JCI The Journal of Clinical Investigation

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J Clin Invest. 1924;1(1):25-45. https://doi.org/10.1172/JCI100003.

Research Article

BLOOD REACTION AND BLOOD GASES IN PNEUMONIA

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(Received for publication, July 3, 1924)

Peabody in 1912 (1) published a paper on the metabolism in pneumonia, in which he reviewed the previous literature and studied among other factors the blood gases and acid-base balance. Since Peabody, a number of other investigators have studied the blood gases and the question of the existence and importance of acidosis in pneumonia. The present paper is a report of observations on these subjects, in which recently developed methods have made possible the attainment of more complete results and apparently have justified the drawing of deductions more definite in some respects than those attainable from previous data.

Peabody stated that "the high excretion of ammonia and the low excretion of sodium chloride are two of the most characteristic features of the urine during fever. The diminution of the carbon dioxide of the blood is apparently a constant accompaniment of fever." These results indicated a shift of some degree towards acidosis in the acid-base balance, but whether it was sufficient to be of clinical significance was at the time uncertain. Peabody states, "The evidence points against the theory that the retention of sodium chloride in fever depends on acidosis. . . . . In diabetic acidosis and in experimental poisoning by mineral acids there is not a retention of bases as there is in pneumonia." In the gases of the venous blood much greater variations in oxygen than in carbon dioxide were found.

Palmer (2) found that, although considerable amounts of a very weak unknown organic acid could be titrated in the urine in certain severe cases of pneumonia, the alkali reserve of the venous blood plasma, as determined by the CO_2 capacity method of Van Slyke and Cullen (3) was uniformly normal or nearly so. Acidosis of metabolic origin was consequently excluded.

Stillman, Van Slyke, Cullen, and Fitz, (4), in a series of acidosis cases of which the rest were diabetic, published protocols on one case with a greatly diminished plasma CO_2 capacity in acute nephritis *after* pneumonia. The alkali deficit, which was accompanied by an almost complete cessation of ammonia excretion (unpublished data) was apparently due to the pneumonic nephritis. It disappeared as the kidney function and ammonia excretion improved.

Stadie (5) and Stadie and Van Slyke (6) studied the oxygen content and capacity of the blood and the carbon dioxide content and capacity of the plasma in both venous and arterial blood of pneumonia patients. They confirmed Palmer's observation of normal alkali reserve as indicated by the CO_2 capacities. From the gas contents of the blood they observed that "even when pulmonary conditions in pneumonia become so involved that the arterial blood is incompletely oxygenated, the arterial and venous carbon dioxide values are not increased above the usual normal levels." There was no evidence of pulmonary CO_2 retention.

The data up to this point (1920) indicate that an acidosis of metabolic origin, such as would result in an alkali deficit in the body, is a rarity in pneumonia. Because the data do not include either determinations of the CO_2 tension in the blood, or of the pH from which the CO_2 tensions can be estimated, they do not exclude the possibility of a CO_2 acidosis, due to hindrance in the evolution of CO_2 from the pathologically involved lungs) although the combined determinations of CO_2 content and CO_2 capacity by Stadie and Van Slyke made a CO_2 retention appear improbable.

Barach, Means and Woodwell (7) in 1922 estimated the pH of the arterial and venous blood in pneumonia by interpolation on the CO_2 absorption curve, corrected for the observed oxygen unsaturation. They found the alkali reserve normal or slightly subnormal. In 3 of 10 patients, however, a low pH was observed which rose to normal after the crisis, the alkali reserve showing appreciable rise at the same time, although it had not been seriously lowered. The results indicated the possibility of a tendency towards both alkali deficit and lowered pH in some cases, and the authors suggested the use of alkali therapy in such cases.

Binger, Hastings and Neill (8), however, found that the continued use of even moderate amounts of bicarbonate in a case of pneumonia with somewhat diminished salt excreting power led to a dangerous alkalosis and edema, due to apparent inability to excrete the alkali.

It seemed advisable to make further observations on the blood changes in pneumonia patients, the more so because at the time of the work of Barach, Means and Woodwell, the colorimetric pH method for blood was not available, and their pH values were determined by a method which makes such demands on technique that a certain proportion of irregular results is difficult to avoid. Thirty observations of the pH and CO_2 content, and 22 observations of the oxygen content and oxygen capacity of the arterial blood have accordingly been made on 16 patients.

In our observations the gases were determined on the Van Slyke (9) manometric apparatus. The pH's were determined colorimetrically by Cullen's method (10), in all cases, electrometrically in 12 cases, and by calculation from the CO_2 absorption curves in 2 cases. All precautions to collect and preserve the blood without change in its constituents were observed (Austin, Cullen, et al., 11).

THE pH DETERMINATIONS

Electrometric and colorimetric values.—In table 1 are given the results of the colorimetric determinations of pH at 20°C. and the

TABLE 1

The difference between the electrometric pH at 38° and the colorimetric pH at 20° of the arterial blood of pneumonia patients

No.	pH		ΔpH	No.	pH		ΔpH
	Col. 20°	Elect. 38°			Col. 20°	Elec. 38°	
19	7.72	7.42	0.30	25	7.77	7.48	0.29
20	7.72	7.42	0.30	26	7.72	7.43	0.29
21	7.71	7.49	0.22	27	7.70	7.43	0.27
22	7.63	7.40	0.23	28	7.76	7.46	0.30
23	7.66	7.40	0.26	29	7.82	7.50	0.32
24	7.55	7.32	0.23	30	7.59	7.35	0.24

Average ΔpH = 0.27.
Average deviation = 0.03.
Maximum deviation = 0.05.

electrometric determinations made on the same blood at 38°C. The average difference, ΔpH, between these determinations is 0.27 pH. The difference for the blood of normal individuals was found by Cullen to be 0.22 pH. This value has been confirmed in this laboratory. The reason for the difference between the ΔpH found for normals and for pneumonia patients is unknown. The formula we have used in the calculation of the blood pH at 38° from the colorimetric pH at 20° is

$$pH_{38°C.} = pH_{20°C.} - 0.27$$

Since there were rather wide differences in the temperatures of the patients at the time the blood analyses were made, a correction was

applied to convert the pH at 38° to its corresponding value at the temperature of the patient. The temperature coefficient for the pH of blood is not well established. Direct determinations of the electrometric pH of serum at 38° and 20° have indicated that the ΔpH per 1°C. is approximately 0.01. This value has been used to correct the pH to the temperature of the blood as drawn.

