CO_2 EFFECTS ON ACID-BASE BALANCE IN AIR SATURATION DIVES

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SUMMARY PAGE

THE PROBLEM

To evaluate the effects of low levels of ambient CO_2, frequently found in diving operations due to inadequate CO_2 scrubbing, on acid-base parameters in air saturation dives to depth equivalents of 50 and 60 feet.

FINDINGS

In two saturation dives, rats of the Sprague-Dawley strain were exposed to increased air pressures equivalent to 50 and 60 feet of sea water for varying periods of time up to 60 days. The average ambient CO_2 concentrations (surface equivalent) were 0.41% CO_2 during the 60 day-50 foot saturation dive and 0.33% CO_2 during the 35 day-60 foot saturation dive.

In both experiments, consistent increases in arterial CO_2 tensions to 55-60 mm Hg, and decreases in pH were found. Plasma bicarbonate was elevated 5-7 mEq and plasma chloride correspondingly decreased. These findings indicate the existence of a pronounced respiratory acidosis which was thought to be caused by the low levels of ambient CO_2 in conjunction with increased oxygen and nitrogen levels. Neither the elevated nitrogen tension or oxygen tension alone could have produced such changes in acid-base parameters.

APPLICATION

A systematic study of the effects of increased ambient CO_2 levels, frequently found in diving operations, on acid-base parameters during saturation dives on air had, to our knowledge, not been made.

The findings indicate an approximate 10-fold increase in the effectiveness of ambient CO_2 under increased air pressures on acid-base parameters as compared to ambient CO_2 effects under normal atmospheric conditions. This could contribute significantly to accidents in diving operations.

It is recommended that greater attention be given to elimination of ambient CO_2 by installing efficient CO_2 scrubbing systems in diving operations.

ADMINISTRATIVE INFORMATION

This investigation was conducted as part of Bureau of Medicine and Surgery Research Work Unit MR041.01.01-0063BOKL. The present report is Number 9 on this work unit. It was submitted for review on 6 March 1973, approved for publication on 11 April 1973 and designated as NavSubMedRschLab Report No. 742.

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ABSTRACT

Blood gas tensions, pH, plasma chloride, sodium and potassium, blood urea, ammonia and amino acid nitrogen were measured in mature Sprague-Dawley rats at various intervals during and after exposure to pressures equivalent to depth at 50 feet and 60 feet, lasting for 60 and 35 days, respectively.

In both experiments, a consistent decrease in pH to values of about 7.30 and a rise in CO_2 tensions to 55-60 mm Hg in the arterial blood were found. Plasma bicarbonate was elevated 5-7 mEq and chloride correspondingly decreased. Plasma potassium was consistently increased in both experiments. These findings indicate the existence of a pronounced respiratory acidosis during the saturation dives to 50 and 60 feet.

There is no evidence in the literature which would indicate that, elevated pressures of nitrogen or oxygen, per se; in the ranges observed in these experiments could cause significant acid-base alterations. It must, therefore, be concluded that the increased ambient CO_2 levels in conjunction with the effects of increased oxygen and nitrogen is responsible for the observed changes. When compared with data obtained in rats during acute and chronic hypercapnia, the observed $PaCO_2$, pH, and bicarbonate values correspond to the range of values obtained by exposure to 3% and 5% CO_2.

CO_2 EFFECTS ON ACID-BASE BALANCE IN AIR SATURATION DIVES

INTRODUCTION

There is a lack of knowledge concerning the effects of increased levels of ambient CO_2 at increased pressures during short and chronic exposures to different gas mixtures. In previous studies involving saturation dives by men to pressures equivalent to 200, 800 and 1,000 feet of seawater, differences in ambient CO_2 resulted in marked differences in the response characteristics of respiration, acid-base balance and electrolyte excretion[8,9]. In the first experiment, in which three subjects were exposed for 12 days to a Helium-Oxygen-Nitrogen atmosphere at 200 feet, the average ambient CO_2 level amounted to 1.17% sea level equivalent. Alveolar carbon dioxide tension measured under these conditions corresponded with alveolar CO_2 tensions measured during inhalation of 5.5% CO_2 at normal atmospheric pressure. Electrolyte excretion in the urine showed patterns similar to those observed during exposure of dogs and man to 3% CO_2.

None of these findings were observed during saturation excursion dives to much greater depths at 800 and 1000 feet, breathing helium-oxygen under conditions, in which ambient CO_2 was below measurable levels. The alveolar CO_2 tensions were found to be normal and the pattern of urine electrolyte excretion did not show any evidence of CO_2 retention.

These findings indicated that increased pressure in a helium-oxygen-nitrogen atmosphere caused an amplification of ambient CO_2 effects by a factor of 5. The present report deals with a study of acid-base characteristics during air saturation dives at 50 and 60 feet in rats.

Due to external circumstances, the life-support system used in the chamber during the dives could not reduce the ambient CO_2 levels below 0.4 - 0.33% CO_2 (surface equivalent). The results indicate that the increased air-pressure produced an amplification of ambient CO_2 effects by a factor of 10 as compared with the effects of CO_2 exposure at normal atmospheric conditions.

