two phases amounted to but 2.51 volumes per cent. It was not possible to make a satisfactory collection of a venous sample, owing to the much slower rate of flow of venous blood, collected without stasis.

Little variation can take place in the gas content of the blood in the radial artery, as compared with that leaving the left heart, and there can be no changes in the particular sample due to variations in metabolism or local variations in the blood flow, which are not common to the arterial blood as a whole, as it exists in the larger vessels. The close agreement of the gas content in blood removed from various arteries (Case 15, Table I) furnishes experimental evidence on this point. The oxygen content of normal venous blood varies widely,[3] due to a combination of many factors. The most important of these are the variations in the gas content of the arterial blood and the local variations in the blood flow. The influence of the first factor is well illustrated in the determinations on patients with severe anemia, given in Table II.

The very low oxygen content of the venous blood in these patients with the resulting extremely low pressure-head in the capillaries is an indication of the ability of the tissues to take up oxygen from the

[3] According to Lundsgaard between 18 and 9.5 volumes per cent.

TABLE II.

Case No.	Date.	Oxygen capacity.	Oxygen content of arterial blood.	Arterial oxygen unsaturation.	Oxygen content of venous blood.	Oxygen consumption.	Diagnosis.
	1919	*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	
1	Mar. 27	4.12	3.31	0.81	1.55	1.76	Acute lymphatic leucemia.
2	Apr. 1	4.17	3.83	0.34	1.31	2.52	Uremia. Bleeding from gums and vagina.
3	" 15	3.68	3.50	0.18	0.87	2.63	Pernicious anemia.

blood over a wide range of oxygen pressures. Even at these low levels the oxygen consumption remained within practically normal limits.

The effect upon the blood gases of alteration in the blood flow may be shown by a comparison of the analyses of blood samples taken during periods of rest and after exercise.

The experiment shown in Table III was made upon a normal individual, white, male, age 29 years. Two determinations were made (*a*) after lying down quietly for 30 minutes, and (*b*) immediately after 15 minutes of brisk exercise, consisting of arm and trunk movements, and vigorous hopping about the room until quite dyspneic. The pulse and respiration readings were then made while the arterial blood was being withdrawn. The collection of the venous blood sample was then made.

TABLE III.

Condition.	Oxygen capacity.	Oxygen content of arterial blood.	Percentage saturation of arterial blood.	Oxygen content of venous blood.	Oxygen consumption.	Carbon dioxide content of arterial blood.	Carbon dioxide content of venous blood.	Temperature.	Pulse.	Respirations.
	vol. per cent	*vol. per cent*		*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	*°F.*		
Resting	22.04	21.09	95.6	15.08	6.01	53.33	56.90	98.6	86	18
After exercise	22.41	19.19	85.5	12.93	6.26	32.25	41.14		140	30

I hope to report at a later date a study in detail of the effects of exercise. Presumably later changes of a compensating nature may occur, in the direction of a restoration of the normal arterial oxygen saturation possibly by increased secretion of oxygen by the lung epithelium, or by regulation of the oxygen consumption in the tissues, which may explain the phenomenon of acquiring one's second wind.

Blood Gases in Cardiac Disease.

The methods just described have been applied to the study of the gas content of the blood in persons suffering from cardiac disease.[4] A considerable number of determinations has been made upon the arterial and venous blood of patients, clinically compensated, and without cardiac arrhythmias, at rest in bed. No noteworthy deviation from the values given above for normal persons has been found and it seems of no practical value to publish these results. In Table IV are given the results of determinations upon nine persons with decompensated circulatory disturbances. The clinical data upon these patients are given below.

Case 1.—J., white, male, machinist; age 50 years.

Diagnosis.—Emphysema; arteriosclerosis; hypertension; chronic myocarditis.

Previous History and Symptoms.—Shortness of breath on exertion for many years; four periods of decompensation in the past 6 years. Present attack has lasted for 3 months and has been particularly bad for the past 5 days. No history of syphilis or rheumatic fever.