Recent determinations made in this laboratory indicate that by making the colorimetric pH determinations at the temperature of the patient, the necessity for temperature correction is avoided (Hastings and Sendroy, paper in press) but the technique for such determinations had not been developed when the present work was done.

TABLE 2

A comparison of electrometric, colorimetric and calculated pH's

	Electrometric pH	Colorimetric pH	Calculated pH
Normals			
1	—	7.38	7.40
2	7.39	7.41	7.38
Pneumonia patients			
9	—	7.44	7.44
10	7.42	7.47	7.47

pH values calculated from the carbon dioxide absorption curve. Since the pH results of Barach, Means and Woodwell (7) were all determined by calculation from the carbon dioxide content and the carbon dioxide tension, four experiments were performed in order to compare values obtained thus with those obtained by direct electrometric or colorimetric determinations.

Two of the experiments were on the venous blood of normal individuals and two were on the arterial blood of pneumonia patients. The comparisons between the pH values obtained by direct determination and by calculation are given in table 2. The agreement is within the limits of error of the method employed. Since difficulty has been experienced in obtaining satisfactory agreement by these two methods, certain points which contributed to the success of our experiments may be mentioned.

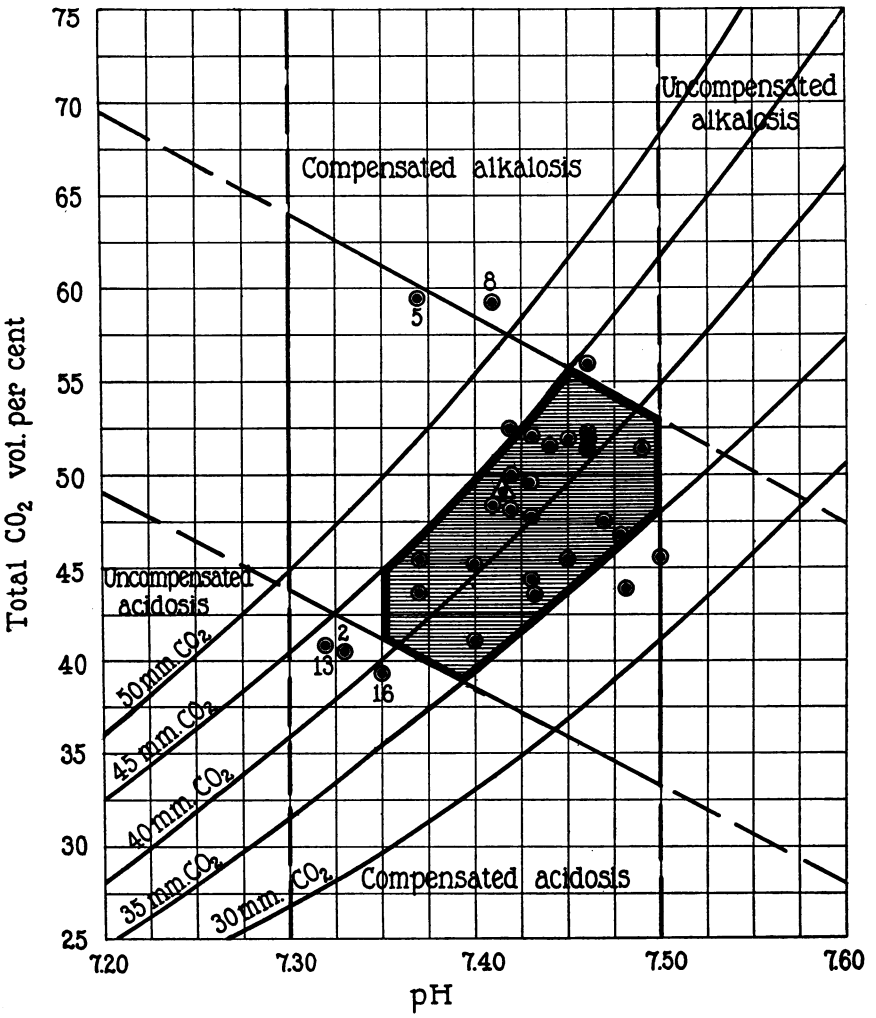

FIG. 1. ACID-BASE DETERMINATIONS ON PNEUMONIA PATIENTS ARE PLOTTED WITH pH AS ABSCISSAE AND CO_2 CONTENT AS ORDINATES

Constant CO_2 tension lines run diagonally across the chart. The shaded area represents the revised normal acid-base area of arterial blood. The point ▲ is the average of a number of normal arterial acid-base values obtained by Barr and his collaborators.

