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ON THE PARTIAL PRESSURES OF OXYGEN AND CARBON DIOXIDE IN ARTERIAL BLOOD AND ALVEOLAR AIR.

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Ever since the work of Haldane and Priestley(1) (1905) on the pressure of CO_2 in alveolar air, and that of Krogh(2) (1910) on the mechanism of gas exchange in the lung, it has been generally believed that the partial pressure of CO_2 is the same (to a close approximation) in arterial blood and alveolar air. The relation, however, of the partial pressure of oxygen in alveolar air to that in the arterial blood has not been determined with the same accuracy. This uncertainty is due in part to the dispute concerning the rôle played by the lungs in oxygen transfer, in part to difference of opinion regarding the mechanics of pulmonary ventilation, and also to lack of precise knowledge of the facts. It is the purpose of this paper to present data with reference to gas exchange in the lung. Our experiments have been performed on normal men while they were breathing air or low oxygen mixtures.

Method.

The modification of the Haldane-Priestley method used in obtaining samples of alveolar air has been previously reported(3). For the present work we have estimated the partial pressures of both CO_2 and O_2 in the same samples of air. In the above-mentioned communication the method used for the determination of arterial CO_2 pressure was described. At that time, however, equilibration of the blood was carried out at 37·5° C. In the present series of experiments all equilibrations were done at the oral temperature of the subject. Since the temperatures found under basal conditions for normal subjects are in general at least one degree below the standard temperature, the earlier determinations are about one millimetre higher than the present determinations, owing to the effect of temperature on the CO_2 dissociation curve. A small error in the earlier work is due to neglect of the effect of acid formation in

blood after its removal from the body. We have now attempted to eliminate this error by determining the magnitude of the acid effect in the blood of each subject by the following procedure. For each case, 10 c.c. of venous blood were equilibrated, immediately after bleeding, for periods of 90 to 120 minutes at 37° C., and $pCO_2 = 40$ mm. Samples of 1 c.c. were removed from the tonometer at intervals of 10 to 30 minutes and the CO_2 content determined. Since the rate of decrease in CO_2 content was found to be a linear function of time, extrapolation to $t = 0$ by means of the line drawn through the determined points should give the true bicarbonate level of the blood in the body. For the sake of convenience the data are plotted as in Fig. 1. They indicate that the rate of

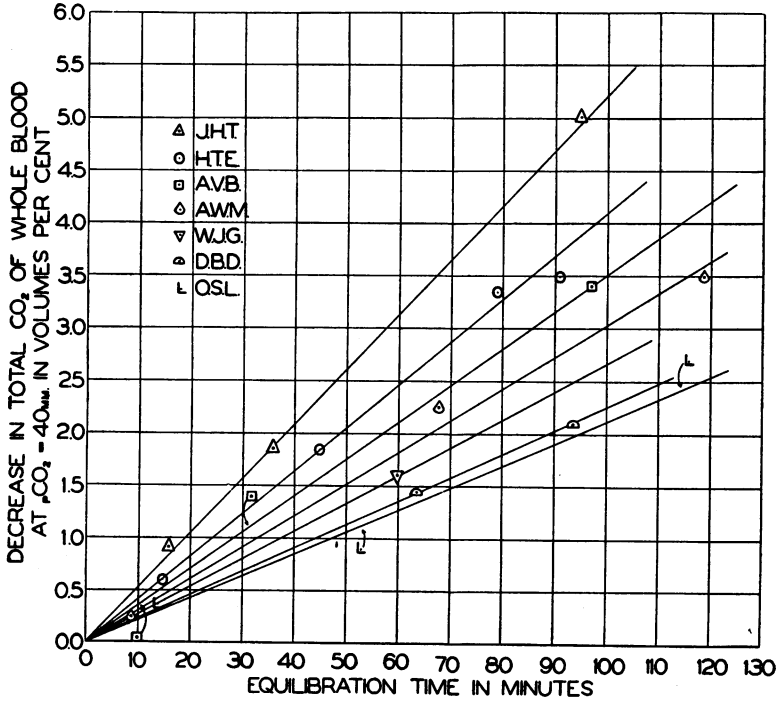

Fig. 1.