Physical Examination (Apr. 25, 1919).—Slight cyanosis of finger-tips. Moderate orthopnea. Moderate engorgement of neck veins. Cardiac borders are hard to define accurately on account of the marked grade of emphysema present, but the dullness is considerably increased both to left and to right; no murmurs; no arrhythmia. Second sounds at aortic and pulmonic areas of about equal intensity. Dullness at right lung base, with many moist râles at either base. No ascites. Pitting edema of lower legs. Wassermann reaction negative.

Apr. 28. Patient has shown rapid response to treatment. Practically no respiratory difficulty in bed. Lungs clear. Edema almost gone. Pulse regular except for one extrasystole with each 10 to 15 heart beats.

May 5. No longer any signs of myocardial insufficiency at rest in bed. Has severe attacks of cardiac dyspnea at night.

[4] The samples were collected in these cases, as in all herein reported, upon bed patients while recumbent. The only exception was in the case of badly decompensated patients with a marked grade of orthopnea, in which instances the blood was taken from the patients when they were in a sitting posture.

TABLE IV.

Arterial and Venous Oxygen and Carbon Dioxide Determinations upon Patients with Decompensated Circulatory Disturbances.

Case No.	Age.	Date.	Oxygen capacity (A).	Oxygen content of arterial blood (B).	Percentage saturation of arterial blood $\left(\frac{B}{A} \times 100\right)$.	Arterial oxygen unsaturation (A-B).	Oxygen content of venous blood (C).	Oxygen consumption (B-C).	Carbon dioxide content of arterial blood.	Carbon dioxide content of venous blood.	Temperature.	Pulse.	Respirations.	Blood pressure.	Diagnosis.
	yrs.	*1919*	*vol. per cent*	*vol. per cent*		*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	*vol. per cent*	*°F.*			*mm.*	
1	50	Apr. 25	20.33	17.21	84.8	3.12	6.90	10.31	46.37	54.04	98.4	108	30	$\frac{138}{80}$	Emphysema; hypertension; chronic myocarditis.
		" 28	21.80	19.56	89.7	2.24	16.30	3.26	46.30	48.71	98.7	78	20	$\frac{160}{100}$	
		May 5	23.12	22.25	96.4	0.87	15.51	6.74	44.29	56.10	98.6	84	20	$\frac{160}{105}$	
2	57	Feb. 24	19.00	17.05	89.7	1.95	9.26	7.79	45.44	48.28	98.0	70	24	$\frac{140}{95}$	Emphysema; chronic myocarditis.
		" 29	18.67	17.90	95.9	0.77	9.49	8.41	48.17	52.71	98.6	72	20	$\frac{135}{90}$	
		Mar. 15	18.50	17.75	95.9	0.75	12.62	5.13	49.59	53.06	98.2	72	20	$\frac{140}{100}$	
3	50	Feb. 21	23.85	19.41	81.4	4.44	9.61	9.80			98.0	90	20	$\frac{120}{85}$	" " bronchitis; chronic myocarditis.
		" 26	23.33	20.33	87.3	3.00	12.37	7.96			98.6	84	20	$\frac{130}{85}$	
		Mar. 4	23.61	21.22	89.9	2.39	13.66	7.56			98.4	80	20	$\frac{136}{90}$	