TABL

A summary of the hydrogen ion concentrations and

Determination number	Case number	History number	Date	Organism	Area of pulmonary involvement at time blood was drawn	Day of disease blood was drawn	Day of disease temperature became normal	Day of disease patient died	Treatment
1	1	4522	4-27-22	Pneumococcus. Type III	Consolidation of left lower lobe, and whole right lung	9	11		Oxygen chamber
2	2	4597	10-17-22	Pneumococcus. Type I	Consolidation of right middle lobe. Resolution of right lower lobe	8			
3			10-23-22		Consolidation of right middle lobe. Resolution of right lower lobe	14			
4			10-24-22		Consolidation of right middle lobe. Resolution of right lower lobe	15		18	
5	3	4619	11- 5-22	Pneumococcus. Group IV	Consolidation of right middle and lower lobes	4	6		
6			11-10-22		Consolidation of right middle and lower lobes	9			
7	4	4623	11-10-22	Pneumococcus. Group IV	Consolidation of left upper lobe. Diffuse bronchitic râles	5			
8			11-13-22		Consolidation of left upper lobe. Diffuse bronchitic râles	8			Oxygen chamber
9			11-14-22		Consolidation the same. Diffuse râles less	9			Oxygen chamber
10			11-15-12		Consolidation the same. Diffuse râles less	10	10	12	
11	5	4630	11-15-22	Pneumococcus. Type III	Consolidation of left upper lobe. Râles left lower and right upper	4			
12			11-16-22		Consolidation of left upper and lower, and right upper lobes	5	5		Oxygen chamber
13			11-17-22		Consolidation of left upper and lower, and right upper lobes	6		6	Oxygen chamber
14	6	4648	12-12-22	Pneumococcus. Type III	Consolidation of right middle and lower lobes	5	12		
15	7	4651	12-18-22	Pneumococcus. Type II	Consolidation of left upper and lower. Diffuse bronchitic râles	5		6	Oxygen by nasal catheter
16	8	4657	12-29-22	Pneumococcus. Type I	Consolidation of left upper and lower, and right upper lobes	6			Antipneumococcus serum

E 3

gas analyses of the arterial blood in pneumonia

Remarks	Rectal temperature	pH at body temperature	Arterial blood analyses									
			CO_2 content				CO_2 tension	O_2 content		O_2 capacity		Per cent saturation
			Whole blood		Plasma							
	°C.		mM	*vol. per cent*	mM	*vol. per cent*	*mm.* Hg	mM	*vol. per cent*	mM	*vol. per cent*	
Respirations 58 per minute.	38.9	7.45	25.0	56.0	—	—	45.2	6.8	15.2	7.3	16.4	92.7
Arteriosclerosis marked	38.8	7.45	23.2	52.0	—	—	43.8	6.7	15.1	8.2	18.4	82.1
Patient had received 600 cc. Type I antipneumococcus Serum before bleeding.	38.5	7.46	23.3	52.3	—	—	42.7	—	—	—	—	
Pneumococcus meningitis	38.1	7.33	17.9	40.4	—	—	42.9	6.7	15.1	8.3	18.6	81.2
	38.7	7.45	20.4	45.7	—	—	38.1	—	—	—	—	
	37.7	7.37	19.5	43.7	—	—	42.3	—	—	—	—	
	39.6	7.42	19.9	44.6	—	—	39.6	6.8	15.2	—	—	
	38.8	7.43	19.4	43.6	—	—	38.2	6.6	14.7	7.8	17.5	84.0
	38.1	7.41	21.5	48.3	—	—	43.1	7.1	15.9	—	—	
Patient died suddenly when afebrile and apparently convalescent	37.3	7.46	23.2	52.0	—	—	41.2	7.0	15.6	7.2	16.2	96.4
	39.6	7.47	20.6	46.2	—	—	45.9	7.6	17.0	7.8	17.5	97.2
	39.0	7.36	20.4	45.7	—	—	46.2	5.8	13.1	8.6	19.2	68.2
Temperature normal for short interval only on 5th day of disease	37.8	7.37	26.6	59.7	—	—	58.2	7.2	16.2	8.2	18.4	88.1
Sterile pleural effusion. Right hydropneumothorax	39.7	7.42	21.4	47.9	—	—	44.0	7.7	17.2	8.9	19.9	86.5
	39.3	7.41	22.3	50.0	—	—	46.8	7.0	15.6	9.2	20.6	75.8
Patient had received 400 cc. Type I antipneumococcus serum before bleeding	39.6	7.46	21.1	47.4	—	—	37.2	5.9	13.3	6.6	14.7	9. 5

The blood was treated with one per cent neutral sodium fluoride as well as potassium oxalate which, as Lovatt Evans (12) has shown, inhibits acid formation during the period of saturation. Further,

TABLE 3

Determination number	Case number	History number	Date	Organism	Area of pulmonary involvement at time blood was drawn	Day of disease blood was drawn	Day of disease temperature became normal	Day of disease patient died	Treatment
17			12-30-22		Consolidation of left upper and lower, and right upper lobes	7	12		Serum. Oxygen by nasal catheter
18	9	4671	1-19-23	Pneumococcus. Group IV	Consolidation of right middle and lower lobes	5	6		
19			1-23-23		Consolidation of right middle and lower lobes	7	6		
20	10	4670	1-20-23	Pneumococcus. Group IV	Consolidation of right upper lobe	4	6		
21	11	4691	2- 7-23	Pneumoccus. Group IV. Streptococcus hemolyticus	Consolidation right lower lobe. Right pleural effusion. Râles over left lower lobe	9		26	
22	12	4690	2- 7-23	Pneumococcus. Group IV	Consolidation of right lower lobe	5	6		
23	13	4708	3- 1-23	Pneumococcus. Type II	Consolidation of left lower lobe	7	11		
24			3- 6-23		Consolidation of left lower lobe	12			
25	14	4710	3- 2-23	Pneumococcus. Group IV	Consolidation of right middle and lower lobes	4	8		
26			3- 7-23		Signs of resolution over entire right lung	9			
27	15	4715	3- 6-23	Pneumococcus. Group IV.	Consolidation of right middle and lower lobes	3	9		
28			3- 7-23		Consolidation of right middle and lower lobes	4			
29			3-14-23		Resolution of right middle and lower lobes	11			
30	16	4729	3-27-23	Pneumococcus. Group IV	Consolidation of whole right lung and left lower lobe. Diffuse bronchitic râles.	7		7	Oxygen by nasal catheter

ε indicates electrometric determination.

the CO_2 saturation curves were determined on both the reduced and oxygenated blood, thereby permitting the proper correction to be made for the unsaturation of the blood. Finally, the pK′ used in

calculating the pH values was obtained by taking into consideration the pH, the concentration of total hemoglobin, and the degree of saturation of the blood (fig. 6 *b* of Van Slyke, Wu, McLean, 13).