acid formation varies from subject to subject. The average loss of bicarbonate for all of our subjects was about 0·4 vol. p.c. during equilibration. For each case the level of the fully oxygenated CO_2 dissociation curve was corrected for the acid change as thus determined. The curve was also corrected to the level corresponding to the p.c. oxyhæmoglobin found in arterial blood. This is a simple correction owing to

the linear relation between the bicarbonate content of blood and the oxygenation of hæmoglobin at constant pressure of carbon dioxide.

In previous experiments it has been customary to refer the experimentally determined saturation of arterial blood to a standard oxygen dissociation curve in order to estimate the partial pressure of oxygen. In our present experiments we have tried to avoid the small errors involved in such a procedure, which by cumulation may become large, by (1) determining two or three points on the oxygen dissociation curve in each experiment, (2) adjusting the carbon dioxide pressure of equilibration near that of blood, and (3) correcting the points to the precisé value for carbon dioxide pressure of arterial blood. To make this correction, typical oxygen dissociation curves of a normal individual at carbon dioxide pressures of 20 and 40 mm. were plotted logarithmically with $\log pO_2$ as a function of $\log \frac{Hb}{HbO_2}$. We then assume that, even though the position of these two curves may vary from case to case, the effect of small changes in carbon dioxide pressure upon percentage saturation is the same for all normal individuals, and further that the effect is linear. Many observations in this laboratory indicate that the first assumption is correct, and that the second assumption involves at most an error of about 1 p.c. in the corrected value for saturation when the difference in carbon dioxide pressures is 10 mm. This is a greater difference than any that has been involved in our corrections. The data are given in Table III.

Arterial blood was used for all points determined on the CO_2 and O_2 dissociation curves, the determinations being made with the van Slyke-Neill manometric apparatus. To insure complete saturation of the blood for oxygen capacity determinations, it was equilibrated in tonometers containing air for ½ hour or longer at 20° C.

Low oxygen mixtures for breathing were obtained by diluting outside air with nitrogen in a spirometer of 600 litres capacity. The duration of experiments when such mixtures were breathed was 35 minutes or longer. The periods for which the total ventilations are recorded in Table III were begun with the 25th minute, each determination representing a period of 10 minutes. Arterial puncture under novocaine anæsthesia was done immediately after this collection period. The subjects were all in basal condition, and were studied in the prone position.

The partial pressure of carbon dioxide.

In 1924 Bock and Field(3) published a series of 24 determinations of alveolar and arterial CO_2 pressures. The average of 21 of these showed a difference in pressure of not more than 0·5 mm. This result agreed closely with that of Krogh's study of rabbits with his aerotonometer method(2). Similar data in man were shortly obtained by Dautrebande(4), and later by Richards(5). Recently Grollman(6) has concluded that true alveolar CO_2 pressures cannot be obtained while the subject lies flat owing to the reduction in vital capacity and complemental air, characteristic of the prone position. However, no experimental data are given in support of this conclusion. We now present another set of determinations, recorded in Tables I and II. With one

TABLE I. Carbon dioxide dissociation curves.

Subject	Date	Oral temp. °C.	pCO_2 mm. Hg	O_2 saturation p.c.	Total CO_2 content, whole blood: Uncorrected vol. p.c.	Total CO_2 content, whole blood: Corrected vol. p.c.
D. B. H.	7 xi. 28	36·8	36·0	100	48·35	49·35
			59·0	100	57·95	58·95
					57·60	58·60
H. T. E.	7 xi. 28	36·9	34·0	100	43·1	44·0
			39·4	—	46·1	47·0
J. H. T.	7 xi. 28	36·8	34·6	100	44·5	46·3
			61·2	100	56·8	58·6
					56·65	58·45
A. W. M.	9 xi. 28	36·8	29·5	100	43·3	44·1
					43·4	44·2
			56·1	100	55·3	56·1
O. S. L.	9 xi. 28	36·8	28·0	100	42·15	42·75
					42·4	43·0
			53·6	100	54·6	55·2
					55·1	55·7
W. J. G.	9 xi. 28	37·1	33·9	100	44·95	45·60
					45·30	45·95
			67·5	100	59·1	59·75
					59·1	59·75
D. B. D.	26 ii. 29	36·5	35·0	100	48·9	50·05
			67·5	100	61·0	62·15
					61·2	62·35
W. J. G.	12 iii. 29	36·2	35·6	100	46·8	47·8
					46·95	47·95
			60·2	100	57·3	58·3
					57·3	58·3