4	60	Mar. 29	16.78	13.90	82.8	2.88	10.84	3.06	41.40	44.19	97.8	80	32	154/100	Chronic myocarditis; auricular fibrillation.
		Apr. 5	16.61	15.23	91.7	1.38	9.58	5.65	43.51	46.48	98.4	48	24	140/98	
5	30	Feb. 16	19.87	17.70	89.1	2.17	6.30	11.40	38.88	41.76	98.8	96	24	110/68	Chronic rheumatic endocarditis; mitral stenosis and insufficiency; auricular fibrillation.
		" 24	19.68	18.15	92.3	1.53	10.42	7.73	42.68	47.05	98.7	92	28	115/80	
		Mar. 4	19.74	17.68	89.5	2.06	8.31	9.37			98.4	96	28	112/78	
		" 18	19.62	17.90	89.8	1.72	13.07	4.83	43.80	47.61	98.6	88	26	116/80	
6	38	Apr. 9	18.68	16.46	88.2	2.22	12.94	3.52			98.6	140	36	110/80	Mitral and aortic insufficiency; adherent pericardium; bilateral hydrothorax.
		" 10	19.09	15.56	81.5	3.53	8.49	7.07			99.0	170	48	103/75	
7	39	Jan. 2	18.85	16.48	87.4	2.37	7.29	9.19	38.65	43.21	98.4	100	36	135	Syphilis of aorta; aortic insufficiency.
		" 10	22.92	20.52	85.9	2.40	16.10	4.42	42.87	46.48	98.6	98	26	145	
		" 20	22.48	21.09	93.7	1.39	15.42	5.67	43.62	49.01	98.4	78	20	145	
8	33	Apr. 29	20.74	19.31	93.2	1.43	11.13	8.18	31.42	37.53	98.2	96	30	128	Aortic insufficiency.
		May 1	22.50	21.30	94.7	1.20	11.64	9.66	42.80	45.70	98.0	84	24	125	
		" 6	23.25	22.49	96.8	0.76	17.55	4.94	41.47	46.52	98.6	88	24	125	
9	42	Jan. 30	17.97	16.71	93.0	1.26	6.64	10.07			97.4	100	28	140	" "
		Feb. 24	16.33	15.96	97.7	0.37	9.82	6.14			98.2	90	22	148	

It will be noticed that the percentage saturation of the arterial blood with oxygen at the time of admission was much lower than normal. The chest showed well marked emphysema, and signs of congestion in the lungs were apparent. With the clearing up of this congestion and the return to compensation the arterial oxygen saturation quickly returned to normal. At the same time the oxygen content of the venous blood rose and the oxygen consumption correspondingly became less.

Case 2.—L., negro, male, laborer; age 57 years.

Diagnosis.—Emphysema; hypertension; chronic myocarditis.

Previous History and Symptoms.—Recurrent periods of decompensation for several years. No history of syphilis or rheumatic fever.

Physical Examination (Feb. 24, 1919).—Moderate dyspnea. No definite cyanosis. Huge heart, with apex in midaxilla; no murmurs; rate regular except for an occasional extrasystole. Moderate dilatation of neck veins. Slight edema of legs. Numerous moist râles and dullness at the lung bases. Liver edge well below the costal margin and tender. Wassermann reaction negative.

Feb. 29. Patient has responded well to treatment. The lungs are clear. No liver tenderness. Practically no respiratory distress. Still some extrasystoles; no cyanosis; no edema.

Mar. 15. Condition is not changed. The extrasystoles persist.

The rapid improvement in the symptoms of decompensation in this case, which responded to treatment almost as rapidly as Case 1, produced coincident improvement in the oxygenation of the arterial blood with the clearing up of the signs of lung congestion.

Case 3.—S., white, male, occupation not given; age 50 years.

Diagnosis.—Chronic bronchitis; emphysema; chronic myocarditis.

Previous History and Symptoms.—Symptoms of emphysema and of chronic bronchitis for many years. Dyspnea on exertion for 25 years. Winter cough for past 10 years. Gradual onset more recently of the cardiac symptoms.

Physical Examination (Feb. 21, 1919).—Signs of right-sided cardiac hypertrophy, with secondary myocardial insufficiency. Voluminous, hyperresonant lungs; bases descend very little, and there are numerous fine râles. Heart borders overlapped by lungs, but distinctly enlarged to the right; the sounds are faint, almost inaudible, but seem clear. Slight edema of legs; no liver tenderness; no particular engorgement of the superficial veins. Marked cyanosis of lips, face, and finger-tips, out of proportion to the involvement of respiration. Expectoration of purulent sputum. Wassermann reaction negative.

Feb. 26. Condition is greatly improved, but there are still many râles at the lung bases, and there is considerable cyanosis.

Mar. 4. Condition further improved. Râles still present at bases. Cyanosis still present.

In this case there was distinctly evident a superimposed lung factor, long standing chronic bronchitis and emphysema, which produced a marked effect on the normal oxygenation of the blood. Even at discharge the percentage saturation of the arterial blood with oxygen was below the low normal value, and the patient was distinctly cyanotic. This chronic state of cyanosis had been present for years, although much aggravated at the time of his cardiac break. The oxygen content of the venous blood was about normal over the period of the observations.