Continued

Remarks	Rectal temperature	pH at body temperature	Arterial blood analyses									
			CO_2 content				CO_2 tension	O_2 content		O_2 capacity		Per cent saturation
			Whole blood		Plasma							
	°C.		mM	*vol. per cent*	mM	*vol. per cent*	*mm.* Hg	mM	*vol. per cent*	mM	*vol. per cent*	
Oxygen discontinued during bleeding	37.5	7.41	26.5	59.3	—	—	50.8	4.5	10.0	6.9	15.5	64.6
	39.3	7.44	23.0	51.6	25.9	58.0	42.8	5.9	13.3	6.3	14.1	94.4
	37.2	7.43e	23.5	52.8	27.5	61.7	45.8	7.1	15.9	7.3	16.4	97.0
	39.1	7.42e	21.4	48.1	26.3	59.0	45.3	8.7	19.5	9.5	21.3	91.6
Streptococcus hemolyticus. Bronchopneumonia. Septicemia (Streptococcus hemolyticus). Death	39.6	7.48e	23.9	51.4	27.7	62.0	53.5	7.7	17.3	8.6	19.2	90.2
	38.2	7.40e	20.2	45.2	25.2	56.4	41.5	—	—	—	—	
	40.2	7.39e	18.4	41.2	21.7	48.7	38.7	—	—	—	—	
	37.2	7.33e	18.2	40.8	—	—	40.8	5.1	11.4	5.7	12.8	89.7
Spread to right upper lobe occurred	39.7	7.47e	19.5	43.6	23.4	52.4	35.3	7.9	17.7	8.2	18.4	96.2
	37.5	7.43e	22.2	49.7	—	—	42.5	7.4	16.6	8.1	18.1	91.8
Urinary retention	39.6	7.42e	23.3	52.2	—	—	45.2	4.7	10.6	7.0	15.6	68.0
	40.2	7.45e	23.2	52.0	—	—	43.2	5.8	13.0	7.2	16.1	80.8
	37.4	7.51e	20.4	45.7	24.5	55.1	32.6	6.2	13.8	7.4	16.5	83.7
Complicated by marked albuminuria with urea retention	40.0	7.34e	17.5	39.3	—	—	41.8	—	—	—	—	

The protocols of these experiments and the figures showing the results are given in tables 5 and 6, and figures 3 and 4.

RESULTS

The experimental results are collected in table 3 and are graphically shown in the acid-base diagram, figure 1.

The pH of the blood. Among the 30 observations on pneumonia patients there was no pH lower than 7.30 or higher than 7.50. (Where both colorimetric and electrometric values were available, the latter were taken as the more accurate.) Since the pH limits of 7.30 and 7.50 for normals have been confirmed in this laboratory, it may be said that no pneumonia patient studied by us had a pH definitely outside the normal limits. Certainly there was no case of uncompensated acidosis among them.

The CO_2 content of the blood. Values of the CO_2 content of arterial blood of normal resting individuals have been determined by Barr (14). The average of these determinations is 22 millimols per liter (49.4 volumes per cent).

The CO_2 contents of the blood of the pneumonia patients are about equally distributed on either side of the mean line. Except for three high and three low values they lie within the original limits of the normal area outlined by Van Slyke (15). Some of these cases require special mention. Of the low ones, No. 2 had a pneumococcus meningitis and died three days later; No. 16 was complicated by nephritis and died the day the observation was made; No. 13 was afebrile at the time of the analyses. Of the high points, No. 5 was moribund and died on the day of the observation; No. 8 had been having oxygen therapy by nasal catheter. The fact that this was discontinued during the bleeding may have contributed to the marked unsaturation of the blood.

It is concluded from determinations of CO_2 content taken in conjunction with those of the pH, that an acidosis, either compensated or uncompensated, is rarely, if ever, encountered in pneumonia uncomplicated by other abnormal conditions.

The CO_2 tension. Twenty-five of the thirty arterial CO_2 tensions, calculated from the serum pH and CO_2 content, were within the 35 to 45 mm. iso-pressure lines of figure 1. The CO_2 tension was calculated from the CO_2 content and plasma pH by formula 5 in table 4 of Austin et al. (11), viz.:

$$p_{CO_2} = \frac{[CO_2]}{0.0587\alpha_{CO_2}(1 + 10^{pH - pK'})}$$

$[CO_2]$ = volume per cent total CO_2 in whole blood.

α_{CO_2} = solubility coefficient of CO_2 in whole blood.

pK' = pK' of Hasselbalch's equation for whole blood, $pH = pK' + \log\frac{[BHCO_3]}{[H_2CO_3]}$