METHODS AND MATERIALS

In two separate dives, male albino rats of the Sprague-Dawley strain, weighing 250 to 450 grams were exposed to hyperbaric conditions equivalent to 50 and 60 ft. of seawater, respectively, for varying periods of time up to 60 days. At the 50 ft. depth the chamber was maintained at 51.06% O_2 and 0.42% CO_2 (surface equivalent), and a temperature of 79.98F. At the 60 ft. depth the chamber was maintained at 56.8% O_2 and 0.33% CO_2 (surface equivalent), a temperature of 82.6F and a relative humidity of 73.9%. The following blood sampling procedure was utilized by trained divers at both depths.

Groups of five animals were anesthesized with sodium pentobarbital (0.1 cc/100 g). The abdominal aorta was exposed and approximately 10 cc of blood drawn with a heparinized glass

syringe. The anaerobic blood samples were cooled in ice and safely decompressed at a rate of 8 ft/min. In the 60-ft. dive a group of five animals at the surface were used as controls, at each time exposure, using the same sampling procedure.

A 6-7 ml aliquot of each sample was drawn off for plasma and red cell determination. The remaining anaerobic whole blood was used immediately for blood gas analysis and bicarbonate (HCO_3) determinations.

The PaO_2, $PaCO_2$ and pH were determined for each sample using the Instrumentation Laboratory (IL) pH/gas Analyzer Model 113. The PaO_2 was adjusted by a temperature correction factor of 1.02 for this instrument. The actual HCO_3 was calculated from an IL nomogram based on pH and $PaCO_2$. The standard HCO_3 was determined by equilibrating each blood sample with high CO_2 (7.94%) and low CO_2 (4.64%), using the Astrup Microtonometer AMT 1. Equilibration time was 3 minutes and each sample was measured twice. Actual and standard HCO_3 concentrations are expressed in mEquivalents/L blood.

Hematocrits were determined by drawing each sample into a 100 μl heparinized capillary tube which was then spun for 10 min. at 10,000 rpm and read on an International Circular Microcapillary reader.

Plasma was obtained by centrifuging 5-6 cc whole blood at 2000 rpm for 10 min. The plasma was drawn off and refrigerated immediately.

Plasma ammonia was measured within 2 hours of sampling with an Orion Ammonia Gas Sensing Electrode Model 95-10. The electrode was adapted for measurement of plasma volumes of less than 0.2 cc by addition of a special flow-thru cap obtained from Orion Research Incorporated and a Technicon Auto Analyzer Proportioning Pump Model 1.

Plasma sodium (Na) and potassium (K) were measured with an IL Flame-photometer Model 343 and plasma chloride (Cl^-) with a Buchler-Cotlove Chloridometer. Plasma urea was measured using a Hyland Laboratories UN Test Kit. Plasma amino acid nitrogen was measured according to Russell (1944)[7].

RESULTS

The 50 ft. dive data on chamber ambient CO_2, whole blood pH, $PaCO_2$, HCO_3 and Cl^- are shown in Figure 1. The CO_2 scrubbing system limitations resulted in a slightly elevated, mean ambient CO_2 of 3.1 mm Hg through the 60-day period.

The whole blood pH is acidotic during the first three days, shifts toward alkalotic at two weeks and then returns an acidotic state during the remainder of the experiment. Animals safely decompressed continue to show a slight acidosis after 5 days at the surface.

The early phase of acidosis is accompanied by a rapid rise in $PaCO_2$ and HCO_3 and a sharp decline in plasma Cl^-. The $PaCO_2$ and HCO_3 slowly recover to normal at two weeks and then