Case 4.—M., negro, male, laborer; age 60 years.

Diagnosis.—Chronic myocarditis; auricular fibrillation.

Previous History and Symptoms.—Onset of disorder in Nov., 1918, with cough and shortness of breath. Swelling of legs 3 days ago.

Physical Examination (Mar. 29, 1919).—Marked orthopnea. Huge heart, enlarged downward and to the left; second pulmonic sound accentuated; no murmurs; rate totally irregular. Pulse deficit about 10 beats per minute. Many moist râles at either base, especially at the right. No particular cyanosis. Slight tenderness at right costal margin. Pitting edema of legs, extending up to the knees. Neck veins somewhat distended. Wassermann reaction negative.

Apr. 5. Condition much improved, although distinct orthopnea is still present. Pulse very slow, 48 per minute, all the beats now coming through to the wrist. No cyanosis or liver tenderness. Edema rapidly clearing up. Râles at lung bases have practically cleared up. Electrocardiogram indicates auricular fibrillation.

Case 5.—C., negro, female; age 30 years.

Diagnosis.—Mitral stenosis and insufficiency; auricular fibrillation.

Previous History and Symptoms.—Rheumatic fever 3 years ago. No history of syphilis. Cardiac symptoms, especially shortness of breath and occasional precordial pain, have been present during the past 10 months.

Physical Examination (Feb. 16, 1919).—Marked orthopnea and dyspnea. Totally irregular heart; about 15 beats per minute fail to come through to the wrist. Râles and dullness at the lung bases. Only moderate cyanosis of lips and finger-tips. Marked edema of legs and hands. Tender, palpable liver; certainly some fluid in abdomen. Systolic and diastolic murmurs at apex. Snapping first sound. Accentuated second pulmonic sound. Wassermann reaction negative.

Feb. 24. Rather slow response to digitalis. Condition, however, seems improved. Signs of congestion somewhat less marked.

Mar. 4. Slow but distinct improvement. Edema gone. Râles and dullness no longer present at lung bases.

Mar. 18. Seems practically compensated clinically. Heart rate 88, all beats coming through to the wrist. Lungs clear of râles.

The percentage saturation of the arterial blood in this patient tended to remain definitely below the normal figure, even after compensation was restored. As far as could be determined clinically, the lung bases were clear. There was nothing to suggest adherent pericardium; the heart, although large, was clearly movable with change in position. Nevertheless, the patient gave every indication of having had long standing cardiac disease, despite her rather short and possibly inexact history. She exhibited well marked mitral facies and clubbing of the fingers. The x-ray of the chest was reported as showing considerable infiltration, particularly of the lower parts of both lungs. The figures for the oxygen consumption tended to show the same erratic irregularities to which, in auricular fibrillation, Lundsgaard (5) has already drawn attention.

Case 6.—N., white, male, sailor; age 38 years.

Diagnosis (*Autopsy*).—Mitral and aortic insufficiency; adherent pericardium; bilateral hydrothorax.

Previous History and Symptoms.—Patient was admitted very ill. Said to have been sick 2 weeks with shortness of breath. Expectoration of blood-tinged sputum for several days.

Physical Examination (*Apr. 9, 1919*).—Marked dyspnea and cyanosis of lips, face, and fingers. Rapid but regular pulse. Lungs practically clear. Marked retrosternal dullness and huge heart. Moderate engorgement of neck vessels. No edema. Soft, pulsating liver, edge at umbilicus. Systolic and diastolic murmurs at apex and soft aortic diastolic murmur to left of sternum. Wassermann reaction negative.

Apr. 10. Condition decidedly worse, with delirium and marked cyanosis. No fever. Pulse weak and rapid. Heart and lung signs unchanged, except that signs of pulmonary edema developed rapidly shortly after the blood specimens were taken. Patient died about 16 hours later.

Autopsy.—Huge heart to which the pericardium was everywhere adherent. The lungs were clear except for the compression due to a bilateral hydrothorax of 1,500 cc. on either side. The liver was enlarged.