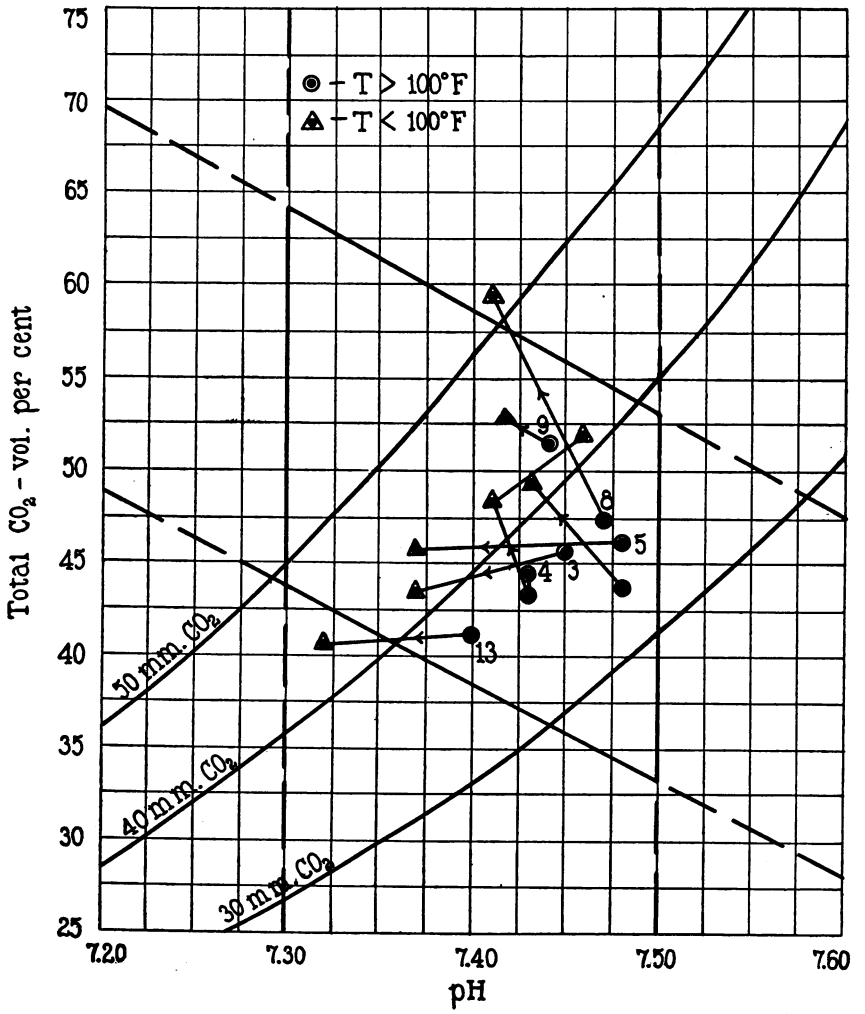

FIG. 2. ACID-BASE DETERMINATIONS OBTAINED ON PNEUMONIA PATIENTS DURING AND AFTER THE FEBRILE PERIOD

The value of α_{CO_2} was estimated to be proportional to the water content of the blood (13). The water content, for blood of varying hemoglobin content, is calculated from Equation 30 of Van Slyke, Wu, and McLean, (13) as

$$\text{cc. } H_2O \text{ per cc. blood} = 0.94 - 0.0067\ Hb$$

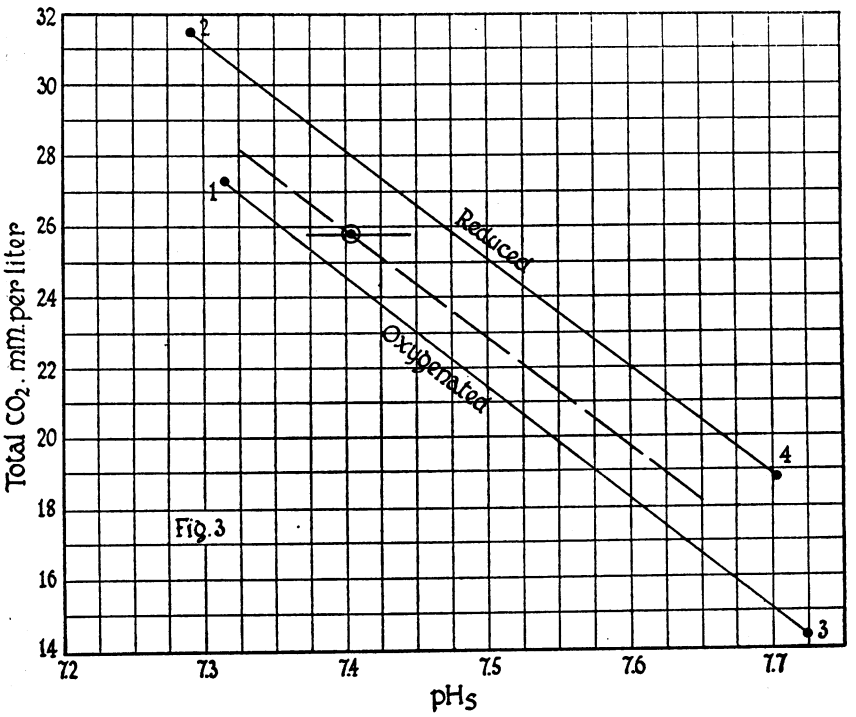

FIG. 3. JANUARY 9, 1923. CO_2 ABSORPTION CURVES OF OXYGENATED AND REDUCED BLOOD OF A NORMAL INDIVIDUAL PLOTTED WITH pH AS ABSCISSAE AND CO_2 CONTENT AS ORDINATES

The point within the ⊙ represents the acid-base balance of the venous blood as drawn determined by the CO_2 content and degree of unsaturation. The pH thus estimated is 7.40; as determined colorimetrically, 7.38.

Hb = hemoglobin content of blood in volume per cent of oxygen capacity. (The constants in their equation are here altered to change the H_2O from terms of weight to those of volume, and the Hb from terms of millimols to those of volumes per cent oxygen capacity.)

Since the solubility coefficient of CO_2 for water is 0.555, the coefficient for blood is estimated as

$$\alpha_{CO_2} = 0.555\ (0.94 - 0.0067\ Hb)$$

The pK' value for whole blood, as shown by Warburg (16) by Van Slyke, Wu, and McLean (13) and by Peters, Bulger, and Eisemann