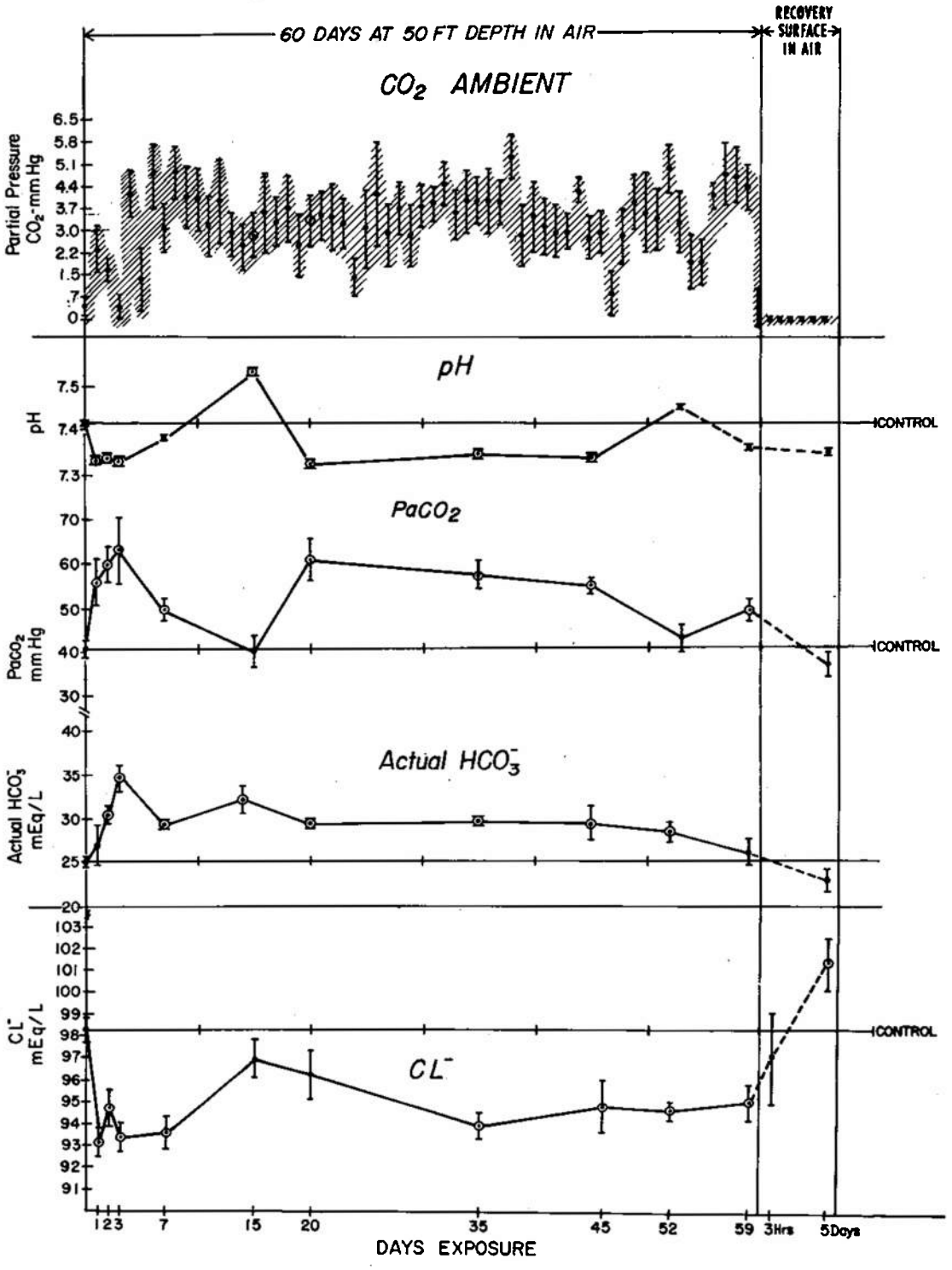

Fig. 1. Ambient CO_2 levels, acid-base parameters (pH, $PaCO_2$, and HCO_3) and plasma chloride during 60 days of exposure to air pressure equivalent to 50 feet of seawater and during 5 days of subsequent recovery at atmospheric pressure.

o control; • experimental
Ⓞ statistically significant difference from controls at the 5% level and better

rise again after three weeks. The Cl^-, on the other hand, remains low after the initial drop and returns to normal only after five days at the surface.

The plasma Na and K values are shown in Table 1. There is a dramatic increase in K levels during the 50 ft. exposure, but only intermittent changes in the Na levels.

The plasma urea and amino-acid nitrogen (N) values are shown in Table 2. Plasma urea values remain nearly normal, while amino acid N shows periodic fluctuations during the exposure.

The 60-ft. dive data on chamber ambient CO_2, whole blood pH, PaO_2 and $PaCO_2$ are shown in Figure 2. In this dive, improvement of the CO_2 scrubbing system resulting in a mean ambient CO_2 of 2.5 mm Hg throughout the 35-day period.

Again the whole blood pH is acidotic during the first week of exposure, then gradually returns to normal within four weeks. There is a marked alkalosis on the first day of recovery following safe decompression and a return to normal by the sixth day. PaO_2 is significantly elevated during the entire exposure, but rapidly returns to normal following safe decompression. As in the 50 ft. dive the $PaCO_2$ is elevated during the entire dive. There is a significant decline in $PaCO_2$ during the first day after safe decompression followed by a slow return to normal by the eighth day.

Figure 3 shows the changes in plasma Cl^-, actual $H\bar{C}O_3$, standard $H\bar{C}O_3$ and hematocrit for the 60 foot dive. As before, plasma Cl^- values exhibit a sharp decline on the first day and a significantly low Cl^- level persists throughout the dive. The recovery animals exhibit a return to normal Cl^- levels on the first day after decompression followed by a significant increase through the sixth day and a return to normal by the eighth day.

Actual $H\bar{C}O_3$ values again show a dramatic rise on the first day followed by a slow decline during the first week. There is a return to normal immediately following decompression.

The standard $H\bar{C}O_3$ shows a similar rapid rise in the first week of exposure and a rapid return to normal after decompression.

There is a very little change in hematocrit levels. A brief decline occurs during the first and second days following decompression.