TABLE I. Carbon dioxide dissociation curves (*contd.*).

Subject	Date	Oral temp. ° C.	pCO_2 mm. Hg.	O_2 saturation p.c.	Total CO_2 content, whole blood: Uncorrected vol. p.c.	Total CO_2 content, whole blood: Corrected vol. p.c.
H. T. E.	13 iii. 29	—	44·2	100	48·0	49·3
					48·0	49·3
			41·3	100	46·9	48·2
					46·9	48·2
J. H. T.	20 iii. 29	36·3	28·2	100	40·5	43·4
					40·55	43·45
			61·2	100	56·6	59·5
					56·6	59·5
A. W. M.	22 iii. 29	—	33·3	100	43·6	44·4
			77·2	100	61·05	61·9
A. W. M.	22 iii. 29	—	36·0	92·5	44·9	47·3
			36·1	85·4	45·2	47·2
			57·0	91·3	54·85	57·2
D. B. D.	27 iii. 29	36·4	41·5	93·7	52·2	52·35
					52·1	52·45
			59·5	100	58·8	59·5
					58·65	59·35
D. B. D.	27 iii. 29	36·6	35·4	75·5	49·9	49·4
					49·9	49·4
			62·8	62·6	62·65	61·35
					62·45	61·25
A. V. B.	29 iii. 29	36·1	38·8	96·6	49·6	50·0
			61·0	100	57·8	58·4
A. V. B.	29 iii. 29	36·0	35·9	88	47·5	48·1
			51·1	80	57·8	58·0
A. V. B.	29 iii. 29	36·3	30·6	70	44·9	44·6
A. V. B.	3 iv. 29	97·2	42·0	97·4	49·5	50·1
A. V. B.	3 iv. 29	97·2	22·3	98·4	39·2	39·8

TABLE II. Comparison of the partial pressure of carbon dioxide in alveolar air and arterial blood.

Subject	Date	Oxygen in inspired air p.c.	Total CO_2 content arterial blood vol. p.c.	Arterial pCO_2 corrected mm. Hg	Alveolar pCO_2 mm. Hg	Δ mm. Hg
D. B. D.	7 xi. 28	20·9	53·0	44·0	44·9	−0·4
					42·9	
					43·0	
H. T. E.	7 xi. 28	20·9	47·8	40·6	41·3	+0·5
			48·0		41·6	
					40·6	

TABLE II. Comparison of the partial pressure of carbon dioxide in alveolar air and arterial blood (*contd.*).