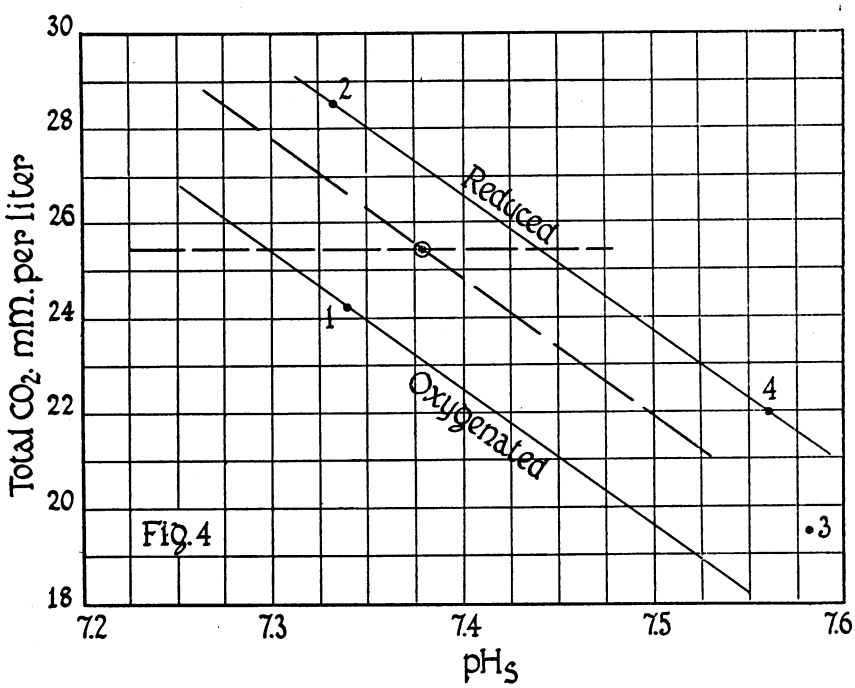

FIG. 4. JANUARY 25, 1923. CO_2 ABSORPTION CURVES OF OXYGENATED AND REDUCED BLOOD OF A NORMAL INDIVIDUAL PLOTTED WITH pH AS ABSCISSAE AND CO_2 CONTENT AS ORDINATES

The point within the ⊙ represents the acid-base balance of the venous blood as drawn determined by the CO_2 content and degree of unsaturation. The pH thus estimated is 7.38; as determined colorimetrically 7.41; electrometrically, 7.39.

(17), is greater for whole blood than for plasma by an amount, $\Delta pK'$, dependent on the oxygen saturation, pH, and hemoglobin content of the blood. Hence, for whole blood,

$$pK' = pK'_s + \Delta pK',$$

where

$$pK'_s = pK' \text{ for serum} = 6.12$$

$\Delta pK'$ is estimated from figure 6 *b* of Van Slyke, Wu, and McLean, with an added correction for oxygen unsaturation.

With the above values for α_{CO_2} and pK′ in whole blood, the formula relating p_{CO_2} to $[CO_2]$ and pH_s becomes,

$$p_{CO_2} = \frac{[CO_2]}{0.0326\,(0.94 - 0.0067\,Hb)\left(1 + 10^{pH - pK'_s - \Delta pK'}\right)}.$$

TABLE 4

The CO_2 tension of the arterial blood during and after the febrile period

Case No.	During febrile period			After febrile period.		
	CO_2 tension	Per cent saturation with oxygen	Temperature.	CO_2 tension.	Per cent saturation with oxygen	Temperature.
	mm. Hg.		°C.	*mm.* Hg.		°C.
3	38.1	—	38.7	42.3	—	37.7
4	39.6	—	39.6	41.2	96.0	37.3
	38.2	84.0	38.8			
	43.1	—	38.1			
5	45.9	97.2	39.6	58.2	88.1	37.8
	46.2	68.2	39.0			
8	37.2	90.5	39.6	50.8	64.6	37.5
9	42.8	94.4	39.3	45.8	97.0	37.2
13	38.7	—	40.2	40.8	89.7	37.2
14	35.3	96.2	39.7	42.5	91.8	37.5
15	45.2	68.0	39.6	32.6	83.7	37.4
	43.2	81.0	40.2			

The progress of the CO_2 tension during the course of the disease is shown in eight cases in which data during and after the febrile period were obtained. These results are shown in table 4 and figure 2. It is seen that there is a lower CO_2 tension during the febrile period than after. Whether this is the result of the increased temperature alone or whether it had its origin in the local pulmonary lesion it is impossible to say. Haggard (18) found that elevation of the body temperature by immersion in hot baths was sufficient to lower the alveolar CO_2 tension and reduce the dissolved CO_2 in the blood; but Fridericia (19) who studied the alveolar CO_2 tension in febrile diseases, believes

that increased temperature alone is insufficient to account for the lower tension. That anoxemia *per se* is not the causal agent in the lowered arterial CO_2 tension is suggested by the fact that the unsaturation of the arterial blood in some cases did not seem to be less in the period of low tension than in the subsequent afebrile period.

That *the respiratory mechanism is unimpaired in its ability to maintain a normal CO_2 tension and blood reaction* is evidenced by the fact

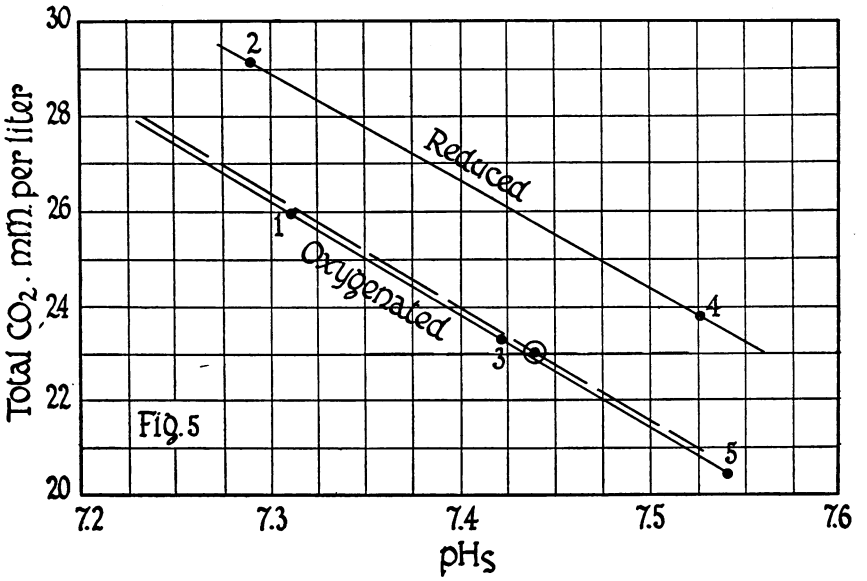

FIG. 5. JANUARY 19, 1923. CO_2 ABSORPTION CURVES OF OXYGENATED AND REDUCED BLOOD OF A PNEUMONIA PATIENT PLOTTED WITH pH AS ABSCISSAE AND CO_2 CONTENT AS ORDINATES

The point within the ⊙ represents the acid-base balance of the arterial blood as drawn determined by the CO_2 content and degree of unsaturation. The pH thus estimated is 7.44; as determined colorimetrically, 7.44.

that in only 2 of the 30 analyses was the arterial CO_2 tension found above 45 mm., and in no case was the pH below 7.30. There was no tendency towards CO_2 acidosis.