The changes in plasma ammonia levels are shown in Figure 4. There is a sharp decrease during the first week of exposure, a return to near normal after ten days and then another rapid decline after four weeks. Following decompression there is a general return to normal, with some fluctuation at six days and two weeks.

The Na and K plasma levels are shown in Table 3. K shows a dramatic increase during the first five days and a return to normal by one week, while Na levels remain normal throughout the duration.

The plasma urea and amino acid N remain nearly normal as seen in Table 4.

TABLE 1

60 DAYS AT 50 FT. DEPTH IN AIR

DAY	Na (mEq./L plasma)			K (mEq./L plasma)		
	mean	S.E.M.	"t"	mean	S.E.M.	"t"
CONTROL	139.31	.25	-	3.52	.21	-
1	139.90	.80	.92	4.50	.00	8.39*
2	139.70	.47	.73	4.20	.14	5.55*
3	140.92	.72	2.72*	4.25	.096	6.56*
7	138.07	.51	2.31*	4.07	.095	4.99*
15	138.00	1.34	1.62	4.17	.18	4.88*
20	139.72	.30	.82	4.12	.048	5.84*
35	141.75	1.09	3.39*	4.15	.13	5.32*
45	140.47	.45	2.22	3.90	.13	3.14*
52	138.87	.46	.83	4.27	.14	6.22*
59	138.15	1.45	1.36	4.25	.029	7.17*
RECOVERY						
3 hrs.	140.20	.50	1.28	4.25	.15	4.87*
5 days	139.10	.32	.38	3.43	.033	.75

*Statistically significant from controls at the 5% level.

TABLE 2

60 DAYS AT 50 FT. DEPTH IN AIR

DAY	AMINO ACID N (mg/dl plasma)			UREA (mg/dl plasma)		
	mean	S.E.M.	"t"	mean	S.E.M.	"t"
CONTROL	6.48	.20	-	17.20	.94	-
1	9.65	1.29	4.69*	15.77	.88	.68
2	6.43	.36	.08	18.25	1.56	.54
3	7.55	.83	1.96	18.30	1.04	.59
7	7.44	.24	2.47*	16.35	1.54	.39
15	7.47	.56	2.51*	14.10	1.32	1.63
20	6.22	.24	.63	22.11	.77	2.68*
35	6.90	.71	1.14	18.81	1.17	.86
45	7.54	.38	2.54*	20.06	2.14	1.38
52	7.64	.59	2.46*	17.43	.87	.12
59	6.41	.66	.11	16.33	1.52	.45
RECOVERY						
3 hrs.	7.55	1.98	.65	15.43	1.12	.83
5 days	7.33	.33	1.89	19.21	1.45	.93

*Statistically significant from controls at the 5% level.