Subject	Date	Oxygen in inspired air p.c.	Total CO_2 content arterial blood vol. p.c.	Arterial pCO_2 corrected mm. Hg	Alveolar pCO_2 mm. Hg	Δ mm. Hg
J. H. T.	7 xi. 28	20·9	49·25 49·50	41·0	41·0 41·0 40·3 40·2	−0·4
A. W. M.	9 xi. 28	20·9	50·2 50·0	41·3	43·4 44·1 44·1	+2·6
O. S. L.	9 xi. 28	20·9	50·2 50·15 50·3	42·2	41·9 41·7 41·3	−0·5
W. J. G.	9 xi. 28	20·9	49·3	41·0	42·1 39·3 40·1	−0·5
D. B. D.	26 ii. 29	14·1	52·8 52·65	40·7	41·2 40·5	+0·1
W. J. G.	12 iii. 29	14·1	50·4 50·45	40·9	39·8 37·6 39·6	−1·9
H. T. E.	13 iii. 29	13·3	47·3 47·2	39·5	38·3 38·3	−1·2
J. H. T.	20 iii. 29	9·6	44·2 44·6	29·5	30·4 29·0 28·3 27·8	−0·7
A. W. M.	22 iii. 29	20·9	49·4	43·7	43·5 43·5 43·8	−0·1
A. W. M.	22 iii. 29	10·4	47·6	34·6	36·2 36·1	+1·5
D. B. D.	27 iii. 29	20·9	53·0	43·0	42·8 42·0 44·3	0·0
D. B. D.	27 iii. 29	10·4	42·4 42·9	23·4	22·5 21·7 21·2	−1·6
A. V. B.	29 iii. 29	20·9	50·64	40·2	40·0 40·7 40·6	+0·2
A. V. B.	29 iii. 29	13·6	49·55	38·8	36·9 37·2 35·2 35·4	−2·4
A. V. B.	29 iii. 29	10·5	45·3	32·2	29·2 30·8 30·4	−2·1
A. V. B.	3 iv. 29	24·7	49·55 49·55	40·5	36·6 36·2	−4·1
A. V. B.*	3 iv. 29	20·9	42·90 42·85	26·8	23·5 20·8	−4·6

* Hyperpnœa, etc.

or two exceptions all experiments done while the subjects breathed outside air show a close correspondence (mean difference 0·2 mm.) of the pressures for CO_2 in alveolar air and arterial blood. This close approximation to equilibrium is presumably due to the rapid rate of diffusion of CO_2. The data further substantiate the usefulness of alveolar CO_2 determinations in normal subjects as a measure of the partial pressure of CO_2 in arterial blood. We do not wish to imply that the pressure of alveolar CO_2 is constant during the respiratory cycle. Our experiments indicate that the pressure of CO_2 found, during the phase when the sample of alveolar air is taken, is approximately equal to that of arterial blood.

In the experiments when low oxygen mixtures were breathed there appears to be an excess pressure of CO_2 in arterial blood (mean difference 1·0 mm.) over that present in alveolar air. More experiments should be done to determine whether or not this second order difference is a real one.

The partial pressure of oxygen.

According to Haldane, the partial pressure of oxygen, as estimated from samples of alveolar air, does not represent the average pressure of oxygen to which the mixed arterial blood is exposed in the capillaries of the lung. He writes as follows: "It is evident that the relation between blood supply and ventilation in individual groups of alveoli is not an even one. In some alveoli the oxygen runs down and CO_2 accumulates faster than in others. Hence in some the blood is less perfectly oxygenated....Hence the oxygen percentage of the mixed alveolar air becomes altogether deceptive as an index of the degree of oxygenation of the mixed arterial blood...." (7). The fan-like expansion of the lung described by Keith (8) supports this theory.

A solution of the problem may be sought from a comparison of pressures of oxygen found in alveolar air and in arterial blood under varying pressure heads of oxygen in the lung. Our experimental data on the subjects are included in Tables III and IV. The arterial oxygen

TABLE III. Oxygen dissociation curves.

			Observations on equilibrated blood				
Subject	Date	HbO_2 capacity vol. p.c.	pCO_2 mm. Hg	pO_2 mm. Hg	Total O_2 content	HbO_2 content	O_2 saturation
D. B. D.	26 ii. 29	17·92 17·87	39·2	58·8	16·51 16·35	16·28	91·0
			40·2	77·2	16·95 17·05	16·81	94·0
			40·2	98·6	17·80 17·70	17·45	97·5

TABLE III. Oxygen dissociation curves (*contd.*).