This case is of particular interest in that the patient showed cyanosis of extreme degree, and it was thought possible that pneumonia was present as well as the cardiac disease. This opinion proved incorrect, however, and the lung changes were all found to be secondary to the cardiac deficiency.

All the evidence pointed to a rapid appearance of serious respiratory embarrassment on account of the acute decompensation and the resulting accumulation of pleural fluid. It seems not unlikely that the time element plays a certain part in some of these cases, and that in this particular one, had the respiratory apparatus had time to adjust itself to the new conditions, a more efficient oxygen saturation of the arterial blood would have been later effected, even if the cause of the lung compression remained.

Case 7.—S., negro, male, laborer; age 35 years.

Diagnosis.—Syphilis of aorta; aortic insufficiency.

Previous History and Symptoms.—Indefinite history of shortness of breath about 1 year ago. This improved but again became serious about 6 months ago, since which time he has worked but little. Swelling of the legs and orthopnea for past 3 months. No history of rheumatism.

Physical Examination (Jan. 2, 1919).—Orthopnea; swelling of neck veins; massive pitting edema of legs; slight cyanosis. Huge heart with loud aortic diastolic murmur; rate regular. Palm's breadth of marked dullness at right base; many moist, bubbling râles at either base. Wassermann reaction positive.

Jan. 10. Condition has improved with rest and the usual cardiac treatment. Nevertheless, the pulse is 98 and there is still distinct orthopnea. Râles still persist at either base and considerable dullness is still present at the right base.

Jan. 20. Patient has gradually improved and there is no longer any respiratory distress. Pulse 78. Lung bases practically clear. No edema and no liver tenderness.

Case 8.—H., negro, male, laborer; age 33 years.

Diagnosis.—Aortic insufficiency.

Previous History and Symptoms.—Shortness of breath for 2 months. Swelling of legs noticed 2 days ago. History of syphilis. Some bloody sputum expectorated the day previous to admission.

Physical Examination (Apr. 29, 1919).—Moderate dyspnea and slight orthopnea. No cyanosis or dilatation of superficial veins. Lungs clear except for a few crackles at the bases; no dullness. Huge heart, with apex in the anterior axillary line; increased retrosternal dullness; musical aortic diastolic murmur along left sternal border and in aortic area. Collapsing pulse, regular. Very slight liver tenderness. Moderate edema of lower legs. Wassermann reaction positive.

May 1. Some bloody expectoration yesterday. Considered likely that lung infarcts have been produced, but no pain in chest is complained of, and lungs show no new findings. Breathing improved. Edema less.

May 5. Compensation is now almost regained. Lungs are clear. Pulse much slower and regular. No cyanosis; no edema; no liver tenderness.

Case 9.—R., negro, male, laborer; age 42 years.

Diagnosis.—Syphilis of aorta; aortic insufficiency.

Previous History and Symptoms.—Has had shortness of breath for several months and swelling of legs for 2 weeks. Hard chancre 4 years ago.

Physical Examination (Jan. 30, 1919).—Orthopnea and dyspnea are marked. No cyanosis. Lungs are practically clear. Wide retrosternal dullness. Huge heart with the apex impulse in midaxilla, with signs of aortic insufficiency. Typical pulse, regular in rhythm. Slight edema of legs. Tenderness at right costal margin. Some engorgement of neck vessels. Wassermann reaction positive.

Feb. 24. Compensation is quite regained. Lungs clear.

The last three patients (Cases 7, 8, and 9) exhibited the same type of cardiac lesion with varying degrees of decompensation. Of these, Case 7 was suffering from a much more severe break than the other two, and his recovery under cardiac therapy (milk diet, restricted fluids, digitalis, and rest) was much slower. His lower chest was filled with moist râles at the time of the first examination, and it seemed likely that there was a certain amount of pleural effusion at the right base.

Although it was considered probable at the time (May 1) that Patient H. (Case 8) had had a pulmonary infarct because of his bloody sputum, no other signs appeared, there was no abnormality found in the content of blood gases, and his return to compensation was prompt.