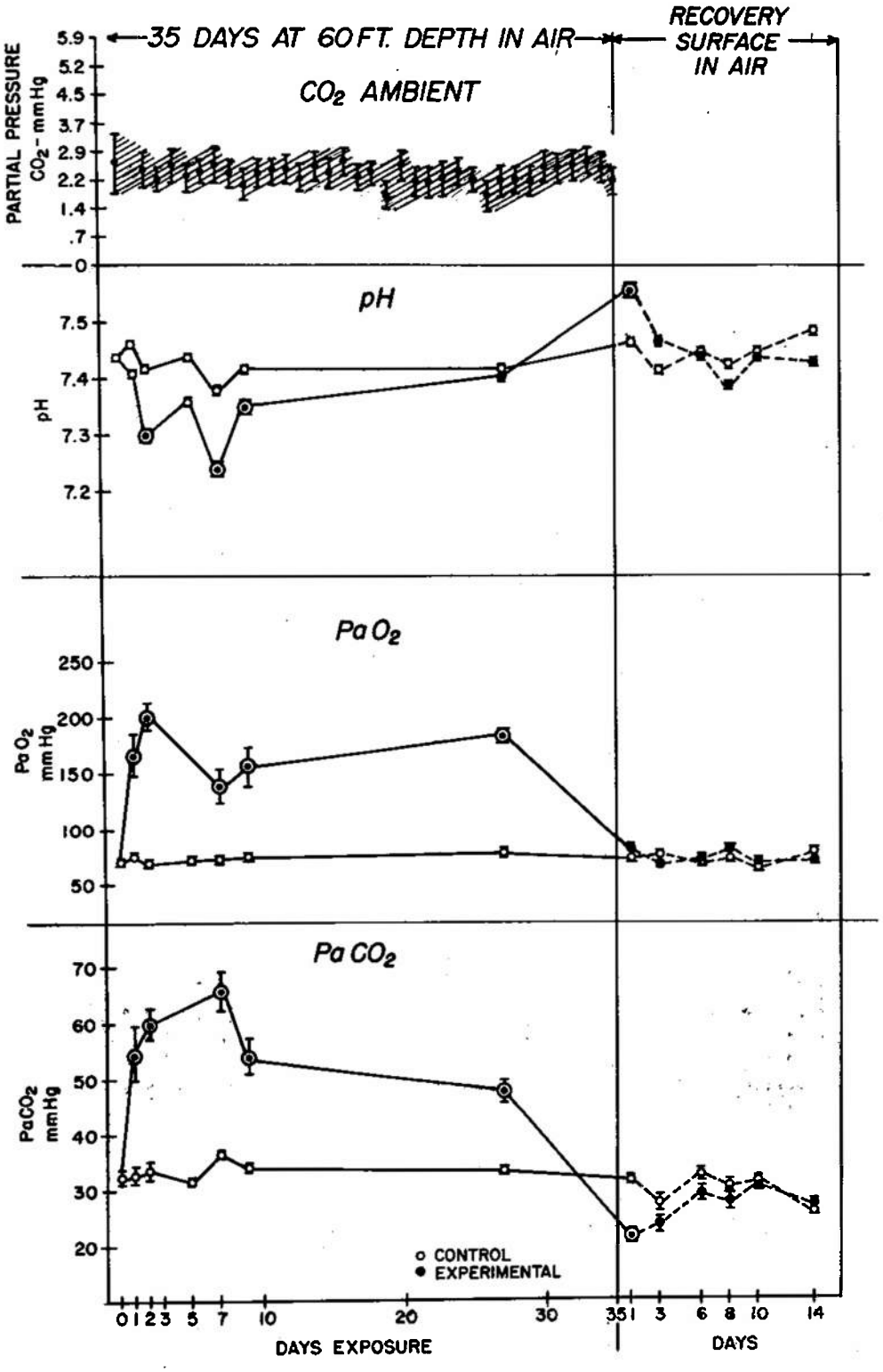

Fig. 2. Ambient CO_2 levels and acid-base parameters (pH, PaO_2 and $PaCO_2$) during 35 days of exposure to air pressure equivalent to 60 feet of seawater, and 2 weeks of subsequent recovery at atmospheric pressure.

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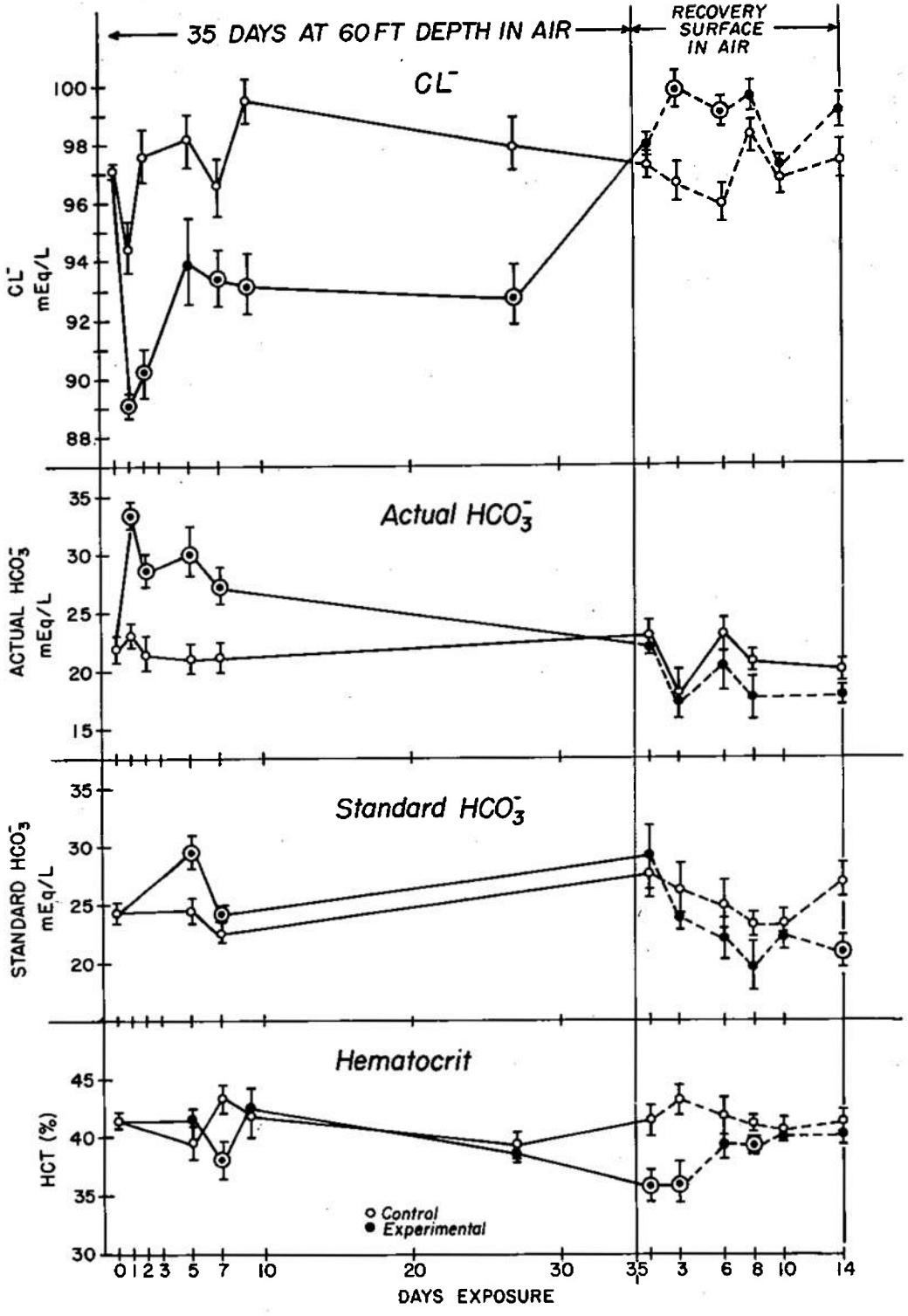