			Observations on equilibrated blood				
Subject	Date	HbO_2 capacity vol. p.c.	pCO_2 mm. Hg	pO_2 mm. Hg	Total O_2 content	HbO_2 content	O_2 saturation
W. J. G.	12 iii. 29	21·30 21·28	40·5	26·2	11·60	11·54	54·2
			41·8	51·3	18·59 18·52	18·43	86·6
			41·5	77·7	20·69	20·50	96·3
H. T. E.	13 iii. 29	21·03 21·10	39·3	23·5	9·70 10·23	9·64 10·17	46·1 48·4
			39·5	49·2	17·36 17·25	17·20	81·7
			38·1	73·8	19·60	19·42	92·3
J. H. T.	20 iii. 29	21·99 21·96	36·6	20·3	5·82	5·77	26·2
			36·5	46·7	18·60 18·60	18·48	84·1
			35·5	66·6	20·86	20·69	94·1
A. W. M.	22 iii. 29	21·03 20·98	43·7	55·6	18·79	18·65	88·8
			39·2	92·2	20·51	20·28	96·7
A. W. M.	22 iii. 29	21·05 21·05	35·8	30·2	12·56	12·48	59·3
			36·1	48·8	18·09	17·97	85·4
			57·0	75·2	19·40	19·21	91·3
			36·0	72·3	19·65	19·47	92·5
D. B. D.	27 iii. 29	18·63 18·57	40·6	37·4	13·94	13·85	74·5
			43·1	62·8	17·03	16·87	90·7
			41·5	75·6	17·62	17·43	93·7
D. B. D.	27 iii. 29	19·26 19·26	35·1	22·7	8·73	8·67	45·0
			35·4	29·0	14·61	14·54	75·5
			35·6	52·0	17·50 17·45	17·35	90·1
A. V. B.	29 iii. 29	21·30 21·40	40·4	54·7	19·87	19·75	92·6
			38·8	79·6	20·71	20·57	96·1
A. V. B.	29 iii. 29	21·92 22·10	35·9	48·9	19·45	19·35	87·9
			36·3	68·7	20·73	20·48	93·1
A. V. B.	29 iii. 29	22·42	30·6	30·3	15·70	15·62	69·6
			31·7	50·7	20·20	20·08	89·5
A. V. B.	3 iv. 29	20·35 20·35	42·0	121	20·12	19·82	97·4
			40·1	86·6	19·90 19·74	19·68 19·52	96·7 95·9
A. V. B.	3 iv. 29	20·62 20·62	22·3	129·4	20·62	20·30	98·4
			21·5	74·1	20·17	19·99	96·9

TABLE IV. Comparison of the partial pressure of oxygen in alveolar air and arterial blood.

Subject	Date	Pulse rate	Respiratory rate	Ventilation l per min.	Oxygen in inspired air p.c.	O_2 saturation arterial blood p.c.	Arterial pO_2 mm. Hg	Alveolar pO_2 mm. Hg	Δ mm. Hg.
D. B. D.	26 ii. 29	60	12	5·4	14·14	85·0	53	55	+2
W. J. G.	12 iii. 29	66	16	5·3	14·09	89·0	55	55 61 58	+3
H. T. E.	13 iii. 29	83	12	6·8	13·32	85·3	55	50 50	−5
J. H. T.	20 iii. 29	96	15	9·3	9·58	64·7	30	31 34 35 36	+4
A. W. M.	22 iii. 29	70	11	5·1	20·93	92·2	63	90 95 94	+30
A. W. M.	22 iii. 29	94	15	7·3	10·42	57·8	29	32 31 31	+2
D. B. D.	27 iii. 29	56	10	5·1	20·93	94·4	77	93 97 95	+18
D. B. D.	27 iii. 29	71	12	9·2	10·42	90·2	45	53 55 55	+9
A. V. B.	29 iii. 29	61	6	4·7	20·93	95·3	78	106 104 100 101	+25
A. V. B.	29 iii. 29	65	6	6·3	13·57	84·0	45	56 58 56 56	+12
A. V. B.	29 iii. 29	75	6	8·2	10·54	81·5	40	47 43 43	+4
A. V. B.	3 iv. 29	58	7	4·8	24·68	95·3	83	139 139	+56
A. V. B.*	3 iv. 29	—	30	—	20·93	96·0	75	136 138	+62