The effect of the lung changes in pneumonia on the *oxygenation of the arterial blood on the other hand is significant.* Of the 10 cases in which the oxygen saturation of the arterial blood was determined,

8 showed on one or more occasions arterial saturation below 90 per cent, which figure is probably lower than occurs in any normal person at rest at sea level. In 6 cases arterial saturation below 85 per cent was noted, a level of arterial saturation at which symptoms of mountain sickness may begin in normal individuals who are transferred to high altitudes (20).

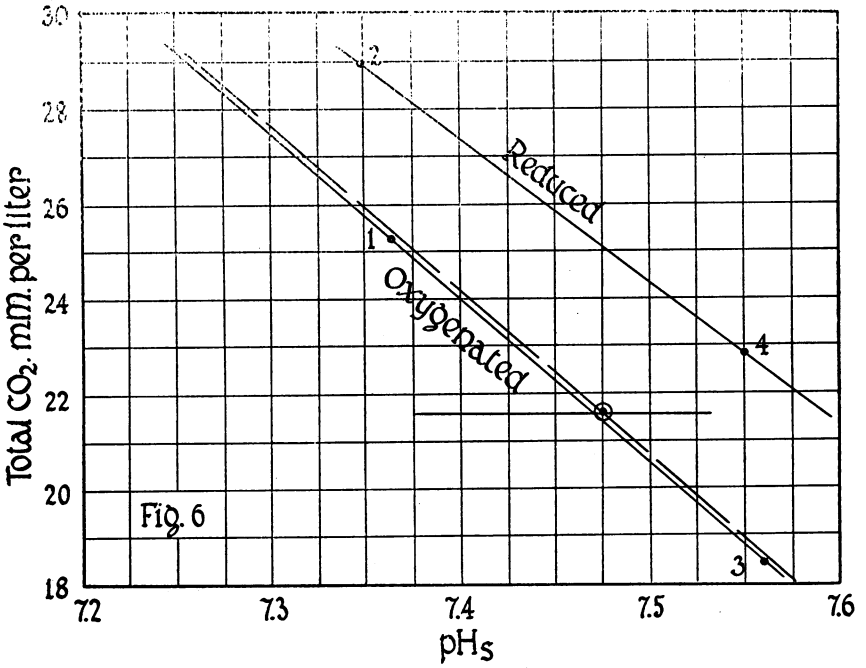

FIG. 6. JANUARY 20, 1923. CO_2 ABSORPTION CURVES OF OXYGENATED AND REDUCED BLOOD OF A PNEUMONIA PATIENT, PLOTTED WITH pH AS ABSCISSAE AND CO_2 CONTENT AS ORDINATES

The point within the ⊙ represents the acid-base balance of the arterial blood as drawn, determined by the CO_2 content and degree of unsaturation. The pH thus estimated is 7.47; as determined colorimetrically, 7.47; electrometrically, 7.42.

Various known facts indicate that the oxygenation of the blood in the lungs is much more susceptible to failure than is the removal of CO_2. Krogh (21) has shown that CO_2 diffuses through animal membranes 30 times faster than O_2. Henderson (22) has calculated that

TABLE 5

A comparison of the pH of normal human blood determined directly and calculated from the CO_2 saturation curves. Blood treated with 0.4 per cent $K_2C_2O_4$ + 0.1 per cent NaF

Normal, No. 1.
Date: January 9, 1924.
$pK' = 6.12 + \Delta pK'$
$H_2CO_3 = 0.0271\ pCO_2$

Analysis of blood after saturation with definite CO_2 and O_2 tensions.

No.	pO_2	HbO_2	pCO_2	H_2CO_3	Total CO_2	$BHCO_3$	$\log \frac{BHCO_3}{H_2CO_3}$	pK′	pH
	mm.	*mM.*	*mm.*	*mM.*	*mM.*	*mM.*			
1	(135)	9.18	66.1	1.79	27.28	25.49	1.154	6.165	7.319
2	(0)	0.11	79.5	2.15	31.50	29.35	1.135	6.154	7.289
3	(135)	9.23	15.0	0.41	14.53	14.12	1.537	6.190	7.727
4	(0)	0.15	20.0	0.54	18.88	18.34	1.531	6.173	7.704

Analysis of blood as drawn

Total $Hb + HbO_2$	HbO_2	$\frac{HbO_2}{Hb + HbO_2}$	Total CO_2	pH
mM.	*mM.*	*per cent*	*mM.*	
(9.40)	5.97	63.0	25.76	7.40—calculated 7.38—colorimetric

Normal, No. 2.
Date: January 25, 1923.
$pK' = 6.12 + \Delta pK'$
$H_2CO_3 = 0.0270\ pCO_2$

Analysis of blood after saturation with definite CO_2 and O_2 tensions

No.	pO_2	HbO_2	pCO_2	H_2CO_3	Total CO_2	$BHCO_3$	$\log \frac{BHCO_3}{H_2CO_3}$	pK′	pH
	mm.	*mM.*	*mm.*	*mM.*	*mM.*	*mM.*			
1	(135)	9.34	56.2	1.52	24.27	22.75	1.175	6.165	7.340
2	(0)	0.38	65.9	1.78	28.53	26.75	1.177	6.155	7.332
3	(135)	9.38	27.4	0.74	19.48	18.74	1.404	6.178	7.582*
4	(0)	0.49	32.0	0.86	22.00	21.14	1.392	6.168	7.560

Analysis of blood as drawn

Total $Hb + HbO_2$	HbO_2	$\frac{HbO_2}{Hb + HbO_2}$	Total CO_2	pH
mM.	*mM.*	*per cent*	*mM.*	
9.62	4.39	46	25.59	7.38—calculated 7.39—electrometric 7.41—colorimetric

*Not used in plotting oxygenated curve.