Fig. 3. Plasma chloride, actual and standard bicarbonate and hematocrit during 35 days of exposure to air pressure equivalent to 60 feet of seawater and during 2 weeks of subsequent recovery at atmospheric pressure.

o control; • experimental
Ⓞ statistically significant difference from controls at the 5% level and better

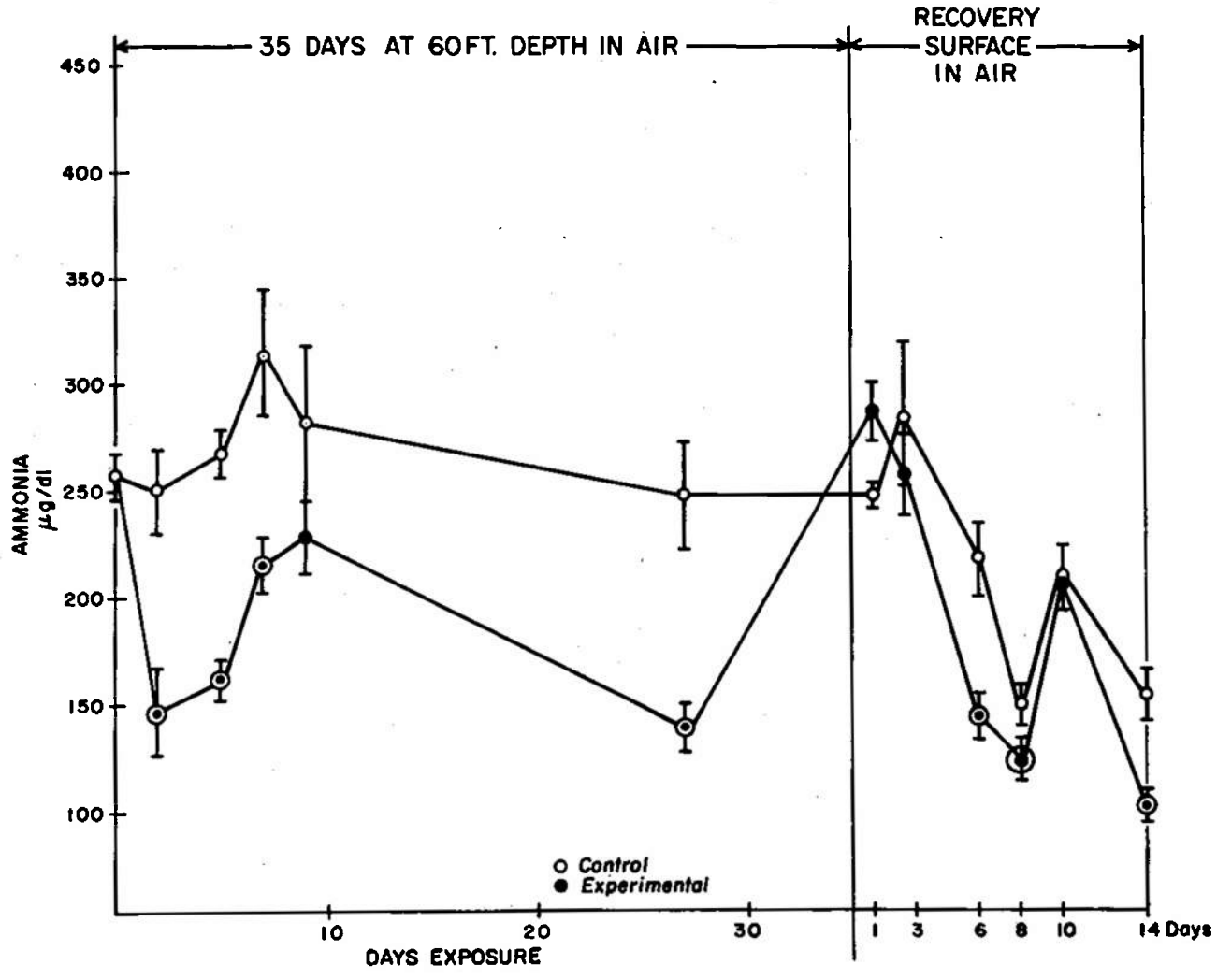

Fig. 4. Plasma ammonia levels during 35 days of exposure to air pressure equivalent to 60 feet of seawater and during 2 weeks of subsequent recovery at atmospheric pressure.