* Hypernœa produced by a respiratory rate of 30 per minute, beginning one minute before arterial puncture and continuing during puncture.

pressures range from 78 mm. down to 29 mm. and the oxygen saturations from 96 p.c. to 57·8 p.c. The alveolar oxygen pressures vary from 139 mm. to 31 mm. As a check on the accuracy of the arterial oxygen

pressure determinations Fig. 2. was constructed. This figure shows (1) the upper portion of the oxygen dissociation curves for the blood of

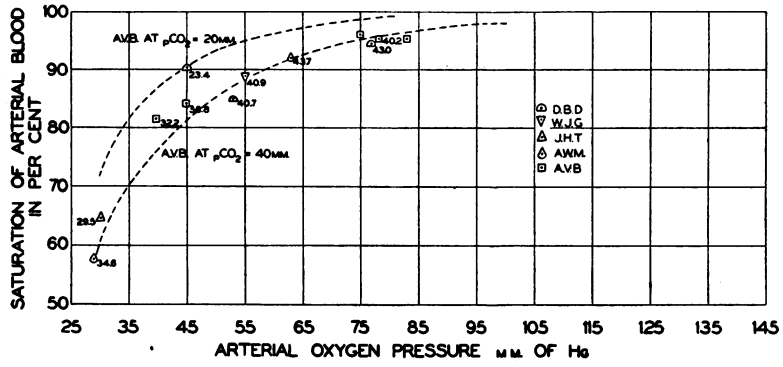

Fig. 2.

A. V. B. at carbon dioxide pressures of 20 mm. and 40 mm. and (2) points experimentally determined for the specimens of blood employed in this investigation. The coordinates of these points give the values of oxygen saturation and oxygen pressure. Beside each point the value of carbon dioxide pressure is printed. The source of the blood is also designated. Inspection of the figure shows that nearly all the points determined fall quite closely in their expected position with reference to normal oxygen dissociation curves. If the alveolar oxygen pressures are plotted against the corresponding arterial oxygen saturations and

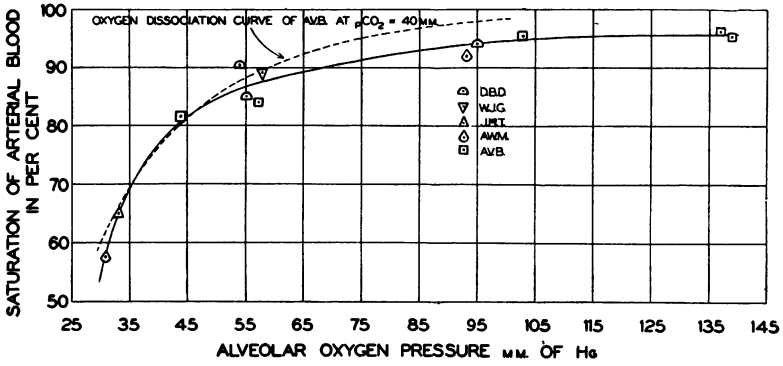

Fig. 3.

a free-hand curve drawn through the points, the result is seen in Fig. 3 in which the curve is compared with a normal oxygen dissociation curve.

The last two experiments recorded in Table IV were performed to see if a large pressure head of oxygen in the lung, produced by different methods, would exert an appreciable effect upon the saturation of arterial blood in the lung. In each instance the arterial saturation was not sensibly changed, even though the pressure difference between the lung and the blood withdrawn from the artery reached approximately 60 mm. These experiments suggest the question whether the saturation of arterial blood in the lung can be raised above about 96 p.c. In one of seven of our experiments during exercise performed two years ago(9) and in the experiments of Himwich and Barr(10), during heavy exercise, saturations of the blood with oxygen were found above 96 p.c. Small experimental errors may, however, account for such figures. In view of the present data, obtained after long practice in the use of the methods, these older determinations are at least open to question.