TABLE 6

A comparison of the pH of pneumonia patients' blood determined directly and calculated from the CO_2 titration curves

Patient, No. 9.
Date: January 19, 1923.
$pK' = 6.12 + \Delta pK'$
$H_2CO_3 = 0.0280\ pCO_2$

Analysis of blood after saturation with definite CO_2 and O_2 tensions

No.	pO_2	HbO_2	pCO_2	H_2CO_3	Total CO_2	$BHCO_3$	$\log \frac{BHCO_3}{H_2CO_3}$	pK′	pH
	mm.	*m*M.	*mm.*	*m*M.	*m*M.	*m*M.			
1	(135)	6.60	57.8	1.62	25.88	24.26	1.175	6.147	7.312
2	(0)	0.57	68.5	1.92	29.17	27.25	1.152	6.141	7.293
3	(135)	6.87	42.1	1.18	23.27	22.09	1.272	6.150	7.422
4	(0)	0.45	33.9	0.95	23.76	22.81	1.380	6.146	7.526
5	(135)	6.73	28.6	0.80	20.32	19.52	1.388	6.156	7.544

Analysis of blood as drawn

Total Hb + HbO_2	HbO_2	Per cent saturation	Total CO_2	pH
*m*M.	*m*M.		*m*M.	
6.90	5.92	94.4	23.02	7.44—calculated 7.44—colorimetric

Patient No. 10.
Date: January 20, 1923.
$pK' = 6.12 + \Delta pK'$
$H_2CO_3 = 0.0270\ pCO_2$

Analysis of blood after saturation with definite CO_2 and O_2 tensions

No.	pO_2	HbO_2	pCO_2	H_2CO_3	Total CO_2	$BHCO_3$	$\log \frac{BHCO_3}{H_2CO_3}$	pK′	pH
	mm.	*m*M.	*mm.*	*m*M.	*m*M.	*m*M.			
1	(135)		56.8	1.53	25.31	23.78	1.191	6.168	7.359
2	(0)	0.25	65.1	1.76	28.88	27.12	1.188	6.158	7.346
3	(135)	9.19	27.4	0.74	18.45	17.71	1.380	6.178	7.558
4	(0)	0.51	33.3	0.90	22.72	21.82	1.385	6.167	7.552

Analysis of blood as drawn

Total Hb + HbO_2	HbO_2	Per cent saturation	Total CO_2	pH
*m*M.	*m*M.		*m*M.	
9.51	8.69	91.4	21.45	7.47—calculated 7.42—electrometric 7.47—colorimetric

the blood attains practically the CO_2 tension of the alveolar air by the time it has passed through about half the length of a lung capillary, while the arterial blood after traversing completely the pulmonary capillaries still has an oxygen tension about 25 millimeters lower than the alveolar air. It has furthermore been shown by various observers that the respiratory mechanism shows a relatively weak response to oxygen lack as compared with that to carbon dioxide excess. Accumulation of CO_2 may cause a man to increase his ventilation per minute by 1000 per cent, while the maximum response to oxygen lack is about 50 per cent. Furthermore, if part of the lung area is cut off from air, it is possible by over ventilation of the remainder to keep the CO_2 tension of the mixed arterial blood down to normal; but since only 20 volumes per cent of oxygen can be held by the average blood, it is not possible to overcharge one portion of the pulmonary blood with oxygen in order to compensate for under oxygenation in another. These considerations would lead to the conclusion that, in any individual breathing the ordinary atmosphere, respiratory hindrance must result in serious anoxemia long before CO_2 acidosis has become at all significant.

It will be noted in figures 1 and 2 that the shaded area indicated as normal is bounded not only by normal pH and alkali reserve lines, but also by the 35 and 45 mm. CO_2 tension lines within which the alveolar tensions of most normal individuals lie. It appears from results at present available that respiration is stimulated by high CO_2 tension of itself, as well as by the high $[H^+]$ it may cause, and that the condition found in the blood is a resultant of the two effects.

SUMMARY

1. A study has been made of the pH, CO_2 content, CO_2 tension, oxygen content, and oxygen capacity of the arterial blood in pneumonia. The CO_2 tension was calculated from the pH and CO_2 content of the blood. The other values were all determined directly.

2. A comparison was made of colorimetric and electrometric pH values with values calculated from the CO_2 absorption curves. A fair degree of consistency was obtained.

3. A lower arterial CO_2 tension during the febrile period than after the return to normal temperature was noted in 7 cases. Oxygen

unsaturation and lowered CO_2 tension do not occur together with sufficient regularity to indicate a causal relationship. Such relationship between temperature and CO_2 tension seems more probable.

4. No tendency towards an acidosis of either metabolic or respiratory origin was noted. The alkali reserve was within or near normal limits in every case. The pH likewise was in each instance within normal limits, 7.30 to 7.50, with the greater number of observations in the more alkaline half of this range.

5. These results contra-indicated alkali therapy in all the pneumonia cases studied.

6. In 8 of the 10 cases in which the arterial oxygen saturation was determined, an abnormally low saturation was observed at some stage of the disease. Taken with the non-occurrence of CO_2 acidosis, these results support the conclusion, made probable by known physiological and physico-chemical data, that when the mechanism for gas exchange in the lungs is affected absorption of oxygen fails before elimination of carbon dioxide is significantly impaired.

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