o control; • experimental
Ⓟ statistically significant difference from controls at the 5% level and better

DISCUSSION

The remarkable increases of arterial $PaCO_2$ observed during the first experiments (saturation air dive at 50 feet) led us to look for a possible artifact due to narcosis at increased pressure or some other incidental effect. However, the corresponding decreases in plasma chloride levels support the findings of a respiratory acidosis obtained by blood gas analysis. The recovery values, showing a decrease in bicarbonate and increase in chloride, give further credence to the conclusion that a real respiratory acidosis existed during the 50-foot saturation dive on air.

TABLE 3

35 DAYS AT 60 FT. DEPTH IN AIR

DAY	Na (mEq./L plasma)			K (mEq./L plasma)		
	mean	S.E.M.	"t"	mean	S.E.M.	"t"
CONTROL	140.01	.256	-	3.88	.40	-
1	140.03	.92	.20	4.37	.88	2.71*
2	141.05	1.42	1.00	4.75	.45	4.66*
5	142.70	.61	2.36	4.83	.58	4.54*
7	141.70	.69	1.70	3.67	.26	1.23
9	140.82	.65	.82	3.70	.11	1.14
27	139.50	.24	.52	4.02	.11	.93
RECOVERY						
1	138.40	.30	1.42	3.67	.12	1.18
3	140.47	.41	.47	3.82	.18	.34
6	139.52	.54	.49	3.37	.14	3.18*
8	140.17	.59	.14	3.90	.00	.11
10	139.77	.28	.24	3.42	.63	2.92*
14	138.22	.37	1.81	3.80	.15	.50

*Statistically significant from controls at the 5% level.

TABLE 4

35 DAYS AT 60 FT. DEPTH IN AIR

DAY	AMINO ACID N (mg/dl plasma)			UREA (mg/dl plasma)		
	mean	S.E.M.	"t"	mean	S.E.M.	"t"
CONTROL	8.81	.10	-	18.50	.58	-
1	8.83	.0	-	14.01	.56	2.43*
2	8.78	.48	.04	17.15	.64	.85
5	8.36	.64	.64	14.17	.22	1.93
7	8.13	.25	1.13	16.87	.60	1.02
9	7.56	.21	2.07	20.40	1.09	1.17
27	8.06	.38	1.23	18.58	.52	.47
RECOVERY						
1	8.34	.10	.57	-	-	-
3	8.51	.29	.50	-	-	-

*Statistically significant from controls at the 5% level.

During the second air saturation dive at 60 feet, measurements of experimental animals exposed to pressure were in each instance carried out simultaneously with measurements of the same number of control animals. Again, the findings revealed a severe respiratory acidosis in the experimental animals, but not in the control animals. Moreover, measurements during an extended recovery period reflect a response in the acid-base parameters; in particular, the decreases in bicarbonate and increases in chloride, which could have been produced only by a persistent respiratory acidosis during the exposure period.

TABLE 5

BICARBONATE-CHLORIDE EXCHANGE IN PLASMA OF RATS AT DEPTHS OF 50 and 60 FEET

		H^+ Nanamols/L.	Δ	PCO_2 mm Hg	Δ	HCO_3^- mEq./L.	Δ	Cl^- mEq./L.	Δ
	CONTROL	35.0		35		25		101	
1. 50 Feet									
	1 Day	47.0	+12	56	+21	28	+3	93	-8
	3 Days	48	+13	63	+28	34	+9.0	93	-8
	7 Days	42	+7	50	+15	30	+5	94	-7
	30 Days	45	+10	58	+23	30	+5	94	-7
	CONTROL	35.3		33		22.0		97	
2. 60 Feet									
	1 Day	48	+13	54	+21	29	+7	89	-8
	3 Days	50	+15	60	+21	29	+7	90	-7
	7 Days	56	+21	66	+33	27	+5	93	-5
	27 Days							93	-5
RECOVERY AIR	1 Day	23	-12	23	-13	21	-1	98	+1
	3 Days	31	-4	25	-8	18	-4	100	+3
	6 Days	34	-1	29	-4	20	-2	99	+2
	14 Days	36	+1	27	-6	18	-4	99	+2

These findings are considered to provide sufficient evidence for the existence of a severe respiratory acidosis during the air saturation dives to 50 and 60 feet.

Three factors could have contributed to the observed changes in acid-base parameters: the increased pressures of nitrogen, oxygen and CO_2. Based on the findings of Lambertsen[3] on acid-base characteristics in human subjects during 14 days of exposure to an increased atmospheric density, increased nitrogen pressure equivalent to 100 feet of seawater, and normal inspired oxygen tension, any effects of nitrogen per se at increased pressures on acid-base parameters seemed to be very unlikely. According to this report, $PaCO_2$ showed only slight elevations from sea level control of 41.5 mm Hg to 44.6 mm Hg on Day 7-9 returning to control levels at Days 12-14 or 41.7 mm Hg. The pH values did not change at all.