In the final experiment recorded in Table IV the subject breathed outside air, but increased his respiratory rate to 30 per minute, the breathing being kept as deep as possible at this rate. The alveolar CO_2 pressure fell to 22 mm., and meanwhile the O_2 pressure rose to 137 mm. The saturation of arterial blood was 96 p.c. In the subject A. V. B., therefore, oxygenation of the blood seems to be equally effective at his normal respiratory rate of six per minute, with an alveolar oxygen pressure of 103 mm., and when the rate of respiration is 30, and the oxygen pressure in the lung is raised to 137 mm.

The mechanism of oxygen exchange in the lung.

In the last column of Table IV we have recorded the difference in the pressure of oxygen between alveolar air and arterial blood. Reference to Fig. 4, in which these pressure differences are plotted against the oxygen saturations of the arterial blood, reveals a physiological relation of more than passing interest. Granting that the data are not numerous it is doubtful if a greater number of experiments would alter the general character of the results. In five experiments the pressure of oxygen in the lung exceeds that of the arterial blood by 4 mm. or less. In only one of these does the saturation of the blood exceed 85 p.c. Above this level of saturation the pressure of oxygen in the lungs rises rapidly, and the curve becomes very steep in the region of high oxygen saturation.

In seeking an explanation of these facts, we believe that the data concerning the movement of carbonic acid from blood to alveolar air should be carefully considered. As stated above, in every one of the present experiments a close approximation to equality of carbon dioxide

pressure in arterial blood and samples of alveolar air collected by our present method has been experimentally demonstrated, and this approximation is closest when the oxygen pressure of the inspired air is

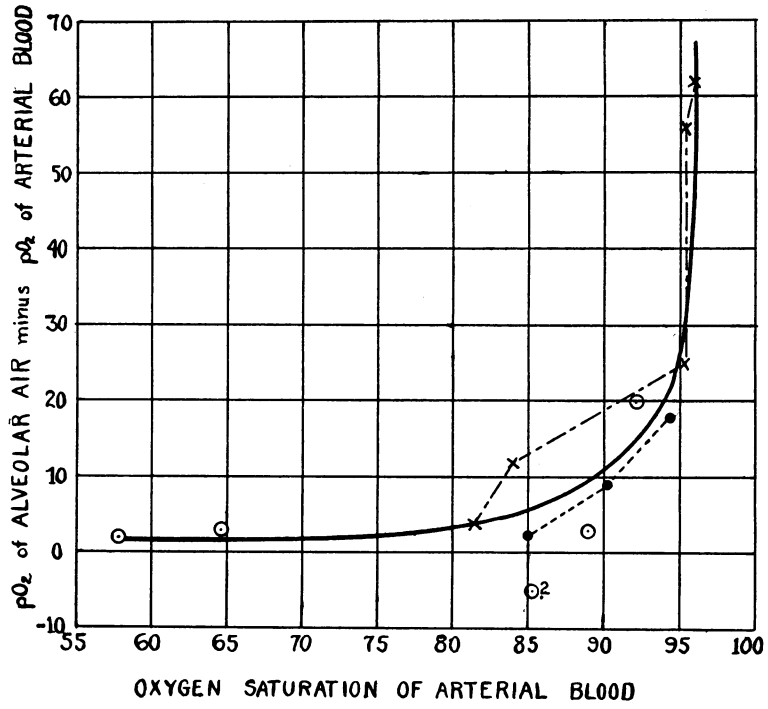

Fig. 4.

normal. Therefore, it seems probable that in general all the blood while passing through the lung reaches a condition at least very close to equilibrium with the alveolar air that we find by our method, in respect of carbon dioxide pressure, and that the number of alveoli in which this condition is not established must be very small indeed, or else that such alveoli must be temporarily supplied with very little venous blood.