The condition of Patient R. (Case 9) at the time of the first blood oxygen examination, on the 4th day following admission, was not serious, except for his rather marked orthopnea and enlarged liver. The lungs showed no outspoken evidence of congestion. The oxygen consumption at this examination was high—presumably there was rather marked increase in the metabolism with the severe respiratory exertion, but the percentage saturation of the arterial blood was about normal. At the time of the second examination (February 24), when the findings were normal, compensation had been entirely regained.

CONCLUSION.

An examination of the data presented in these cases of cardiac disease indicates the importance of the effect of the primary condition of the lungs upon the oxygen saturation of the arterial blood during periods of decompensation. Emphysema and chronic inflammatory processes appear to aggravate greatly the effect of the passive congestion due to the cardiac insufficiency alone.

It is desired to take up at another time the effects produced by disturbances to the passage of air through the trachea and its branches. Sufficient data have accumulated, however, to warrant the expression of an opinion that the time element is of importance. Sudden mechanical disturbances to breathing produce abnormal changes in the concentration of the blood gases. These disturbances, after a period, even if unrelieved, are in large part compensated.

Because of the striking variation in the color of blood due to changes in its oxyhemoglobin content, it appears possible to relate the degree of cyanosis in individuals to the extent of oxygen unsaturation. The determining factor in the production of cyanosis must be the color of the blood in the superficial capillaries and veins, although the amount of skin pigmentation no doubt influences greatly its appreciation clinically. The cause of the phenomenon probably varies. One type is produced where incomplete saturation of the arterial blood is at fault, due to disturbance in the normal gas exchange in the pulmonary alveoli, as in pneumonia. In many cases of heart disease it may be a combination of both arterial and primarily venous unsaturation; in others it is the venous unsaturation due to stasis which is chiefly at fault. It is probable that in other instances there may be disturbance neither in the pulmonary exchange nor in the blood flow in the larger venous vessels, but that constriction of peripheral vessels due to local stimuli, with consequent local slowing of the blood flow and necessarily increased oxygen consumption, produces a type of "capillary" cyanosis, such as is, for example, strikingly seen in bathers exposed to the cold air when wet.

SUMMARY.

1. The oxygen content of venous and of arterial blood from fifteen essentially normal individuals at rest in bed has been determined.

2. The percentage saturation of the arterial blood has varied between 100 and 94.3. The average is 95.5 per cent.

3. The oxygen consumption has varied between 2.6 and 8.3 volumes per cent.

4. The oxygen content and the percentage saturation of arterial blood taken at close intervals from three different peripheral arteries of a normal individual have shown values agreeing within the limits of error. Analyses of the blood gases of a normal individual, at rest and after exercise, have shown a lowering of the percentage oxygen saturation of the arterial blood and a diminished carbon dioxide content after exercise.

5. In three persons with severe anemia the saturation of the arterial blood has not differed from the normal. Very low absolute values were found for the oxygen content of the venous blood, but the normal oxygen consumption has been maintained.

6. The carbon dioxide content of the arterial blood from ten normal individuals has varied between 54.7 and 44.6 volumes per cent. That of the venous blood has varied between 60.4 and 48.3 volumes per cent.

7. No deviations from the normal values for oxygen and carbon dioxide were found in venous and arterial blood from cardiac patients without arrhythmias, well compensated, and at rest in bed.

8. A series of determinations has been made upon nine cardiac patients with varying degrees of decompensation. The percentage oxygen saturation of the arterial blood on admission was abnormally low in seven of these cases. With the return to compensation and with the clearing up of pulmonary symptoms, the percentage saturation of the arterial blood returned to normal in four of them.

9. In a case of long standing mitral endocarditis with auricular fibrillation it remained low over a period of 1 month of observation.

10. In a case of chronic myocarditis secondary to emphysema and chronic bronchitis, it remained low over the period of observation.

11. Normal values for the percentage saturation of the arterial blood were found in two individuals with decompensated aortic disease but without physical signs of extensive pulmonary involvement.

12. The oxygen consumption tended to be high in individuals with cardiac disease during the periods of marked decompensation and to be lower as compensation was regained.

13. The data presented indicate that at least in many circulatory diseases during decompensation, particularly when there are physical signs of pulmonary congestion, there is a disturbance of the pulmonary exchange, as indicated by the lowering of the percentage saturation of the arterial blood with oxygen.

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