Increased oxygen levels of 51-57% in the ambient atmosphere could only contribute to a very small rise in $PaCO_2$ due to interference with the CO_2 transport of hemoglobin. However, as has been shown by Lambertsen *et al*[4] this increase in $PaCO_2$ results in a hyperventilation and subsequent decrease in $PaCO_2$. Breathing air at 3.5 atm. which corresponds to an ambient oxygen concentration of 73.5% surface equivalent, did not significantly alter the arterial CO_2 tension or the pH.

It must, therefore, be concluded that the increased oxygen levels existing in our experiments could certainly not cause an increase of arterial CO_2 tensions amounting to 20 - 25 mm Hg. Thus, the increased ambient CO_2 level is seen as the only other possible factor which could contribute to such increases in arterial $PaCO_2$ changes. However, on the basis of the available data it is not possible to state how this CO_2 effect is accomplished.

If the $PaCO_2$ and pH data obtained in 50 and 60 foot air saturation dives are compared with $PaCO_2$ and pH data collected under conditions of acute and chronic hypercapnia in rats[11], they are found to lie in the range of values measured during exposure of rats to 3% and 5% CO_2.

This means that under increased air pressure, through possibly potentiating effects of increased oxygen and nitrogen, the CO_2 effects, as far as pH and $PaCO_2$ are concerned, are amplified by a factor of 10.

Adaptation to hypercapnia involves an increase of bicarbonate accompanied by a nearly equal decrease in plasma chloride. It has been shown by Polak *et al*[6] for the dog and Levitin *et al*[5] for the rat, that the magnitude of the urinary chloride excretion during chronic hypercapnia is sufficient to account for the decline in plasma chloride. The increased chloride excretion in chronic hypercapnia represents an important defense mechanism for the maintenance of pH in addition to the increased bicarbonate reabsorption in the kidney[11].

In both saturation dives at 50 and 60 feet the increases in plasma bicarbonate and the corresponding decreases in plasma chloride indicate the existence of a chronic hypercapnia, showing the same pattern of

acid-base changes as seen in hypercapnia under normal atmospheric conditions.

Increased plasma potassium levels have also been observed in chronic hypercapnia induced by exposure of guinea pigs to 15% CO_2[10] and in patients with respiratory acidosis (Kilburn[2]). These findings on potassium changes are therefore additional indicators of a chronic respiratory acidosis.

An increased urinary ammonia excretion has been found in rats during chronic hypercapnia[1,2] which could be reflected in a lowered blood ammonia level in line with observations made in this study.

The stress of rather severe respiratory acidosis imposed by increased ambient CO_2 levels during the air saturation dives did not result in any mortality of the rats maintained on a normal diet. This is in agreement with the high resistance to damage from carbon dioxide observed in rats at normal atmospheric pressure. Rats exposed for prolonged periods to 20% CO_2 did not show any mortality[11].

Exploratory histopathological investigations were carried out in 30 rats exposed for various time intervals to pressures equivalent to 50 and 60 feet of seawater and subsequently these animals were decompressed. Light microscopy studies did not show clearly pronounced signs of pulmonary damage in any of the animals.

This does not exclude, however, the possibility that systematic studies of a larger number of animals might have revealed slight histopathological changes in the lung, i.e., perivascular and alveolar edema as observed in rats during prolonged exposure to 3% CO_2 (Niemoeller and Schaefer[6]). The pH values measured in the present series of saturation dives correspond with the pH values obtained in rats exposed to CO_2 concentrations of 3% and above.

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13. ABSTRACT

Blood gas tensions, pH, plasma chloride, sodium and potassium, blood urea, ammonia and amino-acid nitrogen were measured in mature Sprague-Dawley rats at various intervals during and after exposure to pressures equivalent to depth at 50 and 60 feet, lasting for 60 and 35 days, respectively.

In both experiments, a consistent decrease in pH values to about 7.30 and a rise in CO_2 tensions to 55-60 mm Hg in the arterial blood were found. Plasma bicarbonate was elevated 5-7 mEq and chloride correspondingly decreased. Plasma potassium was consistently increased in both experiments. These findings indicate the existence of a pronounced respiratory acidosis during the saturation dives to 50 and 60 feet.

There is no evidence in the literature which would indicate that as individual agents, elevated pressures of nitrogen or oxygen in the ranges observed in these experiments could cause significant acid-base alterations. It must, therefore, be concluded that the increased ambient CO_2 levels in conjunction with the effects of increased oxygen and nitrogen is responsible for the observed changes. When compared with data obtained in rats during acute and chronic hypercapnia the observed $PaCO_2$, pH and bicarbonate values correspond to the range of values obtained by exposure to 3% and 5% CO_2.

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Acid-base balance						
Respiratory acidosis						
Air-Saturation						
Chronic hypercapnia						