The second observation to be accounted for is that when low oxygen mixtures are breathed the pressure of oxygen in arterial blood differs from that of the same alveolar air that is in carbon dioxide equilibrium with the same blood by no more than 3 or 4 mm. In view of the well-known fact that for equal pressure gradients oxygen is much less diffusible than carbonic acid, it seems probable, therefore, that under these circumstances the ventilation of the alveoli which are being supplied

with venous blood is also sufficient for the movement of oxygen. It is to be regarded as sufficient because under the conditions of these experiments the pressure of dissolved oxygen in the blood at the surface of the red cell when it leaves the lung must differ very little from its pressure in alveolar air. Then what is the explanation of the large differences between oxygen pressure in alveolar air and arterial blood when normal air is breathed? For this condition both rate and volume of breathing are slightly lower than when the inspired air is rarified. It cannot, therefore, be positively denied that the ventilation of certain portions of the lung may in this manner become inadequate. But it seems probable that no great change of this kind can occur, and our observation that forced breathing of normal air produces no measurable increase of the oxygen content of arterial blood seems to be an argument against the importance of such a factor.

Thus we are disposed to turn in another direction for an explanation of the facts and to suggest the following working hypothesis. In normal man the ventilation of the physiologically active lung alveoli and the nature of the diffusing membranes are such that the difference in oxygen pressure on the two sides of the capillary wall at the point where the blood passes out of contact with the inflowing oxygen is but a few millimetres. But the blood, because it is heterogeneous and because mixing in the interior of the red cells cannot be effective, requires perhaps several seconds to attain a state of equilibrium. Ordinarily the results of this condition are not always manifest. But when the hæmoglobin is nearly completely oxygenated the unoxygenated molecules will be, in general, deep in the interior of the red cells, and this may modify the rate of diffusion of oxygen at that point. In any case, however, equal differences in oxygenation are associated with small differences of oxygen pressure when oxygenation is not nearly complete, with large differences of oxygen pressure when oxygenation is nearly complete. Thus the final stages of oxygen diffusion, taking place after the blood is cut off from further oxygen supply, should produce little change in oxygen pressure at the surface of the red cell when the final stage of oxygenation is less than, say, 80–90 p.c., and large differences if the final stage lies above that amount. This conclusion, thus roughly stated, appears to be a consequence of the shape of the oxygen dissociation curve.

In order to fix our ideas we may consider the simple problem of the mixing of equal volumes of two portions of blood in which equilibrium has been reached under such conditions that there exists a difference of 5 p.c. in the saturation of the hæmoglobin. If these saturations are

55 p.c. and 60 p.c. the oxygen pressures before mixing, for the blood of A. V. B., will be 28·0 mm. and 30·4 mm. respectively, and after mixing the pressure will be 29·2 mm. But, if the saturations are 93 p.c. and 98 p.c., the pressures before mixing will be 65·6 mm. and 108·0 mm., and after mixing the pressure will be 79·0 mm.

We suggest, therefore, that when normal air is breathed the pressure of oxygen at the surface of the red cell as it leaves the lung is some 20 mm. greater, the oxygenation of the hæmoglobin some 0·2 p.c. less than a few seconds later when equilibrium has been established. No doubt other possible explanations may be devised, and we are far from believing that it is yet possible to give a complete explanation of the complex conditions. For example, there appears to be a real difference between the two individuals, A. V. B. and D. B. D., as shown by the broken line of Fig. 4. For this we have at present no explanation unless it be related to the low "diffusion coefficient" or to the slow breathing of the subject A. V. B. Our preference for the suggested hypothesis rests upon the belief that the present investigation yields at least a strong presumption that the diffusion process across the capillary wall of the lung is not the principal cause of the difference of 20 mm. or more between the oxygen pressures of alveolar air and samples of arterial blood when ordinary air is breathed, and the belief that a homogeneous chemical reaction between hæmoglobin and oxygen, if stirring is adequate, should reach a state of equilibrium at 96 p.c. oxygenation no less rapidly that at 50 p.c. oxygenation.

Summary.

Carbon dioxide partial pressures in alveolar air and arterial blood are found to be approximately equal, while oxygen partial pressures are lower in arterial blood than in alveolar air. This difference is larger when oxygen partial pressure is high, far smaller when oxygen partial pressure falls below 40–60 mm. Hg. It is suggested that these observations may be explained by the hypothesis that oxygen equilibrium is not attained until after passage through the lung capillaries